THE STUDY OF THYROID DYSFUNCTION IN PATIENTS WITH ABNORMAL UTERINE BLEEDING

By

Dr. DHANUSHA NEKKANTI MBBS

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SURGERY

IN

OBSTETRICS AND GYNAECOLOGY

Under the Guidance of

DR. VASANTHA KUMAR. S

PROFESSOR

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

SRI DEVARAJ URS MEDICAL COLLEGE

TAMAKA, KOLAR 563101

JUNE 2023

ALMA MATER

R.L. JALAPPA HOSPITAL AND RESEARCH CENTRE

SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR-563101

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"THE STUDY OF THYROID DYSFUNCTION IN PATIENTS WITH ABNORMAL UTERINE BLEEDING**" is a bonafide and genuine research work carried out by me under the guidance of **Dr. VASANTHA KUMAR. S,** Professor, Department of Obstetrics and Gynaecology, Sri Devaraj Urs Medical College, Tamaka, Kolar.

Place: Kolar

Dr. DHANUSHA NEKKANTI

SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR-563101

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"THE STUDY OF THYROID DYSFUNCTION IN PATIENTS WITH ABNORMAL UTERINE BLEEDING"** is a bonafide research work done by **DR. DHANUSHA NEKKANTI** in partial fulfillment of the requirement for the Degree of MASTER OF SURGERY in Obstetrics and Gynecology

Date:

Place: Kolar

Dr. VASANTHA KUMAR. S

Professor
Department of OBG
Sri Devaraj Urs Medical College,
Tamaka, Kolar

.

SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR-563101

ENDORSEMENT BY THE HEAD OF THE DEPARTMENT,

PRINCIPAL / HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"THE STUDY OF THYROID DYSFUNCTION IN PATIENTS WITH ABNORMAL UTERINE BLEEDING"**is a bonafide research work done by **DR DHANUSHA NEKKANTI** under the guidance of **DR VASANTHA KUMAR. S,** Professor, Department of Obstetrics and Gynecology.

DR. RATHNAMMA. P
Associate Professor & HOD
Department Of OBG
Sri Devaraj Urs Medical Medical College,
Tamaka, Kolar

DR.P.N.SREERAMULU
Principal
Sri Devaraj Urs Medical Medical College
Tamaka, Kolar

SRI DEVARAJ URS MEDICAL COLLEGE

KOLAR-563101

ETHICS COMMITTEE CERTIFICATE

This is to certify that the Ethics committee of Sri Devaraj Urs Medical College, Tamaka, Kolar, has unanimously approved **Dr. DHANUSHA NEKKANTI,** a post-graduate student in the subject of **OBSTETRICS AND GYNAECOLOGY** at Sri Devaraj Urs Medical College, Kolar to take up the dissertation work entitled **"THE STUDY OF THYROID DYSFUNCTION IN PATIENTS WITH ABNORMAL UTERINE BLEEDING"** to be submitted to **SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, TAMAKA, KOLAR.**

Date:

Place: Kolar

Member Secretary

Sri Devaraj Urs Medical College,

Kolar–563101

SRI DEVARAJ URS MEDICAL COLLEGE TAMAKA,

KOLAR-563101

COPY RIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that the Sri Devaraj Urs Academy of Higher Education and Research, Kolar, Karnataka, shall have the right to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic /research purposes.

Date: **Dr. DHANUSHA NEKKANTI**
Place: Kolar

PlAGIARISM CERTIFICATE

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH
Tamaka, Kolar 563103

Certificate of Plagiarism Check

Title of the Thesis/Dissertation	THE STUDY OF THYROID DUSFUNCTION IN PATIENTS WITH ABNORMAL UTERINE BLEEDING
Name of the Student	DR. NEKKANTI DHANUSHA
Registration Number	20OG1035
Name of the Supervisor / Guide	DR. VASANTHA KUMAR. S
Department	OBSTETRICS AND GYNAECOLOGY
Acceptable Maximum Limit (%) of Similarity (PG Dissertation /Ph.D. Thesis)	10%
Similarity	8%
Software used	Turnitin
Paper ID	1992092049
Submission Date	13-JAN-2023

Signature of Student

Signature of Guide/Supervisor

Dr. VASANTHA KUMAR S
Professor of OBG, SDUMC
KMC Reg. No. 19324

HOD Signature

Dr. KATHNAMMA.P
Assoc. Professor
Dept. of OBG
KMC Reg. No. 16761
Date: Time:

University Librarian

University Library
Learning Resource Centre
SDUAHER, Tamaka
KOLAR-563103

Coordinator UG and PG Program

Co-Ordinator,
UG&PG Program ,Faculty of Medicine,
Sri Devarj Urs Medical College ,
Tamaka, Kolar- 563103

turnitin

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	Nekkanti Dhanusha
Assignment title:	PG DISSERTATION 2022
Submission title:	THE STUDY OF THYROID DYSFUNCTION IN PATIENTS WITH A...
File name:	THYROID_BOOK_3_less_than_10_13-1-23.docx
File size:	1.8M
Page count:	73
Word count:	12,095
Character count:	67,297
Submission date:	13-Jan-2023 08:41AM (UTC+0530)
Submission ID:	1992092049

ABSTRACT:

Background:

Methodology:

Results:

Conclusion:

University Library
Learning Resource Centre
SDUAHER, Tamaka
KOLAR-563103

Copyright 2023 Turnitin. All rights reserved.

Dr. VASANTHA KUMAR.S
Professor of OBG, SDUMC
KMC Reg. No. 19324

Document Viewer

Turnitin Originality Report

Processed on: 13-Jan-2023 08:42 IST
ID: 1992092049
Word Count: 12095
Submitted: 1

THE STUDY OF THYROID DYSFUNCTION IN PATIENTS ...
By Nekkanti Dhanusha

Similarity Index	Similarity by Source	
8%	Internet Sources:	5%
	Publications:	4%
	Student Papers:	2%

include quoted | exclude bibliography | excluding matches < 10 words | mode:
quickview (classic) report | print | refresh | download

1% match ()
Minaxi Thakur, Meenu Maharjan, Heera Tuladhar, Yam Dwa, Sunita Bhandari, Smrity Maskey, Manisha Bajracharya. "Thyroid Dysfunction in Patients with Abnormal Uterine Bleeding in a Tertiary Care Hospital: A Descriptive Cross-sectional Study", JNMA: Journal of the Nepal Medical Association

1% match (student papers from 10-Jan-2023)
Submitted to Sri Devraj Urs Acaedmy of Higher Education and Research, Kolar on 2023-01-10

1% match (Internet from 17-Oct-2021)
https://www.jemds.com/data_pdf/1_manjusha%20agrawal--feb-17-.pdf

<1% match ()
Stephen P. Fitzgerald, Henrik Falhammar. "Redefinition of Successful Treatment of Patients With Hypothyroidism. Is TSH the Best Biomarker of Euthyroidism?", Frontiers in Endocrinology

<1% match ()
Lucy Whitaker, Hilary O.D. Critchley. "Abnormal uterine bleeding", Best Practice & Research. Clinical Obstetrics & Gynaecology

<1% match ()
Surya P Nuguru, Sriker Rachakonda, Shravani Sripathi, Mashal I Khan, Naomi Patel, Roja T Meda. "Hypothyroidism and Depression: A Narrative Review", Cureus

<1% match (Internet from 20-Aug-2022)
https://www.ncbi.nlm.nih.gov/books/NBK532913/

<1% match ()
Iñigo Murga Gandasegui, Larraitz Aranburu Laka, Pascual-Ángel Gargiulo, Juan-Carlos Gómez-Esteban et al. "Myalgic Encephalomyelitis/Chronic Fatigue Syndrome: A Neurological Entity?", Medicina

<1% match (Internet from 26-Sep-2022)

Dr. VASANTHA KUMAR. S
Professor of OBG, SDUMC
Kolar Reg No. 19324

University Library
Learning Resource Centre
SDUAHER, Tamaka
KOLAR-563103

ACKNOWLEDGEMENT

First and foremost, I thank God for giving me his endless blessing and giving me the strength both mentally and physically during my post-graduation and to make this dissertation book possible.

This dissertation has been one of the most significant academic challenges I have ever had to face. Without the support, patience and guidance of the following people, this study would not have been impossible. It is to them I owe my deepest and most sincere gratitude.

Firstly, I would like to thank my Guide **Dr. VASANTHA KUMAR. S**, Professor of Department of Obstetrics and Gynecology, SDUMC Kolar, for him utmost patience, continuous support, guidance and contribution. I would also like to thank him for his constant encouragement to highest peak, paying close and continuous Attension towards me to finish all tasks with respect to every aspect of my professional life.

I am sincerely thankful to **Dr. RATHNAMMA P**, Associate professor and head of Department of Obstetrics and Gynaecology for encouraging me to highest peak, paying close and continuous Attention towards me to finish all tasks, and also providing her kind support, valuable suggestions ,Immense patience and great care. Her precious advice on both the dissertation work as well as the path of my career has been priceless

I whole heartly acknowledge **DR SHEELA S R**, **Dr MUNIKRISHNA. M** Professor in the department of obstetrics and Gynaecology , for their Valuable teaching of perseverance professional ethics ,moral support and commitment . The precious advice on both the dissertation as well as on my career was invaluable.

I extend my gratitude towards all the patients who agreed to participate in this study,

without their precious support it would not be possible to conduct this research.

I thank my fellow post graduates and my friends **Dr SHRAVYA MONICA, Dr LISLEY KONAR, Dr. AKSHITHA SAI RAGAM, Dr SAHITHYA SIVAPRASAD, Dr RAVEENA KETHINENI, Dr PRATYUSHA BORTHAKUR**, for their unflinching support, for staying with me emotionally and constantly supporting me and bearing with me through all the deadlines.

I sincerely thank all assistant professor **Dr VIMARSHITHA, Dr PAVITHRA J, Dr DIVYA, Dr NANDINI S, Dr CHANDRACHUR and all the senior residents Dr DEEKSHA, Dr SUDHA MALLIDI, Dr MADANA JYOTSNA PRIYA , Dr SHREYA, Dr NEHA, Dr YASHASWANI, Dr CHAITANYA** in the department of OBG ,SDUMC , KOLAR, for their constant guidance and encouragement.

I thank all the staff nurses who are our pillars of support special thank to all labour room staff for their help and support throughout my study.

I expressed my profound gratitude to my beloved grandparents **Mr. EDARA KONDAYYA**, **Mrs**. **EDARA VIJAYA LAKSHMI** for constantly entertaining me and keeping up to good spirit.

I expressed my profound gratitude to my beloved maternal uncle **Mr. ASHOK CHAKRAVARTHY. EDARA** and my beloved parents **Mrs. NEKKANTI ADILAKSHMI NEKKANTI ,Mrs. NEKKANTI SURYA PRAKASA RAO** for always inspiring me, for giving me continuous encouragement , unfailing support , and unconditional love throughout my life.

I would like to thank my little brother **Dr. NEKKANTI VIJAY ANAND** for staying with me emotionally, even being miles apart and constantly supporting me and bearing with me through all the deadlines.

Last but not the least, I extend my gratitude towards all the patient who agreed to participate in this study, without their precious support, it would not be possible to conduct this research.

PLACE:

DATE: **DR. DHANUSHA NEKKANTI**

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
AUB	Abnormal uterine bleeding
TFT	Thyroid functional test
TSH	Thyroid stimulating hormone
TRH	Thyroid releasing hormone
T3	Triiodothyronine
T4	Tetraiodothyronine
IMB	Inter menstrual bleeding
PPH	Postpartum hemorrhage
HMB	Heavy menstrual bleeding
GnRH	Gonadotropin releasing hormone
SHBG	Sex hormone binding globulin
FSH	Follicle stimulating hormone
LH	Luteinizing hormone
DHEA-S	Dehydroepiandrosterone- Sulfate
IUCD	Intra uterine contraceptive device
PID	Pelvic inflammatory disease
PCOS	Poly cystic ovarian syndrome
PT	Prothrombin time
APTT	Activated partial thrombin time
BMR	Basic metabolic rate
CNS	Central nervous system
WHO	World health organization
ATDs	Anti-Thyroid drugs

ABSTRACT

Background:

Term "abnormal uterine bleeding" refers to a wide range of disturbances in the menstrual cycle, including variations in flow volume, length, and frequency that don't occur during pregnancy. Either uterine structural defects or other factors may be involved in the etiology of AUB.. Endocrinological disturbances form a significant group in etiology of AUB. Among the endocrinological causes production and activity of thyroid hormones have profound effect on menstrual abnormality.

Methodology:

This is a prospective, observational study to detect, evaluate thyroid disorders in patient with unusual vaginal bleeding and to link thyroid dysfunction to various menstrual bleeding patterns. Thyroid dysfunction and Type of bleeding were considered as primary outcome variables. Age group, parity, TSH, T3, T4 were considered as Primary explanatory variables.

Results:

32% of AUB cases are discovered in the study to be related to some form of thyroid malfunction. Many of study participants had very low levels of TSH and other 17.74% also have just normal levels of TSH. Among study participants detected with hypothyroidism majority of them (20.83%) had oligomenorrhoea and among study participants detected with subclinical hypothyroidism majority of them (37.50%) had polymenorrhoea followed menorrhagia reported in (23.40%) participants.

Conclusion:

This study found higher percentage of participants with abnormal uterine bleeding had thyroid dysfunction in comparison with other similar studies. 32% of AUB cases are found to be connected, according to the study, to some form of thyroid malfunction. The most common bleeding disorder observed in participants detected with hypothyroidism was oligomenorrhoea followed by menorrhagia.

INTRODUCTION

THE STUDY OF THYROID DYSFUNCTION IN PATIENTS WITH ABNORMAL UTERINE BLEEDING

INTRODUCTION

Term "abnormal uterine bleeding" refers to a wide range of anomalies in the menstrual cycle, including variations in flow volume, length, and frequency that don't occur during pregnancy.. Though mortality from abnormal uterine bleeding is rare it has profound effect on quality of life of a woman. Epidemiological data indicates that around 10 to 30% of women in reproductive age are affected by heavy menstrual bleeding called menorrhagia [1]. Assessment of menstrual bleeding is done using six parameters namely frequency, length, periodicity, quantity, intermenstrual bleeding, and unexpected bleeding [2].

Either uterine structural defects or unrelated conditions can be the cause of AUB, according on its aetiologies. With the acronym “PALM- COEIN”- “polyp, adenomyosis, leiomyoma, malignancy and hyperplasia, coagulopathy, ovulatory dysfunction, endometrial, iatrogenic”, and not otherwise defined, they are grouped together.Appropriate and effective management of AUB can be achieved only with accurate diagnosis of AUB etiology. AUB is a symptom of an underlying disease and is not a disease itself. It can strike at any age and manifest itself in various ways [3].

Endocrinological disturbances form a significant group in etiology of AUB. Among the endocrinological causes production and activity of thyroid hormones have profound effect on menstrual abnormality. The two thyroid hormone disturbances- hypothyroidism and hyperthyroidism both cause menstrual abnormalities [4]. Thyroid disorders are widely prevalent world-wide. In India there has been rapid increase in thyroid disorders in recent years due to various reasons including rise in autoimmunity, rapid iodination and obesity [5].

The prevalence of overt hypothyroidism in developed countries is estimated to be 4 to 5% while the prevalence of subclinical hypothyroidism is 4 to 15%. Hypothyroidism among women in reproductive age causes menstrual irregularities, polycystic ovaries, miscarriages and infertility. The physiology of hypothalamus – pituitary thyroid axis depends on thyroid hormone levels. AUB or menstrual irregularities occur before onset of overt hypo or hyperthyroidism [6].

Women in normal population are ten times as more likely as men to experience thyroid problems [7]. Menorrhagia has been linked to hypothyroidism, and oligomenorrhea and amenorrhea has been linked to hyperthyroidism [8]. Many studies have shown that treating thyroid dysfunction will help in improvement of menstrual abnormalities[9].

NEED OF THE STUDY:

Thyroid disorders are found to increase rapidly in India in recent years. Thyroid disorders are also found to be the causative factor for AUB in majority of cases. Thyroid disorders are easy to diagnose and treat. The sensitivity and specificity of tests to determine how well the thyroid is working have enhanced with the development of "serum thyroxine" (T3) and "thyroid stimulating hormone" (TSH) radioimmunoassay.

Establishing association between thyroid disorders and AUB will help in ease of diagnosis and treatment for most of AUB cases which might otherwise go undiagnosed. It will help in avoiding unnecessary surgical intervention, hormonal treatment and related complications. The goal of this study is to determine the prevalence of thyroid disorders in AUB patients between the ages of 20 and 45 who have AUB in order to better treat their condition moving forward.

AIMS & OBJECTIVES

AIMS AND OBJECTIVES:

1. To detect and evaluate thyroid disorders in patient with abnormal uterine bleeding.
2. To correlate different menstrual patterns with thyroid dysfunction

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Abnormal uterine bleeding:

Definition:

The following is definition of abnormal uterine bleeding according to FIGO 2018 guidelines:

Regularity: 7 to 9 days

Duration of bleeding: uptill 8 days

< 8 days: Normal

>8days: Prolonged.

Bleeding which does not follow above characteristics is termed abnormal uterine bleeding [10].

According to the definition of chronic AUB, it refers to bleeding from the uterine corpus that has been present for the majority of the previous six months and is aberrant in volume, regularity, or timing. Values beyond the typical range of 5-95th percentile rank were abnormal [11].

Acute AUB is profuse bleeding that needs to be treated right away to stop more blood loss. Chronic AUB may coexist with acute AUB, which is defined as abnormalities in periodic bleeding for the bulk of the last six months[12].

Intermenstrual Bleeding:

In a woman who is not pregnant and who is not taking any hormonal treatment uterine bleeding occurring between normal pattern of menstruation is called as intermenstrual bleeding.

There are two types of intermenstrual bleeding- one that occurs only once between normal menstrual cycles and other type that occurs repeatedly between menstrual cycles. The first type of intermenstrual bleeding occurs in about 20 to 30% of women or 1 -2% of all menstrual cycles. This intermenstrual bleeding usually occurs between day 10 and day 16 and last for 12 to 72 hours. The bleeding is usually very light or scanty and hence is called spotting of blood.

Physiological IMB is associated with ovulation. The increased levels of serum oestrogen towards end of follicular phase gives negative feedback to pituitary gland resulting in increased levels of Lutenizing hormone. This sudden increase in Lutenizing hormone levels causes abrupt dip in circulating oestrogen levels before ovulation. This change in levels of oestrogen precipitates breaking down of endometrium resulting in intermenstrual bleeding [13].

The following table gives suggested normal limits of menstrual bleeding:

Clinical parameter	Descriptive term	Normal limits (5- 95th percentiles)
Frequency of menses (days)	Frequent Normal Infrequent Intermenstrual bleeding	<24 24–38 >38 Occurs Between 10 to 16 days of normal menstrual pattern or occurs on and off between normal menstrual pattern.
Regularity of menses, cycle to cycle (Variation in days over 12 months)	Absent Regular Irregular	No bleeding Variation ± 2–20 days Variation >20 days
Duration of flow (days)	Prolonged Normal Shortened	>8.0 4.5–8.0 <4.5
Volume of monthly blood loss (mL)	Heavy Normal Light	>80 5–80 <5[14].

CONFIDENTIAL

Anatomy of uterus:

The centre of the abdominal pelvic cavity is where the uterus is located. It is a muscular structure with a thick wall. It has 3 layers: an interior one called the endometrium, a middle one called the myometrium, and an outermost one called the perimetrium. In reaction to hormonal stimulation, the endometrium's thickness and shape change.

The four parts of the uterus are the fundus, corpus, isthmus, and cervix. The largest component, the corpus, is joined to the cervix by the isthmus. The cervix connects the vaginal lumen to the uterine body. The bladder is located behind the uterus. Uterus sits posterior to the bladder and anterior to the rectum[15].

Uterus is connected to the abdominal wall by round ligament having artery of Sampson, it is connected to fallopian tube and ovary by a broad ligament. The broad ligament encompasses the uterine artery, cardinal arteries, and ureter. Uterus has its main blood supply through uterine artery and ovarian artery adds onto vasculature of uterus by providing collateral blood supply. Both hypogastric nerve and pelvic splanchnic nerves innervate the uterus in a sympathetic and parasympathetic manner [16].

Menstrual cycle:

Menstrual cycle is controlled by hormones in negative and positive feedback manner. With onset of puberty Gonadotropin releasing hormone (GnRH) is secreted by hypothalamus which is transported to anterior pituitary gland. On reaching anterior pituitary gland GnRH activates 7-transmembrane G-protein to secrete follicle stimulating hormone and Lutenizing hormone. These two hormones stimulate hormone producing cell types of ovary. Through the action of the cholesterol desmolase enzyme, lutenizing hormone increases the production of progesterone and androstenedione by theca cells. Androstenedione diffuses to granulosa cells

which upon stimulation by Follicle Stimulating Hormone convert it to testosterone and then to 17-beta estradiol through action of aromatase enzyme. The levels of 17-beta oestradiol and progesterone are controlled by negative feedback mechanism.

Phases of menstrual cycle:

Phase I- Follicular phase:

When the average menstrual cycle lasts 28 days, the follicular or proliferative phase, also known as the first phase, lasts from day one to day 14. The length of follicular phase is responsible for variation of length of menstrual cycle. 17-beta- oestradiol (oestrogen) is the main hormone of this phase. Endometrial layer of uterus grows during this phase. This phase also creates channels for entry of sperm withing the abundant watery and elasticity changes of cervical mucous. Primordia follicle matures to Graafian follicle.

Ovulation:

Ovulation happens 14 days before menstruation starts. Positive feedback from "17-beta-estradiol results in increased synthesis of luteinizing hormone (LH) and follicle stimulating hormone (FSH) (LH). High FSH and LH levels cause the mature follicle to rupture, releasing the egg".

Phase2- Luteal or secretory phase:

“This is the phase from day 14 to day 28 of menstrual cycle. Progesterone stimulated by LH prepares corpus luteum and endometrium for implantation of fertilized ovum. Towards the end of luteal phase FSH and LH levels and subsequently 17-beta-oestradiol and progesterone levels are decreased due to negative feedback of progesterone. Progesterone slows down endometrial proliferation decreasing its thickness. It also increases hypothalamic temperature.

Plasma levels of 17-beta-oestradiol and progesterone are produced towards end of secretory phase by corpus luteum. These hormone levels are maintained if pregnancy occurs otherwise they are decreased due to regression of corpus luteum" [17].

Menstruation:

On decrease of hormone levels endometrial layer is not maintained resulting in menstrual flow. Menstrual blood is made up of mostly arterial blood, 25% venous blood, prostaglandins, tissue debris from fibrinolysis of endometrial layer. Menstrual blood flow typically contains no clots unless it is heavy because the fibrinolysis lyses the clot. Blood loss might be as little as a small spotting or as much as 80mL. It's termed abnormal uterine bleeding when there is a blood loss of more than 80mL[17].

FIGO classification of causes of abnormal uterine bleeding- PALM- COEIN:

The abbreviation PALM-COEIN is used to group the following reasons into several categories: "polyp, adenomyosis, leiomyoma, malignancy and hyperplasia, coagulopathy, ovulatory problems, endometrial, iatrogenic, and not otherwise" defined [11]. The PALM is tested visually using histopathology and imaging, whereas the COEIN is assessed structurally.

Polyps:

The endometrial stromal and glands give rise to epithelial proliferations known as endometrial polyps. Most people show no symptoms. From 3.7percent to 65 percent, polyps contribute varying amounts to AUB[18].

Adenomyosis:

Age-related adenomyosis and fibroids may co-exist in certain people. When fibroids are present, adenomyosis can be both localised and diffuse, making the diagnosis more difficult[19].

Malignancy:

Particularly when there is chronic IMB, cervical cancer should be ruled out. Ovarian cancer may very rarely appear with AUB. Although considered to be rare (3-7/100,000 in the USA), uterine sarcoma may contribute to AUB [20].

Coagulopathy:

13% of women presenting with heavy menstrual bleeding are affected with coagulopathy. Most of these ladies are affected with "Von Willebrand disease"[21]. Systemic disorders of hemostasis may be identified in 90% of women using a structured history [22].

Structured history criteria for coagulopathy screening include the following:

1. Significant bleeding ever after menarche

2. Pick one of these:

• Postpartum haemorrhage; • Bleeding resulting from surgery; • Bleeding resulting from dental work [23].

At least two of the following:

Bruises once to twice every month

Epistaxis once to twice every month

bleeding gums

a history of bleeding issues in the family [23].

Ovulatory:

Unopposed oestrogen effects during anovulatory cycles can cause the endometrium to proliferate and thicken significantly, which results in HMB and a changed menstrual cycle frequency. At the most advanced stages of reproductive age, this is seen[11].

Iatrogenic:

Exogenous treatment that can result in unforeseen endometrial bleeding is one of the iatrogenic causes of AUB. Continuous oestrogen or progestin medication (through systemic or intrauterine modes of administration) or therapies that affect ovarian steroid production, such as "gonadotropin-releasing hormone (GnRH) agonists and aromatase inhibitors", are frequently linked to this. Utilizing an IUD may result in low-grade endometritis, which may also lead to AUB [24].

Not otherwise classified:

There will inevitably be pathologies that fall outside of the previously listed categories because they are either uncommon or have a hazy definition. Examples include chronic endometritis (not brought on by an IUD), myometrial hypertrophy, intrauterine pseudoaneurysms, and arteriovenous malformations [11].

Clinical assessment:

The following are steps in clinical assessment for AUB:

1. History

a. Menstrual cycle history:

i. length of menstrual cycles (often 24 days, typically 24–38 days, and rarely >38 days);

ii. Length variability (extended >8 days; typical 4.5–8.0; reduced 4.5);

iii. Calculate the blood loss ("heavy >80 ml; normal 5-80 ml; light 5 ml) and flow rate");

- The frequency of sanitation precautions changes on days with high traffic.
- A change in sanitary protection is required overnight.
- Number and size of blood clots that are expelled.
- An experience of a "flooding" sensation
- Prevalence of Fe^{2+} malnutrition.

b. Effects of symptoms on wellbeing and quality of life:

i. Ask about the effects of the menstrual cycle on social life, such as attendance at work and school, the ability to perform daily tasks, and the effect on personal life.

ii. Mental health like depression and distress.

iii. Desire for health

c. History of sex, reproduction

d. Conditions including hypothyroidism, hyperprolactinemia, polycystic ovarian syndrome, adrenal or hypothalamic problems, which are systemic causes of bleeding

e. Coagulation defects: History of (i) heavy menstrual flow since menarche, (ii) postpartum haemorrhage, (iii) surgery-related bleeding, (iv) dental work-related bleeding, or (iii) two or more of the following: bruising greater than 5 cm once or twice a month, epistaxis once or twice a month, frequent gum bleeding [23]. A referral to haematology colleagues is justified in the presence of a substantial clinical history and abnormalities in investigation results (Table 1)

f. Examine associated symptoms

i. sensations of pressure or pain in the pelvis.

ii. Mood swing

iii. Exhaustion

g. A family history of hereditary clotting disorders, Endometriosis, endometrial or colon cancer, or any additional malignancies, should also be investigated along with any co-morbid diseases, such as cardiovascular problems, hormonally dependent cancers, thromboembolic disease, or other conditions that would affect treatment. [25].

Physical examination:

It is often advised to have a pelvis and abdominal examination to check for pelvic malignancies and other particular disorders [26].

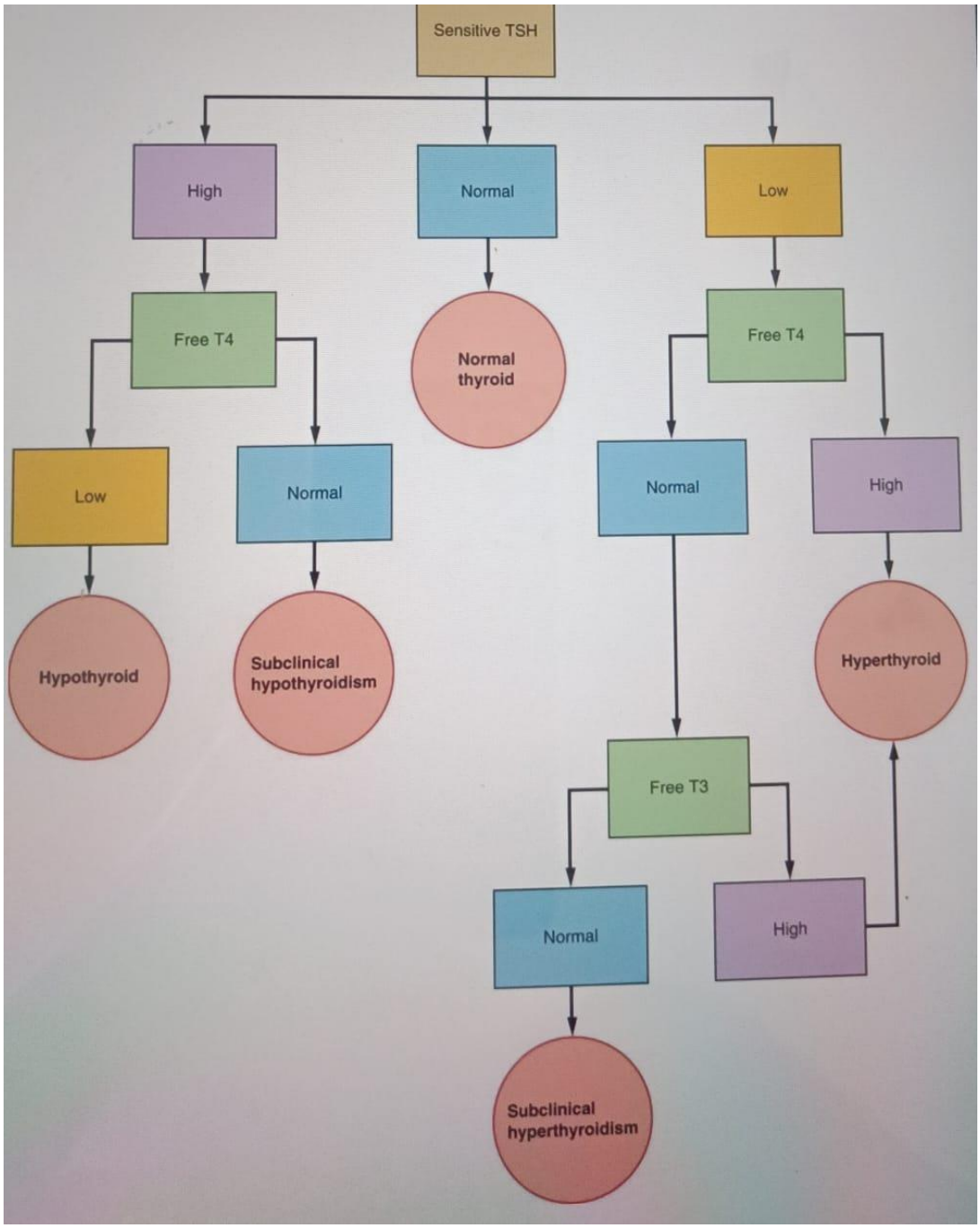
Investigations:

The following table list various investigation for diagnosis of cause for AUB:

Investigation	Rationale
Full Blood count	Exclude anaemia; consider ferritin measurement.
Transvaginal ultrasound scan	• A general evaluation of the uterine size, shape, and presence or absence of adenomyosis, polyps, and fibroids. AUB may be accompanied by uterine malformations such uterine didelphys. • Can visualise appearances that might indicate polycystic ovaries. • Exclude endometrial pathology such as polyps and/or malignancy
MRI	useful for assessing women who may have fibroids preceding myomectomy, fibroid embolization, and potential malignancy.
Hysteroscopy	• Evaluate the endometrial cavity for the presence of polyps and/or endometrial cancer. • Polyp removal and endometrial biopsy can be performed simultaneously[26].
Endometrial sampling	Exclude endometrial hyperplasia and/or cancer
Coagulation disorders screen	• Identify and rule out bleeding disorders, especially in adolescents. • Tests such as the PT, APTT, fibrinogen, and thrombin time to investigate at factor deficiency.

CONFIDENTIAL

By seeing variations of TSH , T3, T4 and bases on levels, thyroid disorders were classified



So variations of TSH and Free T3 and Free T4 levels should be monitored carefully so that exact diagnosis should be made and give proper treatment, prevent unwanted thyroid surgeries

Treatment:

The following table gives treatment options for AUB according to causes of " PALM-COEIN":

AUB sub-classification	specific therapy
Polyp	Resection
Adenomyosis	Surgery: hysterectomy
Malignancy	surgery with adjuvant therapy Progestogens at high doses if surgery is not an option. Palliation (radiotherapy)
Coagulopathy	Tranexamic acid DDVAP (Desmopressin)
Ovulation	Lifestyle modification Cabergoline Levothyroxine
Endometrial	Specific therapies await further delineation of underlying mechanisms
Iatrogenic	Advice for troublesome bleeding with hormonal contraception can be found in the Faculty of Sexual and Reproductive Healthcare Clinical Effectiveness Unit (FSRH CEU) manual.
Not otherwise classified	Antibiotics for endometritis; embolization of AV malformation [26].

CONFIDENTIAL

Surgical treatment:

Hysterectomy or endometrial destruction are the two surgical treatments available in the absence of underlying pathology. Destruction of endometrium is only appropriate if the women has a complete family or uses a highly effective/permanent means of contraception. Both surgical removal of the endometrium (first-generation hysteroscopic procedures) and the Full thickness endometrial necrosis can be caused by the controlled use of energy (second-generation therapies like heat, cold, and microwave) [27].

Thyroid dysfunction:

Definition:

The phrase "thyroid dysfunction," which refers to an impairment in the function of the thyroid gland, covers a range of conditions, from asymptomatic issues to symptomatic thyroid disease. Because of insufficient or absent thyroid hormone production (athyreosis), hypothyroidism is a condition in which the bloodstream has too little thyroid hormone. Hyperthyroidism, on the other hand, is a condition in which the bloodstream has too much thyroid hormone due to an overactive thyroid gland[28].

Based on laboratory results, thyroid dysfunction is classified as either subclinical or overt[29]. TSH, also referred as thyrotropin, is raised in the serum of people with subclinical hypothyroidism but free thyroxine (T4) levels are normal. Conversely, people with overt hypothyroidism have elevated TSH levels in the serum along with subnormal levels of T4[30]. Subnormal serum TSH levels and normal free triiodothyronine (T3) or free T4 levels are indicative of subclinical hyperthyroidism, whereas overt hyperthyroidism is defined as subnormal serum TSH levels and high free triiodothyronine (T3) or free T4 levels [31].

Epidemiology:

One of the main endocrine problems is hypothyroidism. It accounts for between 30 and 40 percent of patients with endocrine problems[32]. According to the American Thyroid Association, more than 12% of US citizens may experience a thyroid issue at some point in their lifetime, and 20 million Americans now have some sort of thyroid illness. Iodine deficiency affects more than a billion people, with Southeast Asia, South America, and Central Africa being the most at risk[33]. Age, gender, race, and location are some variables that may influence the prevalence of thyroid dysfunction. It seems that geographic location affects how much iodine is consumed through food [34].

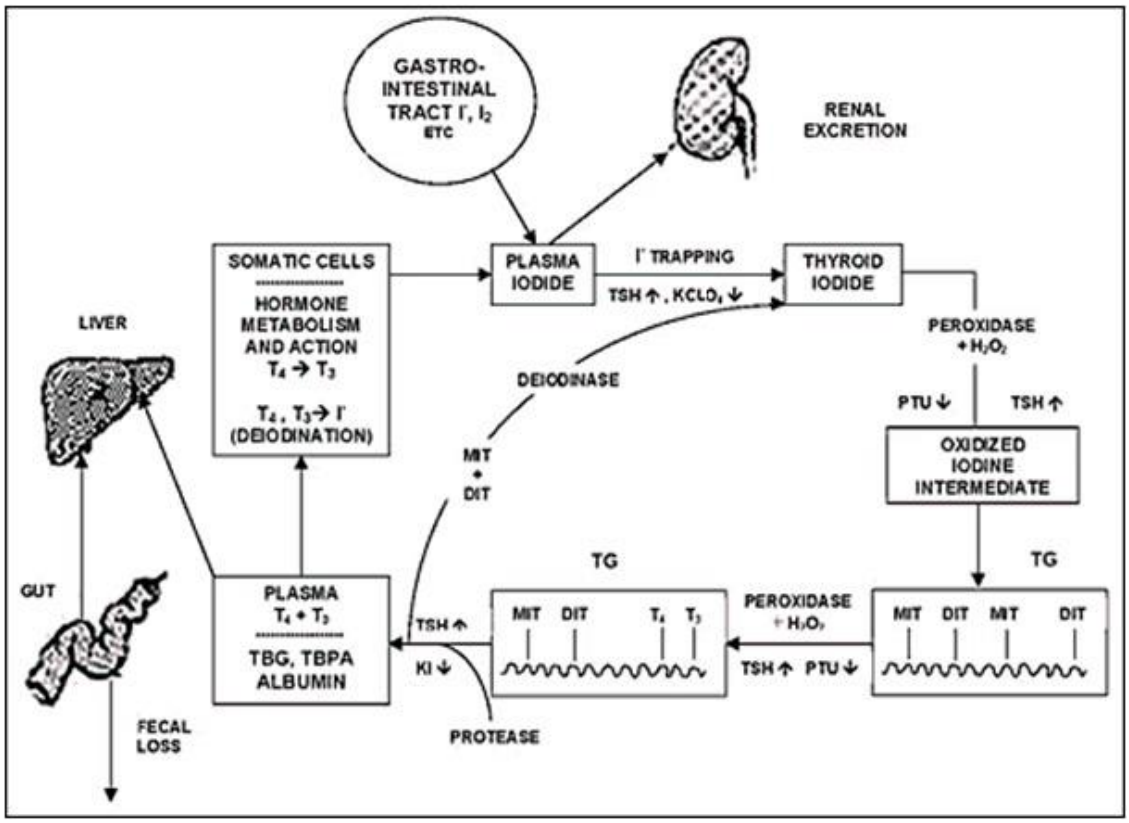
Thyroid gland:

Physiology:

Thyroid gland produces two main hormones namely thyroxine or tetraiodothyronine (T4) and triiodothyronine (T3). Thyroid hormone secretion is regulated by hypothalamus. Thyrotropin releasing hormone is released by hypothalamus into hypothalamic- hypophyseal portal system which on reaching anterior pituitary gland stimulates thyrotropin cells to release TSH. A tropic hormone called thyroid releasing hormone interacts to the anterior pituitary gland's Thyrotropin releasing hormone receptors to activate a signal cascade that is mediated by G-protein coupled receptors. TRH also acts as non-tropic hormone stimulating anterior pituitary lactotrophic cells to produce prolactin. The thyroid hormones are important for many functions of body including growth, development, basal metabolic rate control. Thyroglobulin is produced by thyroid follicular cells and discharged into the follicles as a colloid. Iodine from blood is taken into follicular cells by sodium-iodide cotransporters on basal surface of follicular cells. This iodine is oxidized by thyroid peroxidase. Tyrosine residues on thyroglobulin are iodinated and conjugated via oxidative coupling to form T3 and

$T4^{35}$. 90% of total thyroid hormone is made up of T4 but T3 is 2 to 10 times more bioactive than T4 [36]. The five processes in the production of thyroid hormones are as follows:

- • Thyroglobulin synthesis,
- iodine absorption,
- iodination of thyroglobulin,
- storage
- Release



The following is a list of thyroid secretions' physiological effects:

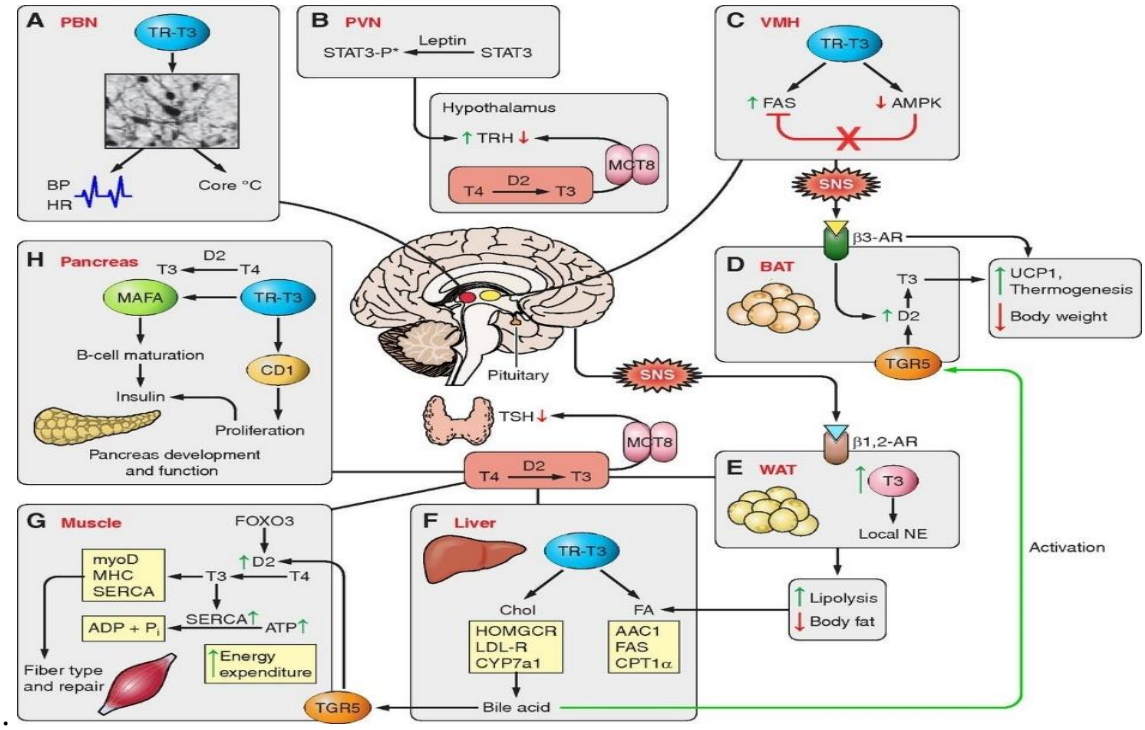
- Boosts the body's basal metabolic rate.
- Depending on the state of the metabolism, it may cause lipid production or lipolysis.
- Encourage the breakdown of carbs
- Protein anabolism . High dosages of thyroid hormones can also cause the catabolism of proteins.
- Tolerable impact on catecholamines.
- To promote bone growth in children, thyroid hormones work in concert with growth hormone.
- Thyroid hormone's effect on the CNS is crucial. It is essential for the brain's development during the prenatal stage. It may impact adult mood. Hyperexcitability and irritability can result from hyperthyroidism. Memory loss, slurred speech, and tiredness can all be symptoms of hypothyroidism.
- Thyroid hormone has an impact on menstruation, ovulation, and fertility.

Thyroid hormone acts through 3 mechanisms:

- Directly at cellular level
- Through sympathetic nervous system.
- Through changing metabolism and affecting circulation and secretion

Regulation of thyroid hormone synthesis:

Thyroid hormone secretion from thyroid gland is regulated by hypothalamic-pituitary axis. "Thyroid stimulating Hormone" is released from pituitary gland on stimulation by Thyroid releasing hormone (TRH) through increase in intracellular cyclic adenosine monophosphate (cAMP) [37]. Dopamine, somatostatin and leptin have modulatory effect on TSH secretion [38]. "Central, nutritional status, circadian rhythms and acute stress modulates thyroid hormone production [39]. Thyroid hormone is produced by thyroid follicular cells on stimulation by thyroid stimulating hormone which binds to G protein coupled "Thyroid stimulating Hormone" receptors. T4 is mainly secreted by thyroid gland [40]. Conversion of T4 to T3 by D2 provides negative feedback to pituitary and hypothalamus [41]. Adequate tissue levels of thyroid hormone reduce TRH and TSH secretion. Serum TSH measurement helps in diagnosis of both hypo and hyperthyroidism due to tight regulation of feedback loop and even small changes in serum T4 are amplified by serum TSH changes"



In the paraventricular nucleus of the hypothalamus, hypophysiotropic neurons generate thyrotropin-releasing hormone (TRH). Pituitary thyrotropin is taken by the hypophyseal portal system from the anterior pituitary's thyrotropes, where it is stimulated by TRH to secrete. “Thyroxine (T4) and triiodothyronine (T3) are released by thyroid follicular cells as a result of TSH's stimulation of these cells”. An overall negative feedback regulation system is necessary to maintain proper thyroid function. Thyroid hormone (T4 and T3) circulating levels therefore affect the production and secretion of TRH in the hypothalamus and TSH in the anterior pituitary (long feedback) [42].

Euthyroid: This is a state of normal thyroid function and normal thyroid hormone levels. In the past, euthyroid disease was thought to be a situation in which a healthy person's normal thyroid function tests represented that person's euthyroid condition, which is a state in which organs and tissues are exposed to the right amount of thyroid hormones. However, numerous studies have demonstrated that each individual's specific TFTs are linked to a unique risk profile of a variety of outcomes, and any alteration to these levels results in certain risks rising and others falling. As a result, it might be challenging to pinpoint the TFT levels that are actually perfect or optimal for any particular person. And even if there were levels that, overall, provided the best likelihood of continuing health for each individual at some point in time, any change in non-thyroid related pathophysiology may later make these levels undesirable.

Thyroid disorders:

Hypothyroidism:

Overt or clinical primary insufficiency is characterized by free thyroxine levels below the permissible limit and thyroid-stimulating hormone (TSH) values above the acceptable range. The criteria for mild or subclinical hypothyroidism, which itself is occasionally considered to be an indication of early thyroid failure, include TSH readings above the suggested levels and free thyroxine concentrations within the normal range**.**

Causes:

There are four different types of hypothyroidism: primary (caused by thyroid hormone insufficiency), secondary (by TSH shortage), tertiary (by thyrotropin-releasing hormone deficiency), and peripheral (by other causes) (extra-thyroidal; panel). Less than 1% of cases of hypothyroidism are either central (including secondary and tertiary hypothyroidism) or peripheral [43].

Primary hypothyroidism:

The most frequent cause of hypothyroidism in locations with insufficient iodine is chronic autoimmune thyroiditis. A crucial part of thyroid hormone is iodine. Iodine shortage can cause hypothyroidism, thyroid nodules, and goitre [44].

Central hypothyroidism:

The symptoms of central hypothyroidism include low or low-to-normal TSH levels and an unusually low level of free thyroxine. TSH levels can occasionally be slightly increased, most likely due to reduced bioactivity [45].

Clinical presentation:

Adult hypothyroidism most frequently manifests in adults as fatigue, apathy, cold sensitivity, weight gain, constipation, change in voice, and dry skin. Clinical effects of hypothyroidism affect practically all major organs.

The following table various clinical presentation symptoms of hypothyroidism:

Organ system	Presentation	Signs and implications
General metabolism	Weight gain, cold intolerance, fatigue	Increase in body-mass index, low metabolic rate, myxoedema[*], hypothermia[46].
Cardiovascular	Fatigue on exertion, shortness of breath	Dyslipidaemia, bradycardia, hypertension, endothelial dysfunction or increased intima-media thickness[*], diastolic dysfunction[*], pericardial effusion[*], electrocardiogram changes[46].
Neurosensory	Hoarseness of voice, decreased taste, vision, or hearing	Neuropathy, cochlear dysfunction, decreased olfactory and gustatory sensitivity
Neurological and psychiatric	Impaired memory, paraesthesia, mood impairment	Impaired cognitive function, delayed relaxation of tendon reflexes, depression[*], dementia[*], ataxia[*], Carpal tunnel syndrome and other nerve entrapment syndromes[*], myxoedema coma[46].
Gastrointestinal	Constipation	Reduced oesophageal motility, non-alcoholic fatty liver disease[*], ascites [46].
Endocrinological	Infertility and subfertility, menstrual disturbance, galactorrhoea.	Goitre, glucose metabolism dysregulation, infertility, sexual dysfunction, increased prolactin, pituitary hyperplasia[46].
Musculo-skeletal	Muscle weakness, muscle cramps, arthralgia	Creatine phosphokinase elevation, Hoffman's syndrome[*], osteoporotic fracture[46].
Haemostasis and haematological	Bleeding, fatigue	Mild anaemia, acquired von Willebrand disease[*], decreased protein C and S[*], increased red cell distribution width[*], increased mean platelet volume
Skin and hair	Dry skin, hair loss	Coarse skin, loss of lateral eyebrows[*], yellow palms of the hand[*], alopecia areata
Electrolytes and kidney function	Deterioration of kidney function	Decreased estimated glomerular filtration rate, hyponatraemia[46].

Diagnosis:

TSH values above the standard range (often 0-4-4-0 mIU/L) and low levels of free thyroxine are considered signs of primary hypothyroidism[29].

Treatment:

Solid preparations of levothyroxine given as a monotherapy on an empty stomach is the recommended treatment for hypothyroidism. In cases of overt hypothyroidism, the ideal daily dose is 1–5–1–8 g per kg of bodyweight 47. Starting doses for patients suffering from coronary artery disease typically range from 12 to 25 g per day, and they should be gradually increased based on symptoms and TSH levels. Younger patients without comorbidities can often receive the full dose immediately away with proper supervision to prevent overtreatment. TSH measurements are performed 4–12 weeks after therapy begins, then every 6 months until they reach a stable level and subsequently yearly. Adjustments should be made in accordance with laboratory results, having in mind that some subjects (e.g., older patients or under- weight) may respond significantly to even small dose changes in serum TSH concentrations. [48]. Coeliac disease, autoimmune atrophic gastritis, and Helicobacter pylori gastritis are gastrointestinal diseases that hinder the absorption of levothyroxine [49]. Normalization of TSH levels and relief from physical and mental symptoms are treatment goals.

Evaluation of therapy:

Adequacy of thyroid hormone replacement therapy can be checked with assessment of TSH levels. The goal is to keep TSH between 0.45 and 2 U/mL, which is the lower part of the normal range. Every patient on thyroid therapy should get TSH levels checked once every year and should use same levothyroxine product for treatment. When TSH level is low then free T4 levels must be measured for adjustment of thyroxine dosage. The changes in TSH and

T4 occur slowly and hence after starting the therapy a minimum time of 8 weeks is required for assessment of TSH.

Osteoporosis and thyroid hormones:

According to the WHO, osteoporosis is characterized by a bone mineral density (BMD) that is 2.5 SDs or more below that of a young adult at any site [50].

Osteoclasts may be affected by thyroid hormones directly, or their impact on resorption of bones may be mediated by osteoblasts or other cell types [51]. A significant contributor to secondary osteoporosis is hyperthyroidism [52]. Adult hypothyroid persons have higher bone density, but because their bones are of worse quality, there may be a greater chance that they will fracture [53]. Thyroid hormone increases bone mineral resorption. Hyperthyroidism causes increase in total and ionized calcium resulting in increase of alkaline phosphatase, serum phosphorus and bone G la protein which is a marker for bone turnover. Increases serum calcium decreases parathyroid hormone resulting in decreased hydroxylation of vitamin D. When there is an excess of thyroid hormone synthesis, all of these result in greater bone resorption and lower bone density.

Subclinical hypothyroidism:

T4 levels that are normal and thyroid stimulating hormone (TSH) levels that are increased are typical signs of subclinical hypothyroidism. Subclinical hypothyroidism is thought to occur between 3 and 15% of the time. Subclinical hypothyroid and hypothyroid share the same etiologies. Subclinical hypothyroidism is asymptomatic most of time [54].

Hyperthyroidism:

When the thyroid hormone generates and secretes an abnormal quantity of thyroid hormone, a known medically as hyperthyroidism develops.

Epidemiology:

Age-related spikes in hyperthyroidism are more common in women. Additionally, it has been noted that the prevalence of mild hyperthyroidism is higher in iodine-deficient areas than in iodine-sufficient ones, and that it has decreased since the implementation of programmes for universal salt iodination [55].

Etiology:

In regions with adequate iodine, Graves' disease is the most typical cause of hyperthyroidism. " Toxic multinodular goitre and solitary toxic adenom"a are two more frequent causes of hyperthyroidism.

Clinical presentation:

Thyroid hormone overproduction has an impact on numerous organ systems. Palpitations, exhaustion, trembling, anxiety, restless sleep, weight loss, heat intolerance, perspiration, and polydipsia are symptoms that are frequently described. Tachycardia, trembling in the extremities, and weight reduction are common physical symptoms [56].

Diagnosis:

To differentiate between overt hyperthyroidism and subclinical hyperthyroidism (having normal levels of circulating hormones), serum TSH concentrations are evaluated, and if they are low, serum free Measured in serum or free T4 indices, and free or total T3 levels should also be tested.[57].

Options in treatment:

The three options for treating hyperthyroidism are radioactive iodine ablation, surgery, and antithyroid drugs (ATDs). All three treatment modalities are effective for treating Graves' disease patients, although surgery or radioactive iodine therapy are the best options for individuals who have toxic neoplasia or toxic multinodular goitre since these patients seldom achieve remission[58].

Association between thyroid dysfunction and AUB:

Around the world, 10-15% of women of reproductive age experience atypical uterine bleeding. The relationship between thyroid problems and AUB may be mediated by changes in thyroid stimulating hormone (TSH) response, rise in prolactin levels, changes in luteinizing hormone (LH) response, changes in peripheral androgen to estrogen conversion, changes in sex hormone binding globulin (SHBG), and changes in coagulation pathways, in addition to effects on lipid profile.[59]

It is well recognized that the thyroid gland is crucial for preserving a regular monthly flow. The existence of thyroid hormone receptors on the ovaries has been reported to have a direct effect on thyroid hormones, while the release of sex hormone binding globulin (SHBG), prolactin, and gonadotropin releasing hormone (GnRH) has been shown to have an indirect effect on thyroid hormones. Both hypothyroidism and hyperthyroidism are associated with anovulatory cycles, delayed puberty, unusually high foetal wastage, and other problems in reproductive function, according to clinical observations. Menorrhagia, or excessive menstrual bleeding, was the most prevalent menstrual abnormality reported in hypothyroid women. To the contrary, oligomenorrhea and hypomenorrhea are more frequently linked to hyperthyroidism. [60].

The effect of hypothyroidism on reproductive health system can be direct by affecting functioning of hypothalamic-pituitary-ovarian axis or indirect by disrupting the operation of the target organs, such as the ovaries. Hyperprolactinemia by decreasing the GnRH release leads to increased levels of FSH with delayed LH response alters the FSH/LH ratio leading to follicular cyst which, in turn, increases the adrenal DHEA leading to arrest in follicle maturation as a result of altered TSH response due to hypothyroidism. Spillover effect of elevated TSH on FSH receptors results in collagen proliferation [61].

Having hypothyroidism causes the amount of SHBG to drop. 2 effects of these include 1) On peripheral conversion of androgen to estrogen , aberrant pituitary feedback that causes a rise in peripheral androgen to oestrogen conversion, and 2) Estradiol and testosterone levels in plasma fall as a result of it. However, there is an increase in oestrogen unbound fractions [62]. Ovarian sensitivity to GnRH is elevated, which causes significant ovarian hypertrophy and the formation of many follicular cysts, which results in PCOS. Additionally, it lowers coagulation factors vii, vii, ix, and xi, leading to significant loss of blood during periods. The earliest clinical symptom of subclinical hypothyroidism is significant menstrual blood loss. [59].

Relevant studies:

Mounika, K.[63] conducted a study that in order to identify thyroid problems in women who had abnormal uterine ibleeding. Subclinical hypothyroidism was the most frequent thyroid condition among patients with hemorrhagic disease.The most typical type of bleeding, reported in 36% of individuals, was menorrhagia. Thyroid illness was found in 32 percent of patients who had subclinical thyroidism in seventeen%, hypothyroid in 11 percent and hyperthyroid in 4% of cases.

Thakur, M., et al.[64] did a study to learn the patient's thyroid status who had irregular uterine bleeding. Euthyroid was common in most females with abnormal uterine flow. Among atypical uterine bleeding patterns, menorrhagia was the most prevalent. Menorrhagia is the most common condition, accounting for 43% of cases, followed by polymenorrhoea (29%), oligomenorrhoea (16.5%), menometrorrhagia (7.6%), metrorrhagia (2.5%), and hypomenorrhea (1.3%).With a mean age of 31 years, the greatest patient population ranged in age from 20 to 25. Seven (8.8%) of those with hypothyroidism had subclinical hypothyroidism, whereas four (5%) had frank hypothyroidism.

Sahu, R., et al.[65] studied about women of reproductive age who have thyroid issues and have unusual uterine bleeding. Menorrhagia was a common menstrual symptom linked to hypothyroidism and subclinical hypothyroidism. In 50% of patients with hyperthyroidism, oligomenorrhea was seen. 20% had a thyroid disorder. 26 of the patients had subclinical hypothyroidism diagnosed, 24 had hypothyroidism, and 6 had hyperthyroidism. Menorrhagia was a common menstrual symptom linked to hypothyroidism and subclinical hypothyroidism. In 50% of patients with hyperthyroidism, oligomenorrhea was seen.

Tara.[66] measured the frequency of thyroid problem in women in Erbil City who had atypical uterine flow. Among women who are of reproductive age, thyroid dysfunction is a prevalent

cause of abnormal uterine bleeding. High levels of thyroid stimulating hormone were significantly correlated with abnormal uterine hemorrhage in females. Inadequate T4 levels were strongly associated with women who had abnormal uterine bleeding. Hypothyroidism and inappropriate uterine hemorrhage were found to be substantially correlated in women.

Singh, S., et al.[67] analyze the menstrual patterns of women with thyroid diseases and the incidence of thyroid disorders in dysfunctional uterine flow. All patients with monthly abnormalities should have their thyroids screened because it is an essential etiological element in menstrual disturbances. The screening of the thyroid would prevent needless surgeries and hormone exposure. As a result, any sort of monthly abnormality should be taken into consideration as a potential presenting symptom of thyroid malfunction and it may even suggest subclinical problem.

Verma, S., et al.[68] evaluated the cyclical and endometrial patterns in women with thyroid issues and to determine the prevalence of thyroid malfunction in dysfunctional uterine haemorrhage. Menorrhagia was the most prevalent menstrual abnormality and was substantially more common in people with thyroid impairment. The study comes to the conclusion that all patients of irregular uterine bleeding should have a biochemical examination of thyroid function performed. This would prevent the need for unneeded procedures and hormone exposure.

Sudha, H, C., et al.[69] detected thyroid issues in people with a tentative diagnosis of AUB (abnormal uterine bleeding). and notify doctors of promising instances so they can be managed further. The study concluded that thyroid disease needs to be taken into account as a significant etiological component for irregular menstruation. To identify both overt and covert thyroid failure, the biochemical examination of T3, T4, and TSH estimations should be made essential in AUB cases.

Phukan, J, K., et al.[70] evaluated the hormonal status in individuals with DUB who appeared to have a normal thyroid function and correlated it with the occurrence of DUB. A prevalent condition among DUB patients is hypothyroidism. To aid in early diagnosis of the etiology and therapy of DUB individuals to prevent surgery, thyroid monitoring should be made mandatory for all people with monthly abnormalities.

Nayak, A, K.[71] studied about thyroid disorders in patients with dysfunctional uterine bleeding. Patients with dysfunctional uterine haemorrhage frequently have thyroid issues, particularly hypothyroidism. To prevent needless hormonal therapy and surgical intervention, a thyroid function test should be performed on all patients with irregular menstruation.

Ajmani et al.[72] studied the prevalence of thyroid disorders and its correlation with menstrual disorders. Twenty percent of individuals with menstrual difficulties had subclinical hypothyroidism, fourteen percent had overt hypothyroidism, and eight percent had overt hyperthyroidism.

Joshi, B, R., et al.[73] studied the frequency of thyroid issues in cases of irregular uterine bleeding that have been diagnosed in patients at an eastern Nepali tertiary hospital. Thirteen percent (7.79%) of all occurrences of abnormal uterine haemorrhage involved thyroid disease.

Sudha Rani, G., et al.[74] In this study, thyroid abnormality affected 44 out of 100 cases. of these, 11% and 20%, respectively, exhibited hypothyroidism and subclinical hypothyroidism. 5% of the cases had hyperthyroidism, and 8% had subclinical hyperthyroidism. The most frequent menstrual disease observed in hypothyroid patients was menorrhagia, which was followed by polymenorrhoea.

Jinger, S, K., et al.[75] studied , at a tertiary care facility in India's northern-western state of Rajasthan, thyroid profile in menstruation problem was examined. In the study among 100 women with menstrual disorders, hypothyroidism in 39, hyperthyroidism in 8 and euthyroid was in 53. Menorrhagia was the most prevalent menstrual abnormality and was substantially more common in people with thyroid impairment. According to the study's findings, thyroid function biochemistry testing should be mandated in all patients of AUB.

Kumari, A., et al. [76] estimated that thyroid impairment is common and is associated with menstruation problems in reproductive age group women. The study found as 41.07% of patients with menstrual disorders also had thyroid conditions, with overt hyperthyroidism in 5.35% of women, 12.5% of patients had overt hypothyroidism, while 17.86% had subclinical hypothyroidism.

Subedi, S., et al. [77] studied, prevalence of thyroid conditions in atypical uterine bleeding and relationship between those conditions and menstrual cycles. The study found that there were 3% more visits to the gynaecological OPD as a result of irregular uterine haemorrhage. and 10.6% of people had thyroid disease, with hypothyroidism being the most prevalent. In DUB, hypothyroidism was more prevalent. So the study recommended that every woman who experiences irregular menstruation should get her thyroid checked; doing so will help her avoid unwanted interventions like inappropriate hormone therapy and surgery.

Lacunae in literature:

Many studies in recent years have shown increased irregular uterine haemorrhage and thyroid problems are frequently linked. Considering the taboo associated with menstrual cycles in India, difficulty of seeking medical care for menstrual problems for majority of women it is necessary to ease diagnosis of causative factor for abnormal uterine bleeding. There are studies available in literature examines the link between irregular uterine haemorrhage and thyroid conditions. But considering the fact that majority of women lack access to proper medical care and resources to manage their menstrual problems this study aims to add on to data available in literature regarding irregular uterine haemorrhage and thyroid problems are frequently linked. This will help in increasing awareness regarding both conditions among women and get appropriate investigations done which in turn will help in appropriate treatment intervention.

MATERIAL & METHODS

MATERIALS AND METHODS

Study site: "Department of Obstetrics & Gynecology RL JALAPPA and Research Center attached to Sri Devaraj Urs Medical College, affiliated to Sri Devaraj Urs Academy of higher Education and Research Tamaka, Kolar- 563101".

Study population: Women with abnormal uterine bleeding who were visiting to "R.L.Jalappa Hospital and Research Center constituent of Sri Devaraj Urs Medical Collage, Tamaka, Kolar were considered as the study population".

Study design: A Prospective observational study

Study period: The data collection was done between January 2021 to August 2022.

METHODOLOGY:-

Inclusion Criteria

- Patients belonging to age group (20-45years) complaining of abnormal uterine bleeding.

• Women with any of the following menstrual disturbances-
Acyclical bleeding
Menorrhagia
Metrorrhagia
Oligomenorrhoea
Polymenorrhoea
Polymenorrhagia
Hypomenorrhoea
Menometrorhagia
• Diagnosed fibroid, polyp, PCOD, endometriosis and malignant tumors

Exclusion Criteria

- Patients who use IUCDs, use medications or hormones, have known thyroid problems, or have a history of bleeding difficulties.
- PID
- Postmenopausal women .
- Patients not willing to give consent

DATA COLLECTION: After receiving informed consent from the patient, data were gathered using a pre-made proforma that matched the study's objectives through personal interviews with the subject. According to the aforementioned criteria, the patient was chosen. After obtaining a thorough menstruation history and asking about the indications and symptoms of hyperthyroid and hypothyroid, the patient underwent the next examination.

A thorough physical examination performed to rule out other potential reasons of abnormal bleeding, with special attention paid to the presence or absence of anaemia, thyroid enlargement, cardiovascular abnormality, abdomen, speculum examination, and pelvic examination work.

Routine tests were performed on all of the patients like hemoglobin count in full, time required for bleeding and bridging to rule out coagulation problems and abdominal pelvic usg)

Then all patients were subjected for T3,T4 and TSH estimation, the morning sample in the fasting state, 5 ml of venous blood will be drawn into a dry, plain glass container without the use of any anticoagulants for the TSH test and T3, T4 estimate.

Following that, the patient was divided into four categories

- Subclinical hypothyroid
- Euthyroid
- hyperthyroid
- Hypothyroid

7.5 Sample size:

The sample size was calculated according to the following formulae

$$Z\alpha^2 Pq/d^2=(1.96)^2 \text{ X}32.4\text{X}67.6/(7.5)^2= 124$$

P=prevelance of thyroid disorder

Q=1-P

With prevelance of thyroid disorder 32.4% at 95% CI with alpha error of 7.5

STATISTICAL METHODS

Thyroid dysfunction and Type of bleeding Were considered as primary outcome variables. Age category, parity, TSH, T3, and T4 were thought to be the main explanatory variables.

For quantitative variables, the mean and standard deviation were used in the descriptive analysis, while frequency and proportion were used for categorical variables. The necessary graphics, such as bar diagrams and pie charts, were also used to illustrate data.

The Mann Whitney u test was used to examine medians and interquartile range (IQR) for quantitative parameters that were not consistently scattered throughout the study groups (2 groups).

Using the Chi square test, categorical outcomes were compared between research groups.

A P value of 0.05 was used to determine statistical significance.

1. BDSS Corp. Released 2020. Co Guide Statistics software, Version 1.0, India: BDSS corp.

RESULTS

RESULTS

The final analysis comprised 124 participants in total.

Table 1: Descriptive analysis of age group in the study population(N=124)

Age in years	Frequency	Percent
<=20	2	1.61%
21-30	51	41.13%
31-40	66	53.23%
>41	5	4.03%

Among the study population, 2 (1.61%) participants were aged <=20 years, 51 (41.13%) were aged between 21 to 30 years, 66 (53.23%) participants were aged between 31 to 40 years and 5 (4.03%) were aged >41 years. (Table 1 & Figure 1)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Age in years	31.23±5.58	32.00	20.00	44.00	30.24	32.21

The mean age was 31.23±5.58, with minimum and maximum 20 and 44 respectively in the study population with 95% C. I (30.24, 32.21). (Table 1)

Figure 1: Bar graph showing distribution of subjects according to age group (N=124)

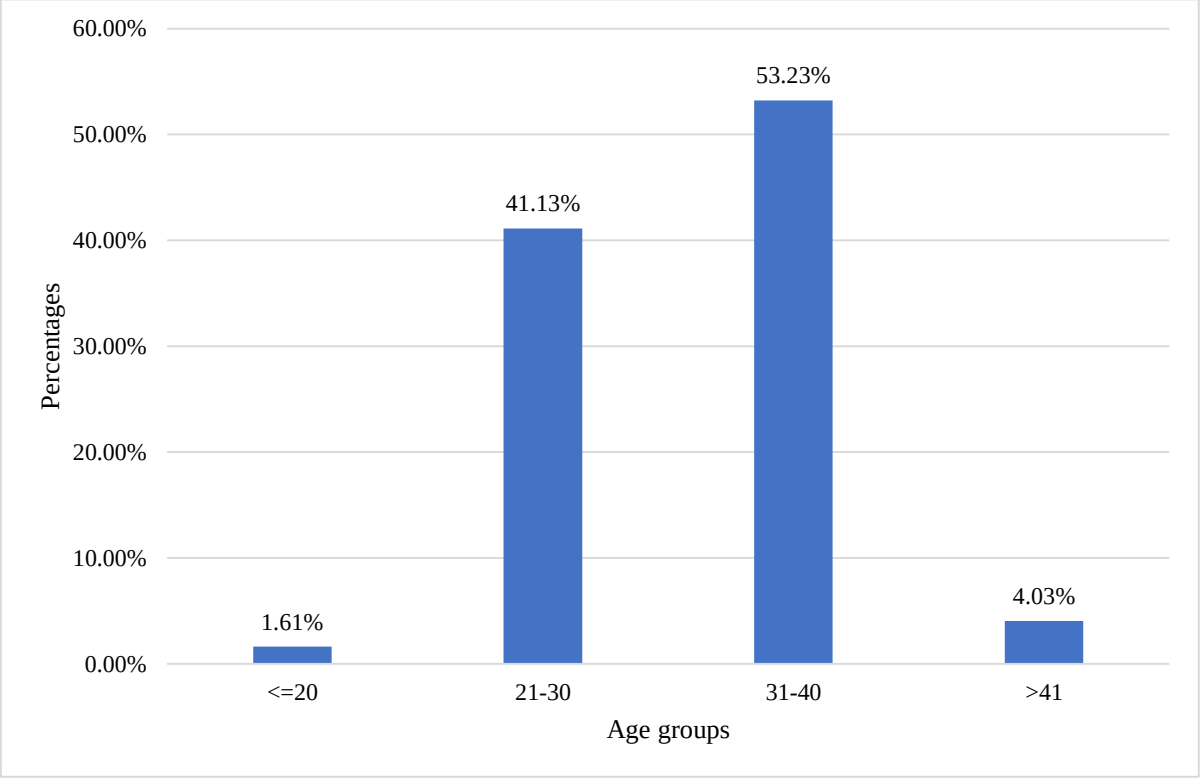


Table 2: Descriptive analysis of parity in the study population (N=124)

Parity	Frequency	Percentage
Nulli	5	4.03%
Para 1	20	16.13%
Multi	87	70.16%
Unmarried	12	9.68%

Among the study population, 5 (4.03%) were nulli, 20 (16.13%) were Para 1, 87 (70.16%) were multi and 12 (9.68%) were unmarried. (Table 2 & Figure 2)

Figure 2: Bar graph showing distribution of subjects according to parity (N=124)

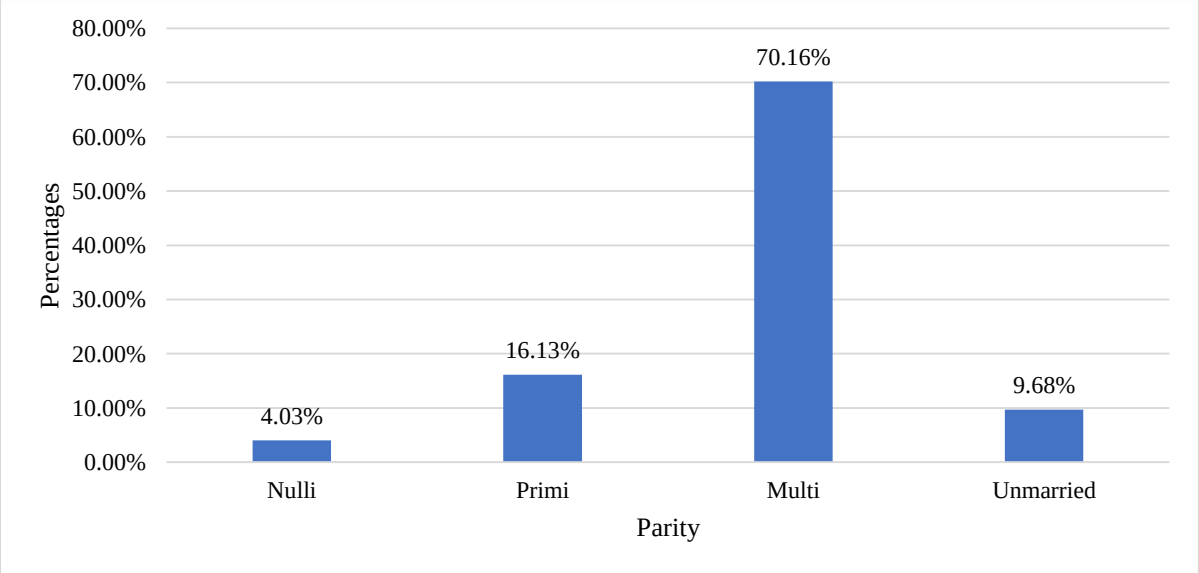


Table 3: Descriptive analysis of duration in the study population (N=124)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Duration of menstruation (in days)	5.99±3.03	7.00	1.00	10.00	5.46	6.53

The mean Duration of menstruation (in days) was 5.99±3.03, with minimum and maximum 1 and 10 respectively in the study population with 95% C. I (5.46, 6.53). (Table 3)

Table 4: Descriptive analysis of Cycle length (in days) in the study population (N=124)

Cycle length (in days)	Frequency	Percentage
20 to 40	83	66.94%
41 to 60	27	21.77%
61 to 80	1	0.81%
81 & above	9	7.26%
Irregular	4	3.23%

Among the study population, 83 (66.94%) participants had a cycle length of 20 to 40 days, 27 (21.77%) participants had a cycle length of 41 to 60 days, 1 (0.81) participant had 61 to 80 days, 9 (7.26%) participants had a cycle length (in days) of 81 & above and for 4 (3.23%) participants it was irregular. (Table 4)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Cycle length in days	37.48±18.72	30.00	20.00	90.00	34.13	40.83

Note: 4 members are irregular.

The mean Cycle length in days was 37.48±18.72, with minimum and maximum 20 and 90 respectively in the study population with 95% C. I (34.13, 40.83).

Table 5: Descriptive analysis of Type of bleeding in the study population (N=124)

Type of bleeding	Frequency	%
Menorrhagia	47	37.90%
Oligomenorrhoea	24	19.35%
Acyclical	19	15.32%
Polymenorrhagia	17	13.71%
Polymenorrhoea	8	6.45%
Hypomenorrhea	5	4.03%
Metrorrhagia	4	3.23%

Among the study population, 47 (37.90%) participants had menorrhagia, 24 (19.35%) had oligomenorrhoea, 19 (15.32%) were acyclical, 17 (13.71%) had polymenorrhagia, 8 (6.45%) had polymenorrhoea, 5 (4.03%) had hypomenorrhea and 4 (3.23%) participants had metrorrhagia. (Table 5 & Figure 3)

Figure 3: Bar chart of Type of bleeding in the study population (N=124)

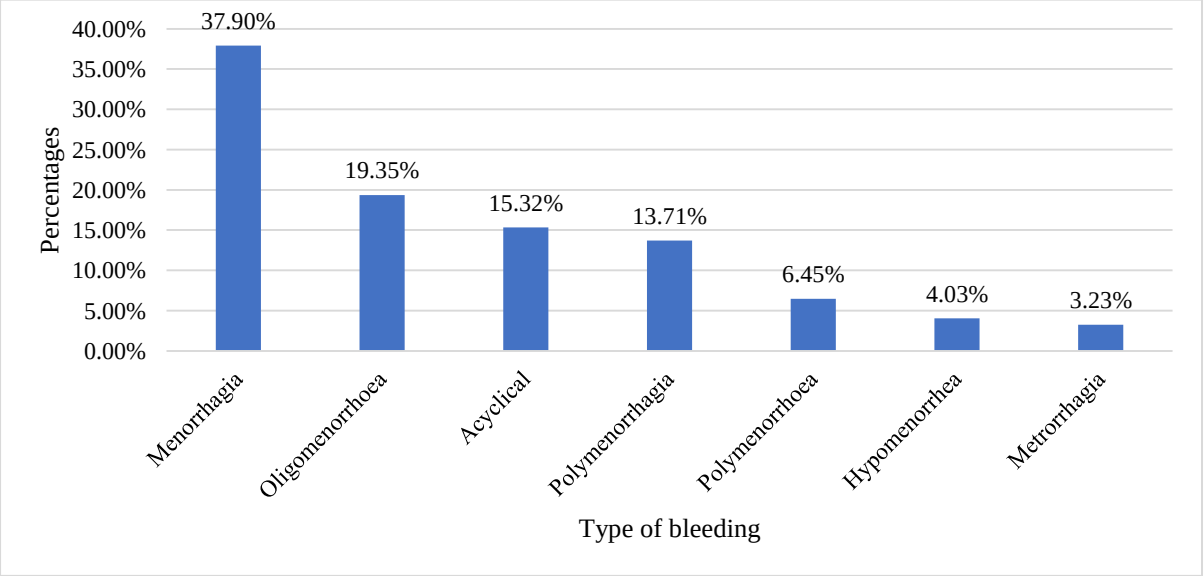


Table 6: Descriptive analysis of T3 ng/ml in the study population (N=124)

T3 ng/ml	Frequency	Percentage
<0.7	14	11.29%
0.7 - 2.0	103	83.06%
>2.0	7	5.65%

Among the study population, 14 (11.29%) participants had <0.7 T3, 103 (83.06%) had T3 between 0.7 to 2.0 and 7 (5.65%) had >2.0 T3. (Table 6)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
T3 ng/ml	1.35±0.57	1.36	0.23	3.61	1.25	1.45

The mean T3 ng/ml was 1.35±0.57, with minimum and maximum 0.23 and 3.61 respectively in the study population with 95% C. I (1.25, 1.45). (Table 6)

Table 7: Descriptive analysis of T4 mcg/dl in the study population (N=124)

T4 mcg/dl	Frequency	Percentage
<=4.5	13	10.48%
4.6 – 12	104	83.87%
>12	7	5.65%

Among the study population, 13 (10.48%) participants had <4.5 T4, 104 (83.87%) had T4 between 4.6 to 12 and 7 (5.65%) had >12 T4. (Table 7)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
T4 mcg/dl	8.51±3.67	8.40	1.40	30.50	7.86	9.16

The mean T4 mcg/dl was 8.51±3.67, with minimum and maximum 1.40 and 30.50 respectively in the study population with 95% C. I (7.86, 9.16). (Table 7)

Table 8: Descriptive analysis of Thyroid Stimulating Hormone mciu/ml in the study population (N=124)

Thyroid Stimulating Hormone mciu/ml	Frequency	Percentage
<=0.39	8	6.45%
0.4 - 4.2	83	66.94%
4.3 – 50	22	17.74%
>50	11	8.87%

Among the study population, 8 (6.45%) participants had <=0.39 TSH, 83 (66.94%) had TSH between 0.4 to 4.2, 22 (17.74%) had TSH between 4.3 to 50 and 11 (8.87%) had TSH >50. (Table 8)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Thyroid Stimulating Hormone mciu/ml	10.73±19.68	2.58	0.03	92.10	7.27	14.20

The mean Thyroid Stimulating Hormone mciu/ml was 10.73±19.68, with minimum and maximum 0.03 and 92.10 respectively in the study population with 95% C. I (7.27, 14.20). (Table 8)

Table 9: Descriptive analysis of Thyroid dysfunction in the study population (N=124)

Thyroid dysfunction	Frequency	%
Euthyroid	84	67.74%
Hypothyroid	13	10.48%
Subclinical hypothyroid	20	16.13%
Hyperthyroid	7	5.65%
Total thyroid dysfunction	40	32.26%

Among the study population, 84 (67.74%) had Euthyroid, 13 (10.48%) had hypothyroid, 20 (16.13%) had subclinical hypothyroid, 7 (5.65%) had hyperthyroid and 40 (32.26%) had total thyroid dysfunction (Table 9 & Figure 4)

Figure 4: Bar chart of Thyroid dysfunction in the study population (N=124)

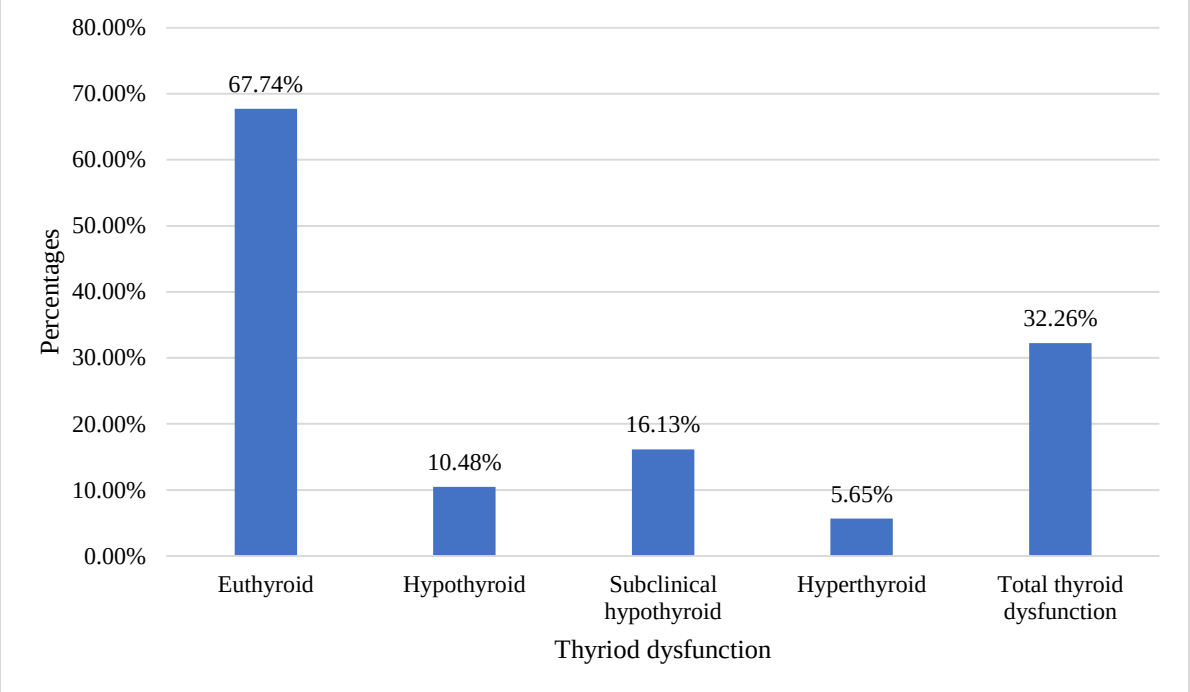


Table 10: Descriptive analysis of Fibroid in the study population (N=124)

Fibroid	Frequency	Percentage
Present	8	6.45%
Absent	116	93.55%

Among the study population, 8 (6.45%) had Fibroid. (Table 10)

Table 11: Descriptive analysis of Adenomyosis in the study population (N=124)

Adenomyosis	Frequency	Percentage
Present	2	1.61%
Absent	122	98.39%

Among the study population, 2 (1.61%) had Adenomyosis. (Table 11)

Table 12: Descriptive analysis of Polycystic Ovarian disease in the study population (N=124)

Polycystic Ovarian Disease	Frequency	Percentage
Present	3	2.42%
Absent	121	97.58%

Among the study population, 3 (2.42%) had Polycystic Ovarian Disease. (Table 12)

Table 13: Descriptive analysis of POLYP in the study population (N=124)

POLYP	Frequency	Percentage
Present	2	1.61%
Absent	122	98.39%

Among the study population, 2 (1.61%) had POLYP. (Table 13)

Table 14: Comparison of Thyroid Stimulating Hormone, T3, T4 with Type of bleeding in the study population (N=124)

Parameter	Type of bleeding Mean ± SD						P Value
	Menorrhagia (N=49)	Oligomenorrhoea (N=26)	Polymenorrhagia (N=16)	polymenorrhoea (N=7)	Hypomenorrhea (N=5)	Acyclical (N=21)	
TSH mciu/ml	10.44 ± 18.55	9.90 ± 17.25	7.84 ± 17.24	6.38 ± 6.13	2.19 ± 1.16	18.13 ± 29.32	0.4648
T3 ng/ml	1.34 ± 0.43	1.58 ± 0.88	1.29 ± 0.37	1.09 ± 0.29	1.66 ± 0.23	1.16 ± 0.50	0.0729
T4 mcg/dl	8.50 ± 2.55	9.84 ± 6.29	8.06 ± 2.06	8.60 ± 1.77	8.53 ± 1.85	7.20 ± 2.93	0.2750

The mean TSH was 10.44 ± 18.55 in menorrhagia, it was 9.90 ± 17.25 in oligomenorrhoea, it was 7.84 ± 17.24 in polymenorrhagia, it was 6.38 ± 6.13 in polymenorrhoea, it was 2.19 ± 1.16 in hypomenorrhea and it was 18.13 ± 29.32 in acyclical. There is no significant difference in TSH among type of bleeding in the study population. (P value 0.4648)

The mean T3 was 1.34 ± 0.43 in menorrhagia, it was 1.58 ± 0.88 in oligomenorrhoea, it was 1.29 ± 0.37 in polymenorrhagia, it was 1.09 ± 0.29 in polymenorrhoea, it was 1.66 ± 0.23 in hypomenorrhea and it was 1.16 ± 0.50 in acyclical. There is no significant difference in T3 among type of bleeding in the study population. (P value 0.0729)

The mean T4 was 8.50 ± 2.55 in menorrhagia, it was 9.84 ± 6.29 in oligomenorrhoea, it was 8.06 ± 2.06 in polymenorrhagia, it was 8.60 ± 1.77 in polymenorrhoea, it was 8.53 ± 1.85 in hypomenorrhea and it was 7.20 ± 2.93 in acyclical. There is no significant difference in T4 among type of bleeding in the study population. (P value 0.2750) (Table 14)

Table 15: Comparison of Thyroid dysfunction with parity in the study population (N=124)

Parity	Thyroid dysfunction						P value
	Hypothyroid	**Subclinical hypothyroid**	**Hyperthyroid**	**Total thyroid dysfunction**	**Euthyroid**	**Chi square**	
Nulli (N = 5)	2 (40.00%)	1 (20.00%)	0 (0.00%)	**3 (60%)**	**2 (40.00%)**	2.06	0.56
Para 1 (N = 20)	2 (10.00%)	5 (25.00%)	0 (0.00%)	**7 (35%)**	**13 (65.00%)**		
Multi (N = 87)	7 (8.05%)	12 (13.79%)	7 (8.05%)	**26 (29.89%)**	**61 (70.11%)**		
Unmarried (N = 12)	2 (16.67%)	2 (16.67%)	0 (0.00%)	**4 (33.33%)**	**8 (66.67%)**		

Among Para 1, 2 (10%) had hypothyroid, 13 (65%) had euthyroid, 5 (25%) had subclinical hypothyroid. Among multi 7 (8.05%) had hypothyroid, 61 (70.11%) had euthyroid, 12 (13.79%) had subclinical hypothyroid, 7 (8.05%) had hyperthyroid. Among unmarried, 2 (16.67%) had hypothyroid, 8 (66.67%) had Euthyroid, 2 (16.67%) had subclinical hypothyroid. It was not statistically significant how thyroid dysfunction varied in parity. (P = 0.56). (Figure 5) and Table 15

Figure 5: Stacked bar chart for Comparison of Thyroid dysfunction with parity in the study population (N=124)

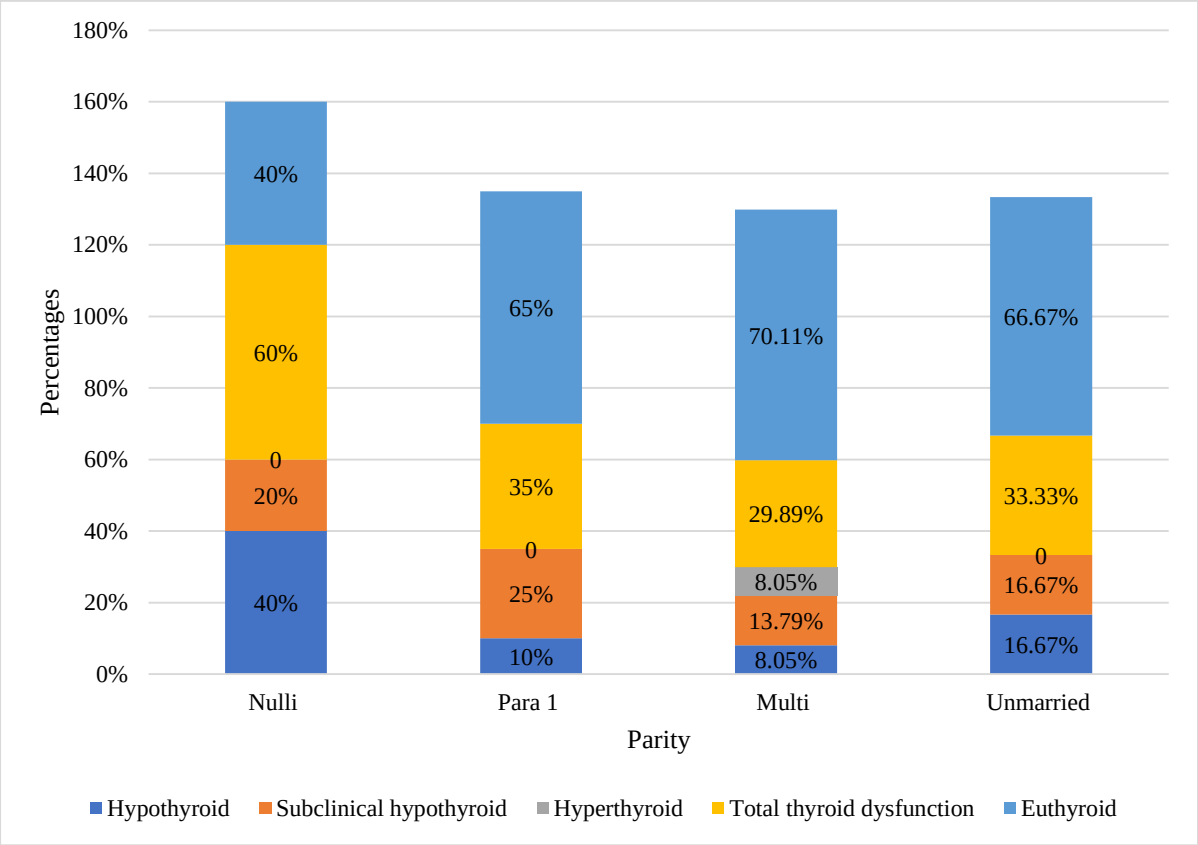


Table 16: Comparison of Type of bleeding with Age groups in the study population (N=124)

Age groups	Type of bleeding						
	Menorrhagia	**Metrorrhagia**	**Oligomenorrhea**	**Polymenorrhagia**	**Polymenorrhea**	**Hypomenorrhea**	**Acyclical**
<=20 (N = 2)	1 (50.00%)	0 (0.00%)	1 (50.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)
21 - 30 (N = 51)	22 (43.14%)	0 (0.00%)	10 (19.61%)	5 (9.80%)	4 (7.84%)	2 (3.92%)	8 (15.69%)
31 - 40 (N = 66)	23 (34.85%)	4 (6.06%)	12 (18.18%)	11 (16.67%)	4 (6.06%)	3 (4.55%)	9 (13.64%)
>41 (N = 5)	1 (20.00%)	0 (0.00%)	1 (20.00%)	1 (20.00%)	0 (0.00%)	0 (0.00%)	2 (40.00%)

Among <=20 years, 1 (50%) had menorrhagia, 1 (50%) had oligomenorrhea. Among 21 to 30 years, 22 (43.14%) had menorrhagia, 10 (19.61%) had oligomenorrhoea, 5 (9.80%) had polymenorrhagia, 4 (7.84%) had polymenorrhea, 2 (3.92%) had hypomenorrhea, 8 (15.69%) were acyclical. Among 31 to 40 years, 23 (34.85%) had menorrhagia, 12 (18.18%) had oligomenorrhoea, 11 (16.67%) had polymenorrhagia, 4 (6.06%) had polymenorrhea, 3 (4.55%) had hypomenorrhea, 9 (13.64%) were acyclical. Among >41 years, 1 (20%) had menorrhagia, 1 (20%) had oligomenorrhoea, 1 (20%) had polymenorrhagia, 2 (40%) were acyclical. (Table 16 & Figure 6)

Figure 6: Stacked bar chart for Comparison of Type of bleeding with Age groups in the study population (N=124)

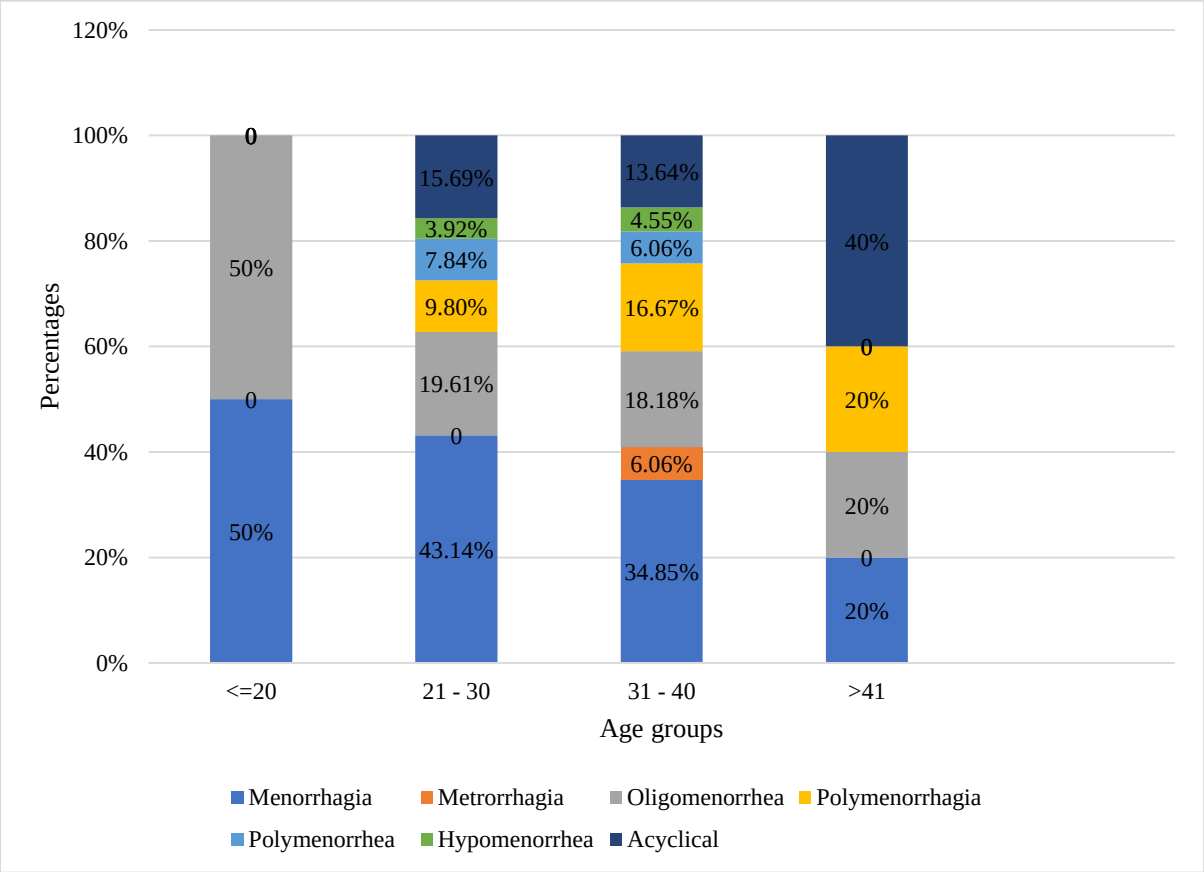


Table 17: Comparison of Thyroid dysfunction with Age groups in the study population (N=124)

Age groups	Thyroid dysfunction					Chi square	P value
	Hypothyroid	Subclinical hypothyroid	Hyperthyroid	Total thyroid dysfunction	Euthyroid		
<=20 (N = 2)	1 (50.00%)	0 (0.00%)	0 (0.00%)	**1 (50%)**	**1 (50.00%)**	3.91	0.2716
21-30 (N = 51)	8 (15.69%)	10 (19.61%)	1 (1.96%)	**19 (37.25%)**	**32 (62.75%)**		
31-40 (N = 66)	3 (4.55%)	9 (13.64%)	5 (7.58%)	**17 (25.76%)**	**49 (74.24%)**		
>41 (N = 5)	1 (20.00%)	1 (20.00%)	1 (20.00%)	**3 (60%)**	**2 (40.00%)**		

Among <=20 years, 1 (50%) had hypothyroid, 1 (50%) had euthyroid. Among 21 to 30 years, 8 (15.69%) had hypothyroid, 32 (62.75%) had euthyroid, 10 (19.61%) had subclinical hypothyroid, 1 (1.96%) had hyperthyroid. Among 31 to 40 years, three (4.55%) had hypothyroid, one (20%) had subclinical hypothyroidism, one (20%) had hyperthyroidism, and 49 (74.24%) had euthyroidism. There was no statistically significant age gap among thyroid problem cases. (P = 0.2716) (Figure 7) and Table 17

Figure 7: Stacked bar chart for Comparison of Thyroid dysfunction with Age groups in the study population (N=124)

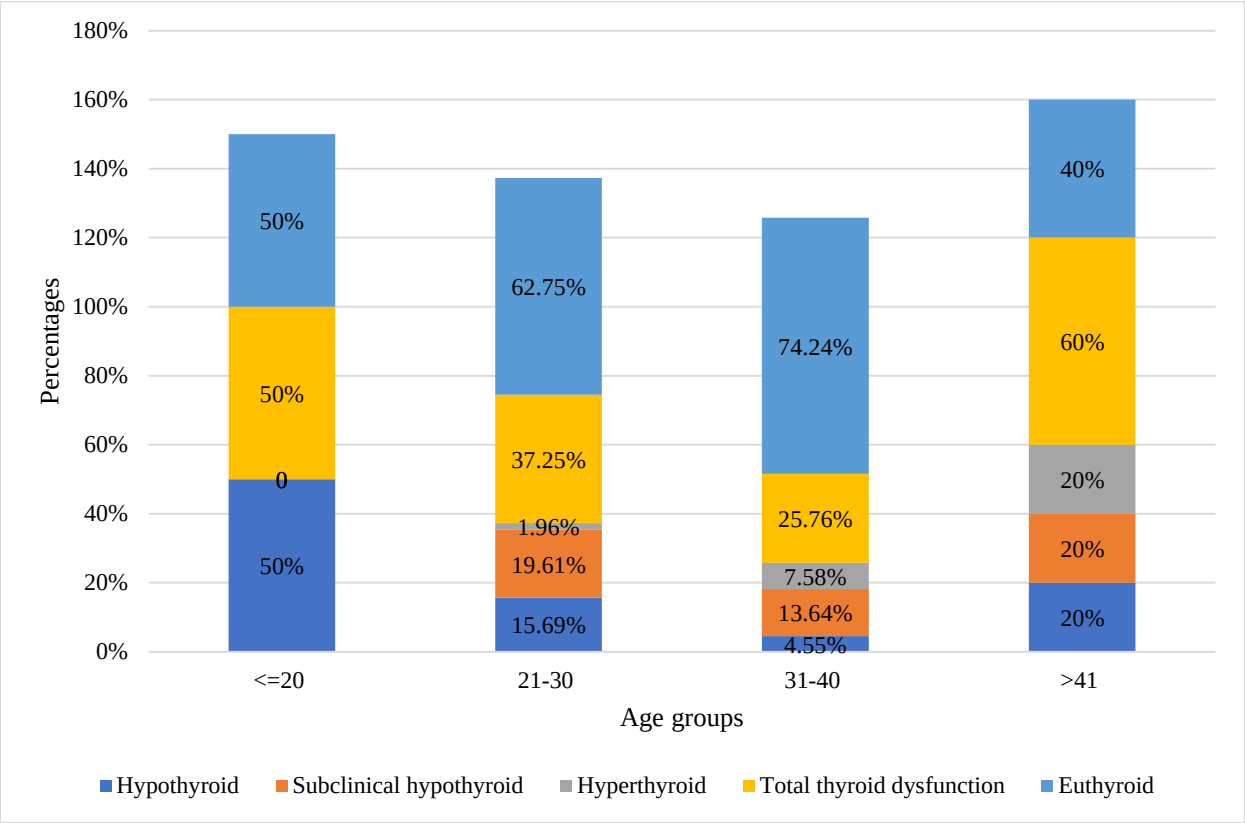


Table 18: Comparison of Thyroid dysfunction with Type of bleeding in the study population (N=124)

Type of bleeding	Thyroid dysfunction				
	Hypothyroid	**Sub clinical hypothyroid**	**Hyperthyroid**	**Total thyroid dysfunction**	**Euthyroid**
Menorrhagia (N = 47)	4 (8.51%)	11 (23.40%)	1 (2.13%)	**16 (34.04%)**	**31 (65.96%)**
Metrorrhagia (N = 4)	0 (0.00%)	0 (0.00%)	0 (0.00%)	**0**	**4 (100.00%)**
Oligomenorrhea (N = 24)	5 (20.83%)	2 (8.33%)	6 (25.00%)	**13 (54.17%)**	**11 (45.83%)**
Polymenorrhagia (N = 17)	1 (5.88%)	1 (5.88%)	0 (0.00%)	**2 (11.76%)**	**15 (88.24%)**
Polymenorrhea (N = 8)	0 (0.00%)	3 (37.50%)	0 (0.00%)	**3 (37.50%)**	**5 (62.50%)**
Hypomenorrhea (N = 5)	0 (0.00%)	0 (0.00%)	0 (0.00%)	**0**	**5 (100.00%)**
Acyclical (N = 19)	3 (15.79%)	3 (15.79%)	0 (0.00%)	**6 (31.57%)**	**13 (68.42%)**

Among menorrhagia, 4 (8.51%) had hypothyroid, 31 (65.96%) had euthyroid, 11 (23.40%) had subclinical hypothyroid, 1 (2.13%) had hyperthyroid. Among metrorrhagia, 4 (100%) had euthyroid. Among Oligomenorrhoea, 5 (20.83%) had hypothyroid, 11 (45.83%) had euthyroid, 1 (5.88%). Among polymenorrhea, 5 (62.50%) had euthyroid, 3 (37.50%) had sub clinical hypothyroid. Among hypomenorrhea, 5 (100%) had euthyroid. Among acyclical, 3 (15.79%) had hypothyroid, 13 (68.42%) had euthyroid, 3 (15.79%) had sub clinical hypothyroid. (Table 18 & Figure 8)

Figure 8: Stacked bar chart for Comparison of Thyroid dysfunction with Type of bleeding in the study population (N=124)

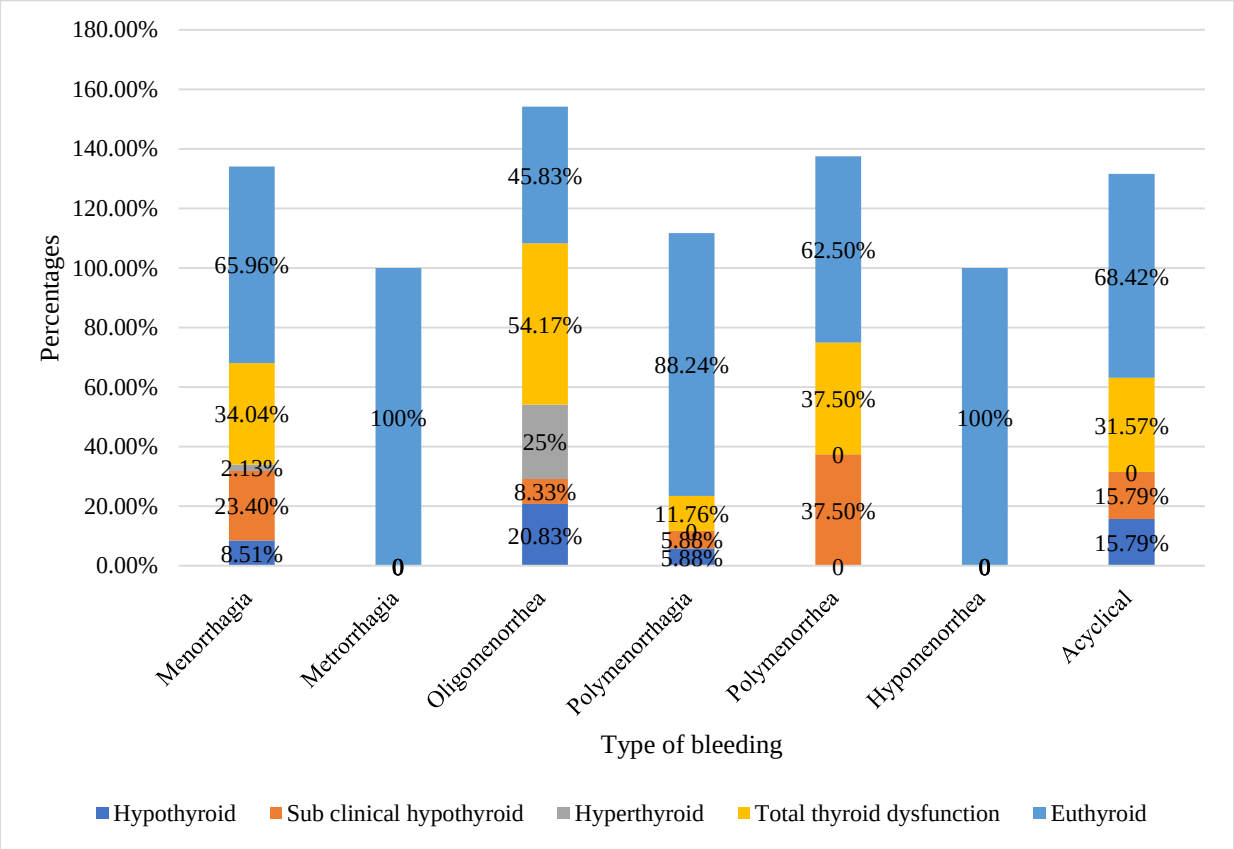


Table 19: Comparison of Type of bleeding with T3 ng/ml in the study population (N=124)

T3 ng/ml	Type of bleeding						
	Menorrhagia	**Metrorrhagia**	**Oligomenorrhea**	**Polymenorrhagia**	**Polymenorrhea**	**Hypomenorrhea**	**Acyclical**
<0.7 (N = 14)	5 (35.71%)	0 (0.00%)	5 (35.71%)	1 (7.14%)	0 (0.00%)	0 (0.00%)	3 (21.43 %)
0.7 - 2.0 (N = 103)	41 (39.81%)	4 (3.88%)	13 (12.62%)	16 (15.53%)	8 (7.77%)	5 (4.85%)	16 (15.53 %)
>2.0 (N = 7)	1 (14.29%)	0 (0.00%)	6 (85.71%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)

*No test is applied due to 0 subjects in the cells.

Among <0.7 T3 5 (35.71%) had menorrhagia, 5 (35.71%) had oligomenorrhea, 1 (7.14%) had polymenorrhagia, 3 (21.43%) were acyclical. Among 0.7 – 2.0 T3, 41 (39.81%) had menorrhagia, 4 (3.88%) had metrorrhagia, 13 (12.62%) had oligomenorrhea, 16 (15.53%) had polymenorrhagia, 8 (7.77%) had polymenorrhea, 5 (4.58%) had hypomenorrhea, 16 (15.53%) were acyclical. Among >2.0 T3 1 (14.29%) had menorrhagia, 6 (85.71%) had oligomenorrhea. (Table 19 & Figure 9)

Figure 9: Comparison of Type of bleeding with T3 ng/ml in the study population (N=124)

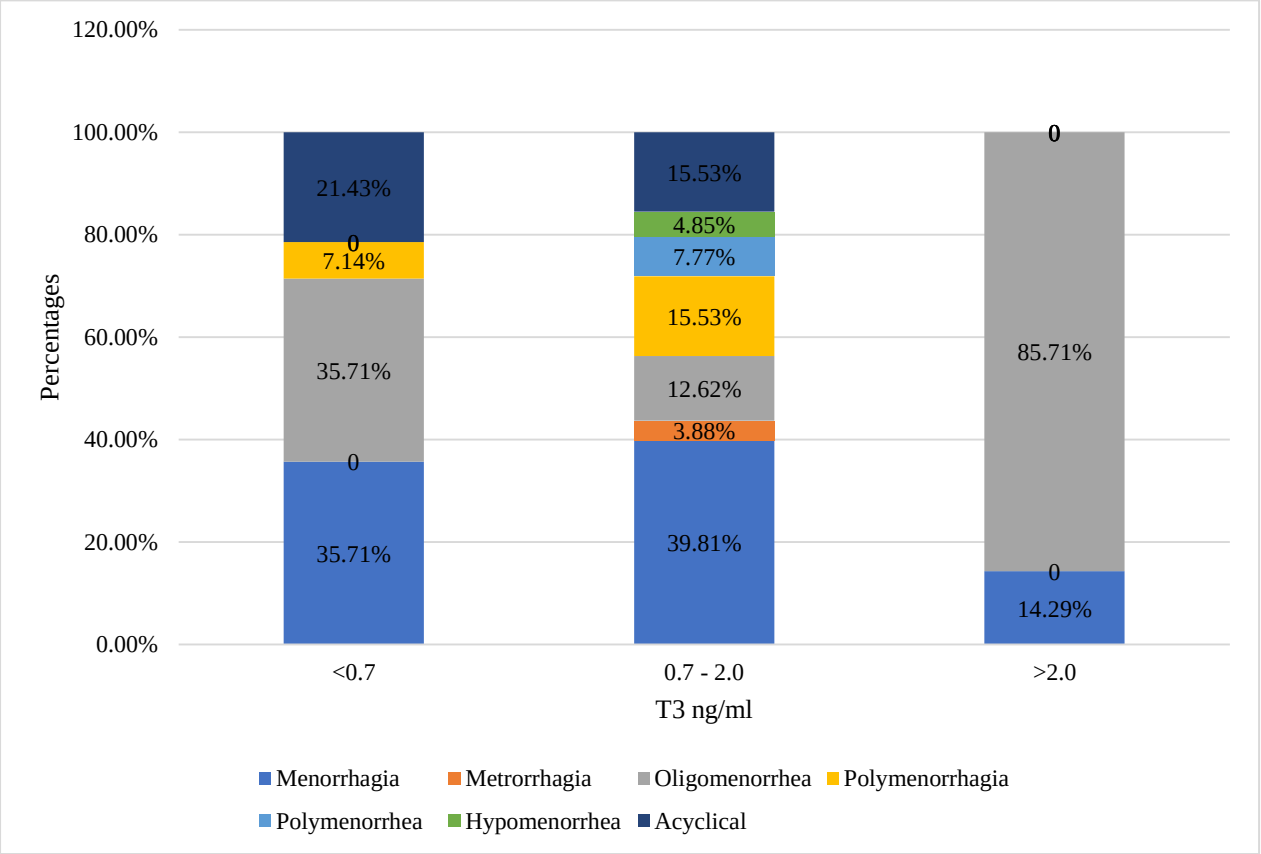


Table 20: Comparison of Type of bleeding with T4 mcg/dl in the study population (N=124)

T4 mcg/dl	Type of bleeding						
	Menorrhagia	**Metrorrhagia**	**Oligomenorrhea**	**Polymenorrhagia**	**Polymenorrhea**	**Hypomenorrhea**	**Acyclical**
<=4.5 (N = 13)	4 (30.77%)	0 (0.00%)	5 (38.46%)	1 (7.69%)	0 (0.00%)	0 (0.00%)	3 (23.08%)
4.6 - 12 (N = 104)	42 (40.38%)	4 (3.85%)	13 (12.50%)	16 (15.38%)	8 (7.69%)	5 (4.81%)	16 (15.38%)
>12 (N = 7)	1 (14.29%)	0 (0.00%)	6 (85.71%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)

*No test is applied due to 0 subjects in the cells.

Among <=4.5 T4, 4 (30.77%) had menorrhagia, 5 (38.46%) had oligomenorrhea, 1 (7.69%) had polymenorrhagia, 3 (23.08%) were acyclical. Among 4.6 – 12 T4, 42 (40.38%) had menorrhagia, 4 (3.85%) had metrorrhagia, 13 (12.50%) had oligomenorrhea, sixteen (15.38%) experienced polymenorrhagia, eight (7.69%) had polymenorrhea, five (4.81%) had hypomenorrhea and 16 (15.38%) were acyclical. Among >12 T4 1 (14.29%) had menorrhagia, 6 (85.71%) had oligomenorrhea. (Table 20 & figure 10)

Figure 10: Stacked bar chart for Comparison of Type of bleeding with T4 mcg/dl in the study population (N=124)

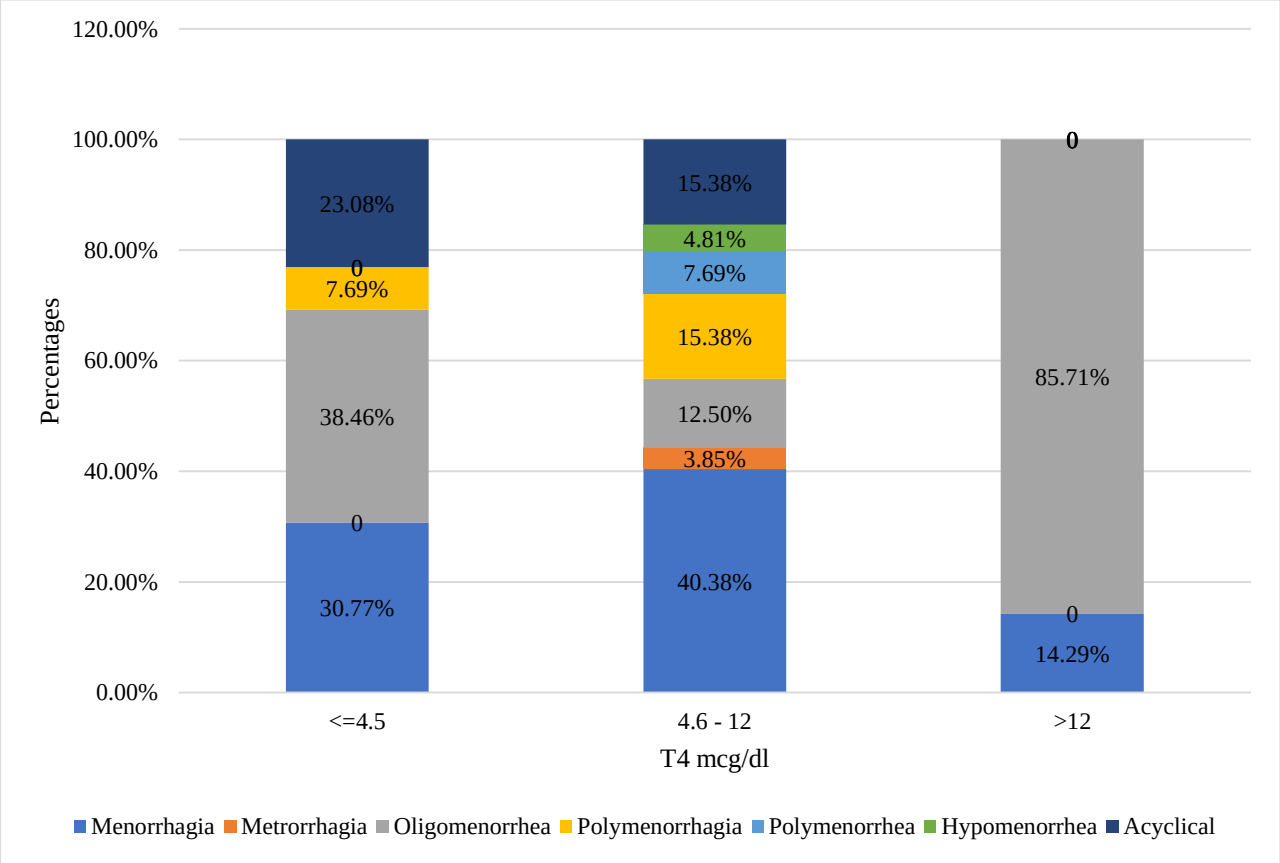


Table 21: Comparison of Type of bleeding with Thyroid stimulating hormone in the study population (N=124)

Thyroid stimulating hormone	Type of bleeding						
	Menorrhagia	Metrorrhagia	Oligomenorrhea	Polymenorrhagia	Polymenorrhea	Hypomenorrhea	Acyclical
<=0.39 (N = 8)	1 (12.50%)	0 (0.00%)	6 (75.00%)	0 (0.00%)	0 (0.00%)	1 (12.50%)	0 (0.00%)
0.4 - 4.2 (N = 83)	31 (37.35%)	4 (4.82%)	11 (13.25%)	15 (18.07%)	5 (6.02%)	4 (4.82%)	13 (15.66%)
4.3 - 50 (N = 22)	11 (50.00%)	0 (0.00%)	4 (18.18%)	1 (4.55%)	3 (13.64%)	0 (0.00%)	3 (13.64%)
>50 (N = 11)	4 (36.36%)	0 (0.00%)	3 (27.27%)	1 (9.09%)	0 (0.00%)	0 (0.00%)	3 (27.27%)

Among <=0.39 TSH, 1 (12.50%) had menorrhagia, 6 (75%) had oligomenorrhea, 1 (12.50%) had hypomenorrhea. Among 0.4- 4.2 TSH 31 (37.35%) had menorrhea, 4 (4.82%) had metrorrhagia, 11 (13.25%) had oligomenorrhea, 15 (18.07%) had polymenorrhagia, 5 (6.02%) had polymenorrhea, 4 (4.82%) had hypomenorrhea, 13 (15.66%) were acyclical. Among 4.3 – 50 TSH, 11 (50%) had menorrhagia, 4 (18.18%) had oligomenorrhea, 1 (4.55%) had polymenorrhagia, 3 (13.645) had polymenorrhea, 3 (13.64%) were acyclical. Among >50 TSH, 4 (36.36%) had menorrhea, 3 (27.27%) had oligomenorrhea, 1 (9.09%) had polumenorrhagia, 3 (27.27%) were acyclical. (Table 21 & Figure 11)

Figure 11: Stacked bar chart for Comparison of Type of bleeding with Thyroid stimulating hormone in the study population (N=124)

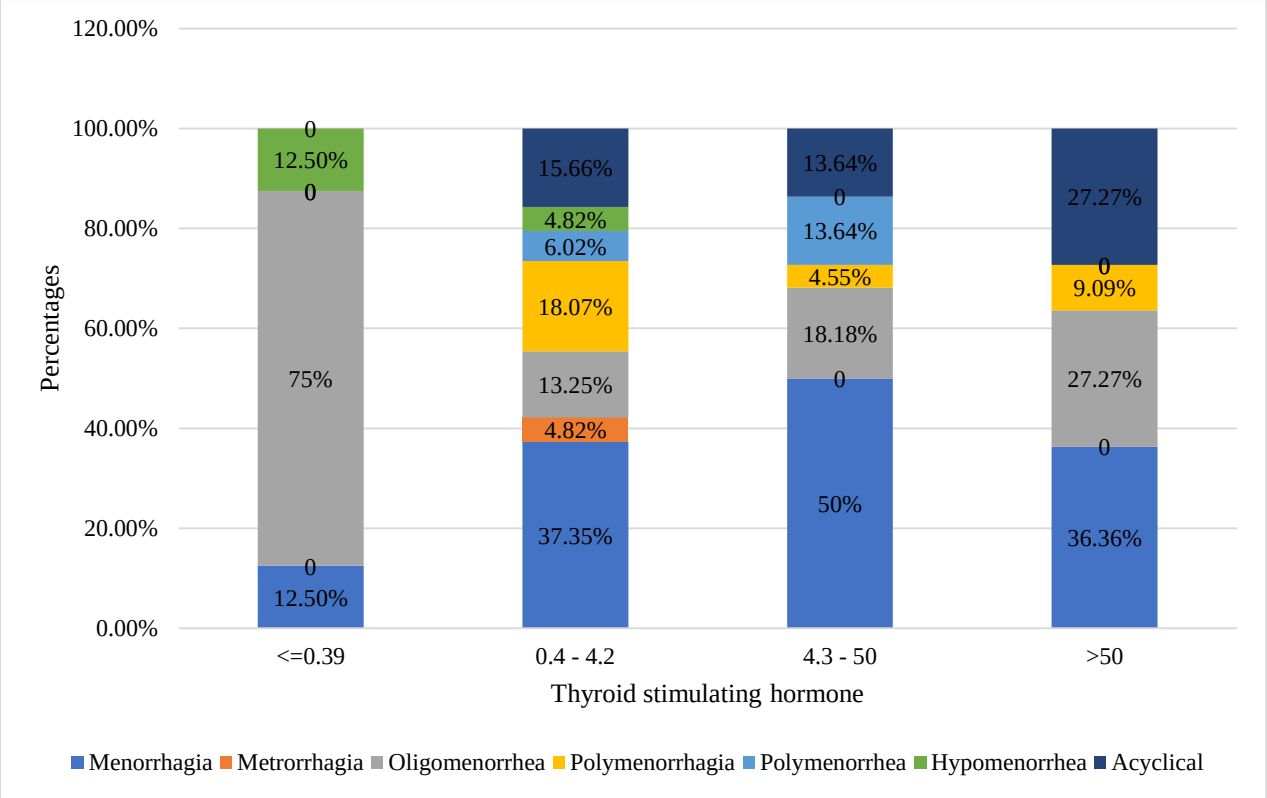


Table 22: Comparison of Thyroid dysfunction with structural abnormalities in the study population(N=124)

Structural abnormalities	Thyroid dysfunction					Chi square	P value
	Hypothyroid	Subclinical hypothyroid	Hyperthyroid	Total thyroid dysfunction	Euthyroid		
Fibroid							
Present (N = 8)	1 (12.50%)	2 (25.00%)	0 (0.00%)	**3 (37.5%)**	**5 (62.50%)**	0.11	0.7120
Absent (N = 116)	12 (10.34%)	18 (15.52%)	7 (6.03%)	**37 (31.90%)**	**79 (68.10%)**		
Adenomyosis							
Present (N = 2)	0 (0.00%)	1 (50.00%)	0 (0.00%)	**1 (50%)**	**1 (50.00%)**	0.29	0.5429
Absent (N = 122)	13 (10.66%)	19 (15.57%)	7 (5.74%)	**39 (31.97%)**	**83 (68.03%)**		
Polycystic Ovarian Disease							
Present (N = 3)	0 (0.00%)	1 (33.33%)	0 (0.00%)	**1 (33.33%)**	**2 (66.67%)**	0	1.00
Absent (N = 121)	13 (10.74%)	19 (15.70%)	7 (5.79%)	**39 (32.23%)**	**82 (67.77%)**		
POLYP							
Present (N = 2)	0 (0.00%)	0 (0.00%)	0 (0.00%)	**0**	**2 (100.00%)**	*	*
Absent (N = 122)	13 (10.66%)	20 (16.39%)	7 (5.74%)	**40 (32.79%)**	**82 (67.21%)**		

Among fibroid, 5 (62.50%) had euthyroid, 2 (25%) had subclinical hypothyroid. Among adenoma, 1 (50%) had euthyroid, 1 (%) had subclinical hypothyroid. Among Polycystic Ovarian Disease, 2 (66.67%) had euthyroid and among POLYP 2 (100%) had euthyroid. The difference in structural abnormalities among thyroid dysfunction was not statistically significant. (P value >0.05) (Table 22)

Table 23: Comparison of Type of bleeding with structural abnormalities in the study population(N=124)

structural abnormalities	Type of bleeding						
	Menorrhagia	**Metrorrhagia**	**Oligomenorrhea**	**Polymenorrhagia**	**Polymenorrhea**	**Hypomenorrhea**	**Acyclical**
Fibroid							
Present (N = 8)	7 (87.50%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	1 (12.50%)	0 (0.00%)	0 (0.00%)
Absent (N = 116)	40 (34.48%)	4 (3.45%)	24 (20.69%)	17 (14.66%)	7 (6.03%)	5 (4.31%)	19 (16.38%)
Adenomyosis							
Present (N = 2)	2 (100.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)
Absent (N = 122)	45 (36.89%)	4 (3.28%)	24 (19.67%)	17 (13.93%)	8 (6.56%)	5 (4.10%)	19 (15.57%)
Polycystic Ovarian Disease							
Present (N = 3)	0 (0.00%)	0 (0.00%)	2 (66.67%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	1 (33.33%)
Absent (N = 121)	47 (38.84%)	4 (3.31%)	22 (18.18%)	17 (14.05%)	8 (6.61%)	5 (4.13%)	18 (14.88%)
POLYP							
Present (N = 2)	0 (0.00%)	2 (100.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)
Absent (N = 122)	47 (38.52%)	2 (1.64%)	24 (19.67%)	17 (13.93%)	8 (6.56%)	5 (4.10%)	19 (15.57%)

* Due to a lack of volunteers in the cells, no test is conducted.

Among fibroid, 7 (87.50%) had menorrhagia, among adenomyosis, 2 (100%0 had) menorrhagia, among Polycystic Ovarian Disease, 2 (66.67%) had Oligomenorrhea and among POLYP, 2 (100%) had Metrorrhagia. (Table 23)

DISCUSSION

DISCUSSION

This Prospective observational study was conducted in women with abnormal uterine bleeding who were visiting to R.L.Jalappa Hospital and Research Center constituent of Sri Devaraj Urs Medical Collage, Tamaka, Kolar. It is estimated that about one-third of women experience abnormal uterine bleeding (AUB) in their life with an global estimated prevalence of 3 to 30% [78]. AUB causes poor health related quality of life for women affecting their social, emotional and mental health. 41% of women believe that there are no treatment options available for AUB and do not seek medical care. Women most of the times do not seek medical consultation thinking it is a waste of time or fearing that surgical intervention is the only treatment option available. Many also do not seek medical care fearing social embarrassment. The cause of AUB is most of the times misdiagnosed adding to the agony of women [79]. Thyroid hormones are found to affect menstrual cycle either through direct impact on ovaries or through impact on sex hormone binding globulin, prolactin and gonadotrophin releasing hormone [8]. Many studies have established association between thyroid dysfunction and AUB. Detecting and treating thyroid function shows improvement in AUB cases. Thyroid disorders are one of the most common endocrine disorders prevalent world-wide and in India. Thyroid diseases are different from other diseases in terms of ease of diagnosis and accessible treatment options. There is widespread availability of thyroid function testing in recent years. When the association between thyroid dysfunction and AUB is strongly established many clinicians will opt for thyroid testing for women consulting for AUB. Hence this study was conducted to estimate the association between thyroid dysfunction with AUB in a tertiary hospital.

Thyroid dysfunction and Type of bleeding Were considered as primary outcome variables. Age group, parity, TSH, T3, T4 were considered as Primary explanatory variables. A total of 124 subjects were included in the final analysis.

Age pattern in menstrual disturbances

The mean age was 31.23±5.58, with minimum and maximum ages being 20 and 44 years respectively. Majority (53.23%) of participants were aged between 31 to 40 years. AUB is found to be more common in the early 40s and late 30s[3] which correlates with this observation. This observation of majority of participants in the age group of 31 to 40 years is similar to that found in a study by Narula et al.[80] in which about 32.8% of patients belonged to the age group 31-40 years and in another study by by Sangeeta Pahwa et al.[81] about 42% of cases belonged to this age group and in another study by Chaitra, M., et al.[82] 42.5% belonged to the age group of 31 - 40 years. This observation is slightly different from that found in two other similar studies in which majority of participants belonged to age group 41 to 50 years. In study by Verma,S,K., et al.[68], most of the AUB patients were in the age group of 41 to 50 years (42.50%) followed by 31 to 40 years (38.50%) and in another study by N Bhavani et al.[83] most of the AUB patients were in the age group of 41 to 50 years (40%) followed by 31 to 40 years (37%).

Parity in menstrual disturbances

Among study participants majority (70.16%) were multiparous. AUB is usually found in women having 3 or more children. This observation is similar to that reported in a study by Ali,J., et al. in which majority of the AUB cases had a parity of >2 and in another study by Singh P., et al.[84] in which majority of participants with AUB were multiparous. In other similar studies including study by Thakur, M., et al.[64], Gowri,M., et al.[85] majority of the cases had a parity of 2. Among the study population, (37.90%) participants had menorrhagia. This observation of menorrhagia being reported in majority of participants is similar to that found in studies by Chaitra,M., et al.[82], Thakur, M., et al.[64], Verma SK et al.[68]., Deshmukh PY et al.[4] all of which had majority of patients with complaint of menorrhagia.

Menorrhagia in thyroid disorders

Among the study participants (67.74%) were Euthyroid, (10.48%) had hypothyroidism, (16.13%) had subclinical hypothyroidism, (5.65%) had hyperthyroidism and (32.26%) had total thyroid dysfunction. In study by Verma, S,K.,et al.[68] 79.55%patients were euthyroid. 19.5% patients were hypothyroid, in a study by Sowers et-al.[86] 90.4% were euthyroid, 6.2% hypothyroid and 3.2% hyperthyroid in perimenopausal age group. Kaur et al.[87] observed in their study that 85% of the patients with abnormal uterine bleeding were euthyroid, 14% hypothyroid and 1% hyperthyroid. In study by Thakur, M., et al.[64] 15.1% cases of AUB had thyroid dysfunction out of which (13.9%) had hypothyroidism and 1 (1.3%) had hyperthyroidism. Among hypothyroid cases 7 (8.8%) had subclinical and 4 (5.06%) had overt hypothyroidism. In a study by Joshi, B,R., et al.[73] 15.79% of total cases of AUB had thyroid dysfunction. Rest 84.21% of total cases of AUB were euthyroid. Out of cases with thyroid dysfunction, hypothyroid was most common followed by subclinical hypothyroid and hyperthyroid. In a study by Kumar AHS et al.[88] out of 200 cases 162 (81 %) cases were euthyroid, (19%) cases had thyroid dysfunction out of which (16.5%) were hypothyroid and (2.5%) were hyperthyroid. In another study done by Gowri M et al.[85] out of 170 cases, 132 (77.6%) cases were euthyroid, 30 (17.6%) of cases had hypothyroidism and 8 (4.7%) had hyperthyroidism. In another study done by Singh P., et al.[84] out of 400 cases, 65% were euthyroid, 26% had hypothyroid, and 9% had hyperthyroidism. In the study carried out by Kattel et al.[89] thyroid dysfunction was present in 20% of abnormal uterine bleeding cases out of which 19% had hypothyroidism and 1% had hyperthyroidism. In the study done by Komathi R et al.[90] about 30% of abnormal uterine bleeding had thyroid dysfunction out of which 27% had hypothyroid and 3% had hyperthyroidism.

The following table gives percentage of AUB cases having euthyroid, hypothyroidism and hyperthyroidism from similar studies:

Study Showing % of cases having	euthyroid	hypothyroidism	hyperthyroidism	subclinical hypothyroidism
Present study	67.74%	10.48%	5.65%	16.18%
Verma, S,K.,et al.[68]	79.55%	19,5%	-	-
Sowers et-al	90.4%	6.2%	3.2%	-
Kaur et al.[87]	85%	14%	1%	-
Kumar AHS et al.[88]	81%	16.5%	2.5%	10.5%
Thakur, M., et al.[64]	84.9%	13.9%	1.3%	8.8%
Joshi, B,R., et al.[73]	84.21%	60%1	13.33%	26.66%
Gowri M et al.[85]	77.6%	17.6%	4.7%	5
Singh P., et al.[84]	65%	26%	9%	-
Kattel et al.[89]	80%	19%	1%	-
Komathi R et al.[90]	70%	27%	3%	-

From above table observations it is noted that the percentage of AUB cases having euthyroid is less in the present study in comparison with other similar studies suggesting higher percentage of AUB cases having some kind of thyroid dysfunction in the study. Around 32% of AUB cases are found to be associated with some kind of thyroid dysfunction in the study.

Subclinical hypothyriodism

Among study participants (16.13%) had subclinical hypothyroidism. This observation is higher than that observed in similar studies. In study by Thakur,M., et al. 8.8% had subclinical hypothyroidism, in study by Kumar AHS et al.[88] 10.5% AUB cases had subclinical hypothyroidism, in study by Chaitra,M., et al.[82], prevalence of subclinical hypothyroidism was 11%.

Among study participants detected with hypothyroidism majority of them (20.83%) had oligomenorrhoea and among study participants detected with subclinical hypothyroidism majority of them (37.50%) had polymenorrhoea followed menorrhagia reported in (23.40%) participants. This observation is contrary to that found in similar studies including study by Thakur, M., et al.[64], study by Kumar AHS et al.[88] , study by Singh P., et al.[84], study by Kattel et al.[89], study by Komathi R et al.[90] all of which reported menorrhagia in majority of participants. Similar to the observation in this study Gowri M et al.[85] reported oligomenorrhoea as the most common bleeding disorder in their study.

Table comparing AUB among thyroid dysfunction individuals(n=) across different studies to present study

Present study				
Type of bleeding	Hypothyroid (n)	Hyperthyroid (n)	Euthyroid (n)	Subclinical Hypothyroidism (n)
Menorrhagia (n=47)	4	1	31	11
Metrorrhagia (n=4)	0	0	4	0
Oligomenorrhea (n=24)	5	6	11	2
Polymenorrhea(n=8)	0	0	5	3
Polymenorrhagia (N = 17)	1	0	15	1
Hypomenorrhea (n=5)	0	0	5	0
Acyclical (n=19)	3	0	13	3
Patil, A et al[91] study				
	Hypothyroid (n)	**Hyperthyroid (n)**	**Euthyroid (n)**	**Subclinical (n)**
Menorrhagia (n=62)	5	0	49	8
Metrorrhagia (n=5)	0	0	05	0
Oligomenorrhea(n=13)	0	2	11	0
Polymenorrhea(n=13)	0	0	12	1
Polymenorrhagia (n=27)	0	0	26	1
Acyclical (n=20)	0	0	20	0

T3, T4 and TSH

Among the study population, (6.45%) participants had <=0.39 TSH, (66.94%) had TSH between 0.4 to 4.2, (17.74%) had TSH between 4.3 to 50 and 11 (8.87%) had TSH >50. The normal TSH levels for adults 21 to 99 years old is estimated to be in the range 0.27 – 4.2 mcIU/mL Majority of study participants have very low levels of TSH and other 17.74% also have just normal levels of TSH. Four of the 17 patients in the Patil, A et al[91] research who had thyroid problems had aberrant T3 levels, two of whom had low T3 values and two of whom had levels that were above the normal range. In Verma, Set al[92] study in two cases, T4 levels were found to be low, while in three cases, T4 levels were found to be high. This observation suggests that TSH screening test can be used for detection of thyroid dysfunction in women with abnormal uterine bleeding as it is cheap and easy to perform.

SUMMARY

SUMMARY:

A total of 124 women, clinically diagnosed as abnormal uterine bleeding, from R L Jalappa Hospital and research institute, in a study period of January 2021 to august 2022 were studied.

Study was aimed to evaluate and detect thyroid dysfunction in patients with provisional diagnosis of AUB and patients showing thyroid dysfunction were referred to physician for further management.

1. Majority (53.23%) of participants with AUB were aged between 31 to 40 years.
2. Among study participants, majority of patients with AUB were multiparous (70.16%).
3. Majority of women in our study were belong to Euthyroid (67.74%)
4. In present study, (32.26%) had total thyroid dysfunction.
5. Among the study participants of thyroid dysfunction, (16.13%) had subclinical hypothyroidism, (10.48%) had hypothyroidism, (5.65%) had hyperthyroidism
6. Thyroid dysfunction was commonest in cases with oligomenorrhoea (54.17%) followed by polymenorrhoea (37.53%), menorrhagia (34.04%), acyclical bleeding (31.57%), polymenorrhagia(11.76%)
7. Predominant thyroid dysfunction was Subclinical hypothyroidism (16.13%) followed by hypothyroidism (10.48%). Less cases presented with hyperthyroidism (5.65%)
8. 25% cases of Hyperthyroidism were oligomenorrhea and only 2.13% cases were menorrhagia
9. Subclinical hypothyroidism was maximum among polymenorrhoeic women (37.50%) and followed in menorrhagic women (23.40%)

10. Oligomenorrhoea was seen in patients with TSH value less than 0.39 mcIU/ml or when TSH value greater than 50 mcIU/ml
11. Out of 40 cases of thyroid dysfunction 100 % cases showed abnormal TSH levels and 48.52 % cases showed abnormal T3 and T4 levels. Thus TSH screening test can be used for detection of thyroid dysfunction in women with abnormal uterine bleeding as it is cheap and easy to perform and helps in correct diagnosis of AUB etiology.

CONCLUSION

CONCLUSION:

1. In our study thyroid dysfunction was noted in 32% of women with abnormal uterine bleeding, of which most common was subclinical hypothyroidism in 16.13%, followed by hypothyroidism in 10.48% and hyperthyroidism in 5.65 %
2. Menorrhagia (34.06%) followed by oligomenorrhea were commonest menstrual abnormalities. In both hypothyroidism and hyperthyroidism, oligomenorrhoea was the commonest menstrual abnormality. The most common bleeding disorder observed in participants detected with hypothyroidism was oligomenorrhoea. In hyperthyroidism, the most common menstrual abnormality was oligomenorrhea followed by menorrhagia. In subclinical hypothyroidism, polymenorrhea followed by menorrhagia was the commonest menstrual abnormality
3. In present study structural causes for abnormal uterine bleeding were noticed in very low percentage of participants. Poly cystic ovarian disease was found only in 2.42% and polyps and **a**denomyosis were reported in 1.61% which is insignificant and may or may not associated with thyroid hormonal dysfunction
4. In patients with thyroid dysfunction, almost all cases showed abnormal TSH levels and half of thyroid dysfunction cases showed abnormal T3 and T4 levels. This suggests that TSH screening test can be used for detection of thyroid dysfunction in women with abnormal uterine bleeding as it is cheap and easy to perform and helps in correct the thyroid dysfunction by proper treatment so that avoid unnecessary surgery and hormonal treatment. So biochemical evaluation of T3, T4 and TSH estimation should be made obligatory in abnormal uterine bleeding cases, to detect thyroid dysfunction

LIMITATION

LIMITATIONS

The Limitation of this study was done in a tertiary care hospital on a small sample size and hence requires data from other multicenter based large sample studies for generalization of results. This study is one of the few studies reported from this state on this important aspect.The knowledge about thyroid dysfunction to prevent menstrual disturbances is necessary to prevent unnecessary surgical interventions or hormonal therapy for abnormal uterine bleeding

RECOMMENDATIONS

This study emphasized that thyroid function test for all the women visiting for abnormal uterine bleeding as association of thyroid dysfunction with abnormal uterine bleeding is more in the study. Thyroid function test and subsequent treatment for the detected thyroid disorder will prevent unnecessary surgical interventions or hormonal therapy for abnormal uterine bleeding.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Liu Z, Doan Q V, Blumenthal P, Dubois RW. A Systematic Review Evaluating Health-Related Quality of Life, Work Impairment, and Health-Care Costs and Utilization in Abnormal Uterine Bleeding. Value Heal. 2007;10(3):183–94.

2. Munro MG, Critchley HOD, Broder MS, Fraser IS. FIGO classification system (PALM-COEIN) for causes of abnormal uterine bleeding in nongravid women of reproductive age. Int J Gynaecol Obstet Off Organ Int Fed Gynaecol Obstet. 2011;113(1):3–13.

3. Shah J V, Pandya MJ, Prajapati PB, Senta DB, Patel MK. An analytical study of abnormal uterine bleeding in women of child bearing age group. Int J Reprod Contraception, Obstet Gynecol Vol 10, No 8 August 2021DO - 1018203/2320-1770Ijrcog20212946 . 2021;

4. Deshmukh PY, Boricha BG, Pandey A. The association of thyroid disorders with abnormal uterine bleeding. Int J Reprod Contraception, Obstet Gynecol Vol 4, No 3 May-June 2015DO - 1018203/2320-1770Ijrcog20150077 . 2017;

5. Menon A. Rising prevalence of Thyroid disorders in India - The time to act is now. Thyroid Res Pract. 2020;17(3):99–100.

6. Kumar S, Kotur P. Effects of hypothyroidism in Indian women of reproductive age group–A review. Indian J Obstet Gynecol Res. 2020;7(1):1–6.

7. Mazzaferri EL. Evaluation and management of common thyroid disorders in women. Am J Obstet Gynecol. 1997;176(3):507–14.

8. Dittrich R, Beckmann MW, Oppelt PG, Hoffmann I, Lotz L, Kuwert T, et al. Thyroid hormone receptors and reproduction. J Reprod Immunol. 2011;90(1):58–66.

9. Michiels JJ, Schroyens W, Berneman Z, van der Planken M. Acquired von Willebrand syndrome type 1 in hypothyroidism: reversal after treatment with thyroxine. Clin Appl Thromb Off J Int Acad Clin Appl Thromb. 2001;7(2):113–5.

10. Munro MG, Critchley HOD, Fraser IS. The two FIGO systems for normal and abnormal uterine bleeding symptoms and classification of causes of abnormal uterine bleeding in the reproductive years: 2018 revisions. Int J Gynaecol Obstet Off Organ Int Fed Gynaecol Obstet. 2018;143(3):393–408.

11. Munro MG, Critchley HOD, Fraser IS. The FIGO classification of causes of abnormal uterine bleeding: Malcolm G. Munro, Hilary O.D. Crithcley, Ian S. Fraser, for the FIGO Working Group on Menstrual Disorders. Int J Gynaecol Obstet Off Organ Int Fed Gynaecol Obstet. 2011;113(1):1–2.

12. ACOG committee opinion no. 557: Management of acute abnormal uterine bleeding in nonpregnant reproductive-aged women. Obstet Gynecol. 2013;121(4):891–6.

13. INTERMENSTRUAL BLEEDING. Gynecol. Clin. Illus., WORLD SCIENTIFIC; 2017, p. 31–8.

14. Fraser IS, Critchley HOD, Broder M, Munro MG. The FIGO recommendations on terminologies and definitions for normal and abnormal uterine bleeding. Semin Reprod Med. 2011;29(5):383–90.

15. Cooke PS, Spencer TE, Bartol FF, Hayashi K. Uterine glands: development, function and experimental model systems. Mol Hum Reprod. 2013;19(9):547–58.

16. Ameer MA, Fagan SE, Sosa-Stanley JN, Peterson DC. Anatomy, Abdomen and Pelvis, Uterus., Treasure Island (FL): 2022.

17. Thiyagarajan DK, Basit H, Jeanmonod R. Physiology, Menstrual Cycle., Treasure Island (FL): 2022.

18. Preutthipan S, Herabutya Y. Hysteroscopic polypectomy in 240 premenopausal and postmenopausal women. Fertil Steril. 2005;83(3):705–9.

19. Hulka CA, Hall DA, McCarthy K, Simeone J. Sonographic findings in patients with adenomyosis: can sonography assist in predicting extent of disease? AJR Am J Roentgenol. 2002;179(2):379–83.

20. Brooks SE, Zhan M, Cote T, Baquet CR. Surveillance, epidemiology, and end results analysis of 2677 cases of uterine sarcoma 1989-1999. Gynecol Oncol. 2004;93(1):204–8.

21. Shankar M, Lee CA, Sabin CA, Economides DL, Kadir RA. von Willebrand disease in women with menorrhagia: a systematic review. BJOG. 2004;111(7):734–40.

22. Kadir RA, Economides DL, Sabin CA, Owens D, Lee CA. Frequency of inherited bleeding disorders in women with menorrhagia. Lancet (London, England). 1998;351(9101):485–9.

23. Kouides PA, Conard J, Peyvandi F, Lukes A, Kadir R. Hemostasis and menstruation: appropriate investigation for underlying disorders of hemostasis in women with excessive menstrual bleeding. Fertil Steril. 2005;84(5):1345–51.

24. Whitaker L, Critchley HOD. Abnormal uterine bleeding. Best Pract Res Clin Obstet Gynaecol. 2016;34:54–65.

25. Munro MG, Critchley H, Fraser IS. Research and clinical management for women with abnormal uterine bleeding in the reproductive years: More than PALM-COEIN. BJOG. 2017;124(2):185–9.

26. Cheong Y, Cameron IT, Critchley HOD. Abnormal uterine bleeding. Br Med Bull. 2017;123(1):103–14.

27. Roberts CP, Rock JA. Surgical methods in the treatment of congenital anomalies of the uterine cervix. Curr Opin Obstet Gynecol. 2011;23(4):251–7.

28. Kravets I. Hyperthyroidism: Diagnosis and Treatment. Am Fam Physician. 2016;93(5):363–70.

29. LeFevre ML. Screening for thyroid dysfunction: U.S. Preventive Services Task Force recommendation statement. Ann Intern Med. 2015;162(9):641–50.

30. Gillett M. Subclinical Hypothyroidism: Subclinical Thyroid Disease: Scientific Review and Guidelines for Diagnosis and Management. Clin Biochem Rev. 2004;25(3):191–4.

31. Biondi B, Palmieri EA, Fazio S, Cosco C, Nocera M, Saccà L, et al. Endogenous subclinical hyperthyroidism affects quality of life and cardiac morphology and function in young and middle-aged patients. J Clin Endocrinol Metab. 2000;85(12):4701–5.

32. Navarro Despaigne D. Epidemiología de las enfermedades del tiroides en Cuba. Rev Cuba Endocrinol. 2004;15(1):0.

33. Taylor PN, Albrecht D, Scholz A, Gutierrez-Buey G, Lazarus JH, Dayan CM, et al. Global epidemiology of hyperthyroidism and hypothyroidism. Nat Rev Endocrinol. 2018;14(5):301–16.

34. Diab N, Daya NR, Juraschek SP, Martin SS, McEvoy JW, Schultheiß UT, et al. Prevalence and risk factors of thyroid dysfunction in older adults in the community. Sci Rep. 2019;9(1):1–8.

35. Mescher A, Mescher A. Junqueira's Basic Histology: Text and Atlas 14e. McGraw Hill Medical Publishing Division; 2013.

36. Costanzo LS. Physiology, E-Book. Elsevier Health Sciences; 2013.

37. Hershman JM. Clinical application of thyrotropin-releasing hormone. N Engl J Med. 1974;290(16):886–90.

38. Ghamari-Langroudi M, Vella KR, Srisai D, Sugrue ML, Hollenberg AN, Cone RD. Regulation of thyrotropin-releasing hormone-expressing neurons in paraventricular nucleus of the hypothalamus by signals of adiposity. Mol Endocrinol. 2010;24(12):2366–81.

39. Hollenberg AN. The role of the thyrotropin-releasing hormone (TRH) neuron as a metabolic sensor. Thyroid. 2008;18(2):131–9.

40. Di Cosmo C, Liao X-H, Dumitrescu AM, Philp NJ, Weiss RE, Refetoff S. Mice deficient in MCT8 reveal a mechanism regulating thyroid hormone secretion. J Clin Invest. 2010;120(9):3377–88.

41. Fonseca TL, Correa-Medina M, Campos MPO, Wittmann G, Werneck-de-Castro JP, Arrojo e Drigo R, et al. Coordination of hypothalamic and pituitary T3 production regulates TSH expression. J Clin Invest. 2013;123(4):1492–500.

42. Lechan RM, Toni R. Functional anatomy of the hypothalamus and pituitary. Endotext [Internet]. 2016;

43. Persani L. Clinical review: Central hypothyroidism: pathogenic, diagnostic, and therapeutic challenges. J Clin Endocrinol Metab. 2012;97(9):3068–78.

44. Bougma K, Aboud FE, Harding KB, Marquis GS. Iodine and mental development of children 5 years old and under: a systematic review and meta-analysis. Nutrients. 2013;5(4):1384–416.

45. Persani L, Ferretti E, Borgato S, Faglia G, Beck-Peccoz P. Circulating thyrotropin bioactivity in sporadic central hypothyroidism. J Clin Endocrinol Metab. 2000;85(10):3631–5.

46. Chaker L, Bianco AC, Jonklaas J, Peeters RP. Hypothyroidism. Lancet (London, England). 2017;390(10101):1550–62.

47. Jonklaas J, Bianco AC, Bauer AJ, Burman KD, Cappola AR, Celi FS, et al. Guidelines for the treatment of hypothyroidism: prepared by the american thyroid association task force on thyroid hormone replacement. Thyroid. 2014;24(12):1670–751.

48. Abdalla SM, Bianco AC. Defending plasma T3 is a biological priority. Clin Endocrinol (Oxf). 2014;81(5):633–41.

49. Vita R, Saraceno G, Trimarchi F, Benvenga S. Switching levothyroxine from the tablet to the oral solution formulation corrects the impaired absorption of levothyroxine induced by proton-pump inhibitors. J Clin Endocrinol Metab. 2014;99(12):4481–6.

50. Wu Q, Xiao X, Xu Y. Evaluating the Performance of the WHO International Reference Standard for Osteoporosis Diagnosis in Postmenopausal Women of Varied Polygenic Score and Race. J Clin Med. 2020;9(2).

51. Delitala AP, Scuteri A, Doria C. Thyroid Hormone Diseases and Osteoporosis. J Clin Med. 2020;9(4).

52. Fraser SA, Anderson JB, Smith DA, Wilson GM. Osteoporosis and fractures following thyrotoxicosis. Lancet (London, England). 1971;1(7707):981–3.

53. Grimnes G, Emaus N, Joakimsen RM, Figenschau Y, Jorde R. The relationship between serum TSH and bone mineral density in men and postmenopausal women: the Tromsø study. Thyroid. 2008;18(11):1147–55.

54. Gosi SKY, Garla V V. Subclinical Hypothyroidism., Treasure Island (FL): 2022.

55. Vejbjerg P, Knudsen N, Perrild H, Laurberg P, Carlé A, Pedersen IB, et al. Lower prevalence of mild hyperthyroidism related to a higher iodine intake in the population: prospective study of a mandatory iodization programme. Clin Endocrinol (Oxf). 2009;71(3):440–5.

56. Boelaert K, Torlinska B, Holder RL, Franklyn JA. Older subjects with hyperthyroidism present with a paucity of symptoms and signs: a large cross-sectional study. J Clin Endocrinol Metab. 2010;95(6):2715–26.

57. Vaidya B, Pearce SHS. Diagnosis and management of thyrotoxicosis. BMJ. 2014;349:g5128.

58. van Soestbergen MJ, van der Vijver JC, Graafland AD. Recurrence of hyperthyroidism in multinodular goiter after long-term drug therapy: a comparison with Graves' disease. J Endocrinol Invest. 1992;15(11):797–800.

59. Byna P, Siddula S, Kolli S, Shaik M V. Thyroid abnormality in perimenopausal women with abnormal uterine bleeding. Int J Res Med Sci. 2015;3(11):3250–3.

60. Koutras DA. Disturbances of menstruation in thyroid disease. Ann N Y Acad Sci. 1997;816:280–4.

61. Kumar S, Kotur P. Occurrence of hypothyroidism and its correlation with reproductive health problems: a cross sectional study 2019;

62. Nanda SS, Dash S, Behera A, Mishra B. Thyroid profile in polycystic ovarian syndrome. J Evol Med Dent Sci. 2014;3(37):9594–601.

63. Mounika K. Study on Thyroid Dysfunction in Patients of Dysfunctional Uterine Bleeding. J Pharm Res Int. 2021;33(40A):44–9.

64. Thakur M, Maharjan M, Tuladhar H, Dwa Y, Bhandari S, Maskey S, et al. Thyroid Dysfunction in Patients with Abnormal Uterine Bleeding in a Tertiary Care Hospital: A Descriptive Cross-sectional Study. JNMA J Nepal Med Assoc. 2020;58(225):333–7.

65. Roshnibala S, Sudhuanshu K. Thyroid disorders in reproductive age presenting with abnormal uterine bleeding. Indian J Obstet Gynecol Res. 2021;2021:14027.

66. Tara. Thyroid Dysfunction and Abnormal Uterine Bleeding. J Gynecol Womens Heal. 2019;15(4).

67. Singh S, Sahoo S, Das PC. A study of thyroid dysfunction in dysfunctional uterine bleeding. Int J Reprod Contraception, Obstet Gynecol Vol 7, No 3 March 2018DO - 1018203/2320-1770Ijrcog20180881 . 2018;

68. Verma SK, Pal A, Jaswal S. A study of thyroid dysfunction in dysfunctional uterine bleeding. Int J Reprod Contraception, Obstet Gynecol Vol 6, No 5 May 2017DO - 1018203/2320-1770Ijrcog20171972 . 2017;

69. Sudha HC, Sunanda KM, Anitha GS. Abnormal uterine bleeding in relation to thyroid dysfunction. Int J Reprod Contraception, Obstet Gynecol. 2018;7(11):4525–31.

70. Phukan JK, Saharia GK, Goswami R. Thyroid status in patients with dysfunctional uterine bleeding in a tertiary care hospital of Assam. Indian J Med Biochem. 2012;20(1):11–5.

71. Nayak AK. Thyroid disorders in patients with dysfunctional uterine bleeding. Panacea J Med Sci. 2021;11(3):503–6.

72. Ajmani NS, Sarbhai V, Yadav N, Paul M, Ahmad A, Ajmani AK. Role of Thyroid Dysfunction in Patients with Menstrual Disorders in Tertiary Care Center of Walled City of Delhi. J Obstet Gynaecol India. 2016;66(2):115–9.

73. Joshi BR, Rizal S, Subedi S. Thyroid Dysfunction in Patient with Abnormal Uterine Bleeding in a Tertiary Hospital of Eastern Nepal: A Descriptive Cross-sectional Study. JNMA J Nepal Med Assoc. 2021;59(239):635–9.

74. Sudha Rani Gandi, Pallavi Vishwekar, Raj Shekhar Yadav NC. Study of thyroid dysfunction in women with menstrual disorders - A prospective study. Medpulse Int J Gynaecol. 2019;9(3).

75. Jinger SK, Verma A, Dayma I, Talreja T. To study the thyroid profile in menstrual disorder at tertiary care hospital in northern western Rajasthan, India. Int J Res Med Sci. 2017;5(5):2212–4.

76. Kumari A, Rohatgi R, Singh A. Evaluation of thyroid dysfunction in patients with menstrual disorders of reproductive age group: a prospective cross-sectional study. Int J Reprod Contraception, Obstet Gynecol Vol 10, No 2 Febr 2021DO - 1018203/2320-1770Ijrcog20210320 . 2021;

77. Subedi S, Banerjee B, Chhetry M. Thyroid disorders in women with dysfunctional uterine bleeding. J Pathol Nepal. 2016;6:1018.

78. Davis E, Sparzak PB. Abnormal Uterine Bleeding., Treasure Island (FL): 2022.

79. Henry C, Ekeroma A, Filoche S. Barriers to seeking consultation for abnormal uterine bleeding: systematic review of qualitative research. BMC Womens Health. 2020;20(1):123.

80. Narula ER. Menstrual irregularities. J Obs Gynecol India. 1967;17:164.

81. Pahwa S, Shailja G, Jasmine K. Thyroid dysfunction in dysfunctional uterine bleeding. J Adv Res Bio Sci. 2013;5(1):78–83.

82. Chaitra M, Anitha GS, Savitha C. A prospective study of thyroid dysfunction in dysfunctional uterine bleeding. Int J Reprod Contraception, Obstet Gynecol. 2019;8(11):4387–91.

83. Bhavani N, Sathineedi A, Giri A, Chippa S, Reddy VSP. A study of correlation between abnormal uterine bleeding and thyroid dysfunction. Int J Recent Trends Sci Tech. 2015;14(1):131–5.

84. Singh P, Dubey P, Yadav S, Yadav SS. Thyroid abnormality in abnormal uterine bleeding: an observational study from Medical College in Western UP, India. Int J Reprod Contraception, Obstet Gynecol. 2018;7(1):308–12.

85. Gowri M, Radhika BH, Harshini V, Ramaiaha R. Role of thyroid function tests in women with abnormal uterine bleeding. Int J Reprod Contracept Obs Gynecol. 2014;3(1):54–7.

86. Sowers M, Luborsky J, Perdue C, Araujo KLB, Goldman MB, Harlow SD. Thyroid stimulating hormone (TSH) concentrations and menopausal status in women at the mid- life: SWAN. Clin Endocrinol (Oxf). 2003;58(3):340–7.

87. Kaur T, Aseeja V, Sharma S. Thyroid dysfunction in dysfunctional uterine bleeding 2011;

88. Ashok KHS, Saravanan S. A study of prevalence of thyroid disorders in patients with abnormal uterine bleeding. Int J Reprod Contraception, Obstet Gynecol. 2017;6(3):1036–40.

89. Kattel P. Thyroid function test in abnormal uterine bleeding. Nepal J Obstet Gynaecol. 2017;12(2):74–8.

90. Komathi R, Mallika A. Shantha. A study of thyroid profile in abnormal uterine bleeding (aub) among Reproductive age group women. Int J Curr Med Sci. 2016;6(7):133–6.

91. Patil A, Shiragur SS, Gobbur V, Mudanur S, Bidri S, Yaliwal R, et al. Association of Bleeding Patterns with Thyroid Dysfunction in Patients with Abnormal Uterine Bleeding: A Prospective Cross-sectional Study. J Clin Diagnostic Res. 2022;10–3.

92. Verma SK, Pal A, Jaswal S. A study of thyroid dysfunction in dysfunctional uterine bleeding. Int J Reprod Contraception, Obstet Gynecol Vol 6, No 5 May 2017DO - 1018203/2320-1770Ijrcog20171972 . 2017;

ANNEXURES

ANNEXURES

A STUDY OF THYROID DYSFUNCTION IN PATIENT WITH ABNORMAL UTERINE BLEEDING

SERIAL NO: **HOSPITAL NO:**

NAME: **OCCUPATION:**

AGE:

ADDRESS:

SOCIAL -ECONOMIC STATUS:

1. **CHIFE COMPLAINTS:**

2. **HISTORY OF PRESENTING COMPLAINTS**

a. Bleeding per vagina :

i. Duration :

ii. Interval :

iii. Quantity : Scanty / moderate / excessive

iv. H/o Dysmenorrhoea : Yes/No

b. Other complaints :

3. **MENSTRUAL HISTORY :**

i. Acyclial (MPH) :

ii. Hypomenorrhoea :

iii. Menorrhagia

iv. Metrorrhagia :

v. Oligomenorrhoea :

vi. Polymenorrhagia :

vii. Polymenorrhoea :

viii. Age of attainment of menarche:

ix. Previous Menstrual cycles –

1. Duration of cycles :
2. Amount of flow :
3. Duration of flow :
4. Associated dysmenorrhea ;

x. Date of late menstrual period:

4.OBSTETRIC HISTORY ;

a. married life: para: living:

b. abortion: last delivery:

c. type of deliveries: tubectomy: yes/no

5. PAST HISTORY:

a. TB/bronchial asthma/RHD/blood transfusion/ any operations

6.FAMILY HISTORY:

a. TB/ /bronchial asthma/diabetes mellitus /hypertension/any cancer/bleeding disorders/ thyroid disorders

7. PERSONAL HISTORY:

a. Diet :

b. Appetite :

c. Micturation :

d. Sleep :

8. EXAMINATION OF PATIENT:

1. GENERAL CONDITION

2. HEAD TO TOE

a. distribution of hair

b. thickening of skin: Dryness/scaling

c. Edema

d. Hoarseness of voice

3. Nutritional status

4. Anaemia

5.CVS

6. Respiratory system

7. Pulse rate

8. Blood pressure

a. operative scar : present /absent

b. Engorged vein : present /absent

c. Ascites : present /absent

d. any enlargement of liver /spleen: present /absent

9. VULVO VAGINA EXAMINATION: healthy /unhealthy

10. PER SPECULUM EXAMINATION :

a. vagina :

b. cervix :

c. bleeding :

11. PER VAGINAL EXAMINATION:

a. Cervix : normal flushed with vault

b. Uterus : Anteverted Retroverted

c. Normal size : Bulky smaller

d. soft : Firm Hard

e. mobile : fixed

f. tender : non tender

g. tenderness in fornix: present absent

h. Uterocervical length:

12. PER RECTAL EXAMINATION:

13. INVESTIGATION:

a. HB% Platelet count, TC, DC

b.USG abdomen pelvis

14. COMPULSORY:

i. Thyroid Function test:

a. T3

b. T4

c. TSH

15. OPTIONAL:

i. pap smear

ii. Histopathology of endometrium

PATIENT INFORMATION SHEET

STUDY TITLE: " ASSOCIATION OF THYROID DYSFUNCTION IN ABNORMAL UTERINE BLEEDING ".

STUDY SITE: RL Jalappa Hospital and Research center, Tamaka, Kolar.

This is to inform you that, you require ultrasound for Making treatment plan for you condition that is AUB. The ultrasound is required to rule out uterine structural abnormalities and for planning of the treatment.

We are conducting this study to predict the prevalence of this condition.

If you are willing you will be enrolled in this study and we will do ultrasound and other relevant investigation which are required for surgical procedures.

You will receive the standard care pre and post operatively.

This will facilitate identifying cause (if any) in an early stage and treating it. You are free to opt-out of the study at any time if you are not satisfied or apprehensive to be a part of the study. Your treatment and care will not be compromised if you refuse to be a part of the study

.

The study will not add any risk or financial burden to you if you are part of the study. In case of any complication during surgery patient will be treated accordingly.

Your identity and clinical details will be confidential. you will not receive any financial benefit for being part of the study. you are free to contact Dr. DHANUSHA NEKKANTI or any other member of the above research team for any doubt or clarification you have.

Dr. DHANUSHA NEKKANTI
Mobile no: 9676230367
E-Mail id: Dhanusha883@gamil.com

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: "ಅಸಹಜ ಗರ್ಭಾಶಯದ ರಕ್ತಸ್ರಾವದಲ್ಲಿ ಥೈರಾಯ್ಡ್ ಅಪಸಾಮಾನ್ಯ ಕ್ರಿಯೆಯ ಅಸೋಸಿಯೇಷನ್".

ಅಧ್ಯಯನ ತಾಣ: ಆರ್.ಎಲ್.ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ, ಕೋಲಾರ.

ನೀವು ಅಲ್ಟ್ರಾಸೌಂಡ್ ಅಗತ್ಯವಿದೆ ಎಂದು ನಿಮಗೆ ತಿಳಿಸಲು ಇದು

AUB ಆಗಿರುವ ನಿಮ್ಮ ಸ್ಥಿತಿಗೆ ಚಿಕಿತ್ಸಾ ಯೋಜನೆಯನ್ನು ಮಾಡುವುದು. ಗರ್ಭಾಶಯದ ರಚನಾತ್ಮಕ ಅಸಹಜತೆಗಳನ್ನು ತಳ್ಳಿಹಾಕಲು ಮತ್ತು ಚಿಕಿತ್ಸೆಯ ಯೋಜನೆಗಾಗಿ ಅಲ್ಟ್ರಾಸೌಂಡ್ ಅಗತ್ಯವಿದೆ

ಈ ಸ್ಥಿತಿಯ ಪ್ರಭುತ್ವವನ್ನು ಊಹಿಸಲು ನಾವು ಈ ಅಧ್ಯಯನವನ್ನು ನಡೆಸುತ್ತಿದ್ದೇವೆ.

ನೀವು ಸಿದ್ಧರಿದ್ದರೆ ನೀವು ಈ ಅಧ್ಯಯನಕ್ಕೆ ದಾಖಲಾಗುತ್ತೀರಿ ಮತ್ತು ಶಸ್ತ್ರಚಿಕಿತ್ಸಾ ವಿಧಾನಗಳಿಗೆ ಅಗತ್ಯವಿರುವ ಅಲ್ಟ್ರಾಸೌಂಡ್ ಮತ್ತು ಇತರ ಸಂಬಂಧಿತ ತನಿಖೆಯನ್ನು ನಾವು ಮಾಡುತ್ತೇವೆ.

ನೀವು ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ಪೂರ್ವ ಮತ್ತು ನಂತರದ ಪ್ರಮಾಣಿತ ಆರೈಕೆಯನ್ನು ಸ್ವೀಕರಿಸುತ್ತೀರಿ.

ಇದು ಆರಂಭಿಕ ಹಂತದಲ್ಲಿ ಕಾರಣವನ್ನು (ಯಾವುದಾದರೂ ಇದ್ದರೆ) ಗುರುತಿಸಲು ಮತ್ತು ಅದಕ್ಕೆ ಚಿಕಿತ್ಸೆ ನೀಡಲು ಅನುಕೂಲವಾಗುತ್ತದೆ. ಅಧ್ಯಯನದ ಭಾಗವಾಗಲು ನೀವು ತೃಪ್ತರಾಗದಿದ್ದರೆ ಅಥವಾ ಭಯಪಡದಿದ್ದರೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹೊರಗುಳಿಯಲು ನೀವು ಸ್ವತಂತ್ರರಾಗಿದ್ದೀರಿ. ನೀವು ಅಧ್ಯಯನದ ಭಾಗವಾಗಲು ನಿರಾಕರಿಸಿದರೆ ನಿಮ್ಮ ಚಿಕಿತ್ಸೆ ಮತ್ತು ಆರೈಕೆಯು ರಾಜಿಯಾಗುವುದಿಲ್ಲ.

ನೀವು ಅಧ್ಯಯನದ ಭಾಗವಾಗಿದ್ದರೆ ಅಧ್ಯಯನವು ನಿಮಗೆ ಯಾವುದೇ ಅಪಾಯ ಅಥವಾ ಆರ್ಥಿಕ ಹೊರೆಯನ್ನು ಸೇರಿಸುವುದಿಲ್ಲ. ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ತೊಡಕುಗಳ ಸಂದರ್ಭದಲ್ಲಿ ರೋಗಿಗೆ ಅನುಗುಣವಾಗಿ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುತ್ತದೆ.

ನಿಮ್ಮ ಗುರುತು ಮತ್ತು ಕ್ಲಿನಿಕಲ್ ವಿವರಗಳು ಗೌಪ್ಯವಾಗಿರುತ್ತವೆ. ಅಧ್ಯಯನದ ಭಾಗವಾಗಿರುವುದರಿಂದ ನೀವು ಯಾವುದೇ ಹಣಕಾಸಿನ ಪ್ರಯೋಜನವನ್ನು ಪಡೆಯುವುದಿಲ್ಲ. ನೀವು ಹೊಂದಿರುವ ಯಾವುದೇ ಸಂದೇಹ ಅಥವಾ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ಡಾ. ಧನುಷಾ ನೆಕ್ಕಂತಿ ಅಥವಾ ಮೇಲಿನ ಸಂಶೋಧನಾ ತಂಡದ ಯಾವುದೇ ಇತರ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ನೀವು ಮುಕ್ತರಾಗಿದ್ದೀರಿ.

ಡಾ. ಧನುಷಾ ನೆಕ್ಕಂಟಿ

ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: 9676230367

ಎ-ಮೇಲ್ ಆಗಿದೆ: Dhanusha883@gamil.com

INFORMED CONSENT FORM

1.Mr./Mrs._____ have been explained in my own understandable language, that I will included in a study which is **"ASSOCIATION OF THYROID DYSFUNCTION IN ABNORMAL UTERINE BLEEDING"**

I have been explained that my clinical findings, investigations, postoperative finding will be assessed and documented for study purpose.

I have been explained my participation in this study is entirely voluntary, and I can withdraw from the study any time and this will not affect my relation with my doctor or the treatment for my ailment.

I have been explained about the interventions needed possible benefits and adversities due to interventions, in my one understandable language.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the finding, my details will be masked.

I have principal investigator mobile number for enquiries.

I in my sound mind give full consent to be added in the part of this study.

Signature of the patient:

Name:

Signature of the witness:

Name:

Relation to patient:

Date:

Place:

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ ನಮೂನೆ

1.Mr./Mrs._____ ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಅಸೋಸಿಯೇಶನ್ ಆಫ್ ಥೈರಾಯ್ಡ್ ಡಿಸ್ಫ್ಯೂಷನ್ ಇನ್ ಅಬ್ನಾರ್ಮಲ್ ಯುಟಿರಿನ್ ಬ್ಲೀಡಿಂಗ್
ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಆವಿಷ್ಕಾರಗಳು ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ, ತನಿಖೆಗಳು, ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರದ ಪತ್ತೆಹಚ್ಚುವಿಕೆಯನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುತ್ತದೆ ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ, ಮತ್ತು ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ನನ್ನ ಕಾಯಿಲೆಯ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ.

ಮಧ್ಯಸ್ಥಿಕೆಗಳಿಂದ ಸಾಧ್ಯವಾದ ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಪ್ರತಿಕೂಲತೆಗಳ ಬಗ್ಗೆ ನನ್ನ ಒಂದು ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಕಂಡುಬರುವ ನನ್ನ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿರಿಸಲಾಗಿದೆ ಮತ್ತು ಫೈಂಡಿಂಗ್ ಅನ್ನು ಪ್ರಕಟಿಸುವಾಗ ಅಥವಾ ಹಂಚಿಕೊಳ್ಳುವಾಗ, ನನ್ನ ವಿವರಗಳನ್ನು ಮುಸುಕು ಮಾಡಲಾಗುವುದು ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ವಿಚಾರಣೆಗಾಗಿ ನನ್ನ ಬಳಿ ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ ಮೊಬೈಲ್ ಸಂಖ್ಯೆ ಇದೆ.

ಈ ಅಧ್ಯಯನದ ಭಾಗದಲ್ಲಿ ಸೇರಿಸಲು ನನ್ನ ಧ್ವನಿ ಮನಸ್ಸಿನಲ್ಲಿ ನಾನು ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ.

ರೋಗಿಯ ಸಹಿ:

ಹೆಸರು:

ಸಾಕ್ಷಿಯ ಸಹಿ:

ಹೆಸರು:

ರೋಗಿಯ ಸಂಬಂಧ:

ದಿನಾಂಕ:

ಸ್ಥಾನ:

MASTER CHART

SL NO	OP/IP	Age in years	parity	duration	cycle length in days		type of bleeding	T3 ng/ml	T4 mcg/dl	TSH mciu/ml		TDF	FIBROID	ADENOM	PCOD	POLYP
1	886318	30	P3L3	7	28		Menorrhagia	0.37	3.4	72.61		HYPO	NO	NO	NO	NO
2	885706	26	P2L2	10	45		Acyclical	0.88	8.13	1.51		EU	NO	NO	NO	NO
3	886241	40	P4L4	1	37		Oligomenorrhoea	0.45	3.2	55.3		HYPO	NO	NO	NO	NO
4	884532	24	P2L2	7	20		Polymenorrhagia	1.87	9.4	2.1		EU	NO	NO	NO	NO
5	885899	35	P3L3	7	30		Menorrhagia	1.39	11.2	3.87		EU	NO	NO	NO	NO
6	892710	23	0	2	40		Oligomenorrhoea	0.66	2.4	35.7		HYPO	NO	NO	NO	NO
7	880784	24	P1L1	7	30		Menorrhagia	1.21	10.9	2.39		EU	NO	NO	NO	NO
8	910284	39	P4L4	7	20		Polymenorrhagia	1.49	10.8	1.33		EU	NO	NO	NO	NO
9	873945	30	P2L2	7	30		Menorrhagia	1.5	8.57	1.49		EU	NO	NO	NO	NO
10	853282	38	P3L3	8	20		Polymenorrhagia	0.89	8.2	2.53		EU	NO	NO	NO	NO
11	907659	25	P1L1	7	30		Menorrhagia	1.38	6.3	1.24		EU	NO	NO	NO	NO
12	929483	38	P2L2	2	30		Oligomenorrhoea	0.67	3.9	27.4		HYPO	NO	NO	NO	NO
13	929460	38	P2L2	8	30		Menorrhagia	1.08	9.5	1.67		EU	NO	NO	NO	NO
14	930147	35	P3L3	10	60		Acyclical	0.54	2.8	68.6		HYPO	NO	NO	NO	NO
15	925226	32	P1L1	1	Irregular		Metrorrhagia	1.27	10.7	1.45		EU	NO	NO	NO	PRESENT
16	926992	23	UNMARRIED	7	30		Menorrhagia	1.96	7.9	3.4		EU	NO	PRESENT	NO	NO
17	931030	36	p3l3	7	20		polymenorrhoea	1.04	9.2	2.76		EU	NO	NO	NO	NO
18	923356	23	0	10	60		Acyclical	1.23	7.8	17.43		SUB	NO	NO	NO	NO
19	929325	29	P2L2	7	20		Polymenorrhagia	1.43	6.8	2.8		EU	NO	NO	NO	NO
20	928033	33	P2L2	9	30		Menorrhagia	1.95	6.7	9.87		SUB	NO	NO	NO	NO
21	934311	33	P2L2	10	90		Acyclical	1.23	8.7	2.3		EU	NO	NO	NO	NO
22	936394	37	P4L4	8	30		Menorrhagia	1.09	9.8	1.49		EU	PRESENT	NO	NO	NO
23	936341	24	0	10	90		Acyclical	1.38	8.1	2.93		EU	NO	NO	NO	NO
24	935862	32	P2L2	1	30		Menorrhagia	1.96	9.6	2.96		EU	NO	NO	NO	NO
25	938349	30	P2L2	2	45	Oligomenorrho		2.93	14.75	0.03		HYPER	NO	NO	NO	NO
26	938640	32	P2L2	7	20		Polymenorrhagia	1.45	10.5	2.96		EU	NO	NO	NO	NO
27	939045	42	P3L3	7	20		Polymenorrhagia	0.96	6.5	1.24		EU	NO	NO	NO	NO
28	939735	32	P3L3	10	90		Acyclical	0.89	7.7	3.64		EU	NO	NO	NO	NO
29	936229	34	P4L4	8	30		Menorrhagia	1.65	11.2	12.42		SUB	NO	NO	NO	NO
30	73938	31	p2l2	1	45		Oligomenorrhoea	1.5	10.5	2.6		EU	NO	NO	NO	NO
31	67972	37	P4L4	8	30	Menorrhagia		0.98	8.9	3.97		EU	NO	NO	NO	NO
32	52047	33	P1L1	6	20		Polymenorrhagia	1.72	7.8	1.98		EU	NO	NO	NO	NO
33	51690	37	P2L2	7	30		Menorrhagia	0.98	9.8	14.8		SUB	NO	PRESENT	NO	NO
34	53374	26	P3L3	7	28		Menorrhagia	1.43	6.8	1.36		EU	NO	NO	NO	NO
35	53980	25	P1L1	10	60		Acyclical	0.87	9.4	4		EU	NO	NO	NO	NO
36	54443	20	UNMARRIED	2	45		Oligomenorrhoea	0.43	1.4	80.2		HYPO	NO	NO	NO	NO
37	55774	31	P5L5	7	20		Polymenorrhoea	1.86	6.8	3.64		EU	NO	NO	NO	NO
38	51101	38	P2L2	6	30		Menorrhagia	1.74	6.7	2.56		EU	NO	NO	NO	NO
39	55547	29	P1L1	6	20		Polymenorrhea	0.86	10.5	3.2		EU	NO	NO	NO	NO
40	72945	43	P4L4	2	45	Oligomenorrhe		2.34	16.74	0.14		HYPER	NO	NO	NO	NO
41	56775	32	P2L2	1	30		Hypomenorrhoea	1.76	9.7	2.85		EU	NO	NO	NO	NO
42	57924	22	UNMARRIED	8	30		Menorrhagia	0.49	5.36	2.5		EU	PRESENT	NO	NO	NO
43	57681	32	P2L2	2	Irregular		Metrorrhagia	1.29	4.67	3.93		EU	NO	NO	NO	NO
44	58325	29	P3L3	9	30		Menorrhagia	0.89	10.6	22.62		SUB	NO	NO	NO	NO
45	58492	28	P1L1	10	60		Acyclical	1.76	6.8	11.78		SUB	NO	NO	NO	NO
46	58996	30	P2L2	7	20		Polymenorrhagia	0.88	8.13	1.51		EU	NO	NO	NO	NO
47	67625	42	P5L5	10	60		Acyclical	1.23	7.8	17.26		SUB	NO	NO	NO	NO
48	59281	26	P1L1	7	30		Menorrhagia	1.35	8.71	1.74		EU	NO	NO	NO	NO
49	56313	24	UNMARRIED	6	30		Menorrhagia	1.89	9.2	13.16		SUB	NO	NO	NO	NO
50	61189	35	P4L4	1	45	Oligomenorrho		1.29	7.87	2.62		EU	NO	NO	NO	NO
51	41184	32	P2L2	8	30		Menorrhagia	1.62	7.8	20.21		SUB	NO	NO	NO	NO
52	62704	33	P2L2	1	30		Hypomenorrhoea	1.44	5.96	3.38		EU	NO	NO	NO	NO
53	62724	28	P3L3	10	90		Acyclical	0.23	1.5	60.4		HYPO	NO	NO	NO	NO
54	63106	30	P2L2	1	30		Hypomenorrheoa	1.67	8.4	2.24		EU	NO	NO	NO	NO
55	63297	32	P2L2	2	45	Oligomenorrho		1.23	7.7	18.86		SUB	NO	NO	PRESENT	NO
56	64174	28	P3L3	7	30		Menorrhagia	1.21	10.3	3.3		EU	NO	NO	NO	NO

SL NO	OP/IP	Age in years	parity	duration	cycle length in days		type of bleeding	T3 ng/ml	T4 mcg/dl	TSH mciu/ml		TDF	FIBROID	ADENOM	PCOD	POLYP
57	64742	35	P1L1	7	30		Menorrhagia	1.43	8.5	16.69		SUB	NO	NO	NO	NO
58	66414	34	P4L4	10	90		Acyclical	0.98	8.7	2.78		EU	NO	NO	NO	NO
59	66645	27	P1L1	2	45		Oligomenorrhea	0.54	2.4	63.7		HYPO	NO	NO	NO	NO
60	66908	28	P3L3	7	30		Menorrhagia	0.63	3.3	76.4		HYPO	PRESENT	NO	NO	NO
61	67893	31	P2L2	5	20		Polymenorrhea	0.89	8.9	19.28		SUB	NO	NO	NO	NO
62	73991	44	P3L3	8	30		Menorrhagia	1.37	9.9	1.81		EU	NO	NO	NO	NO
63	39196	28	P2L2	2	45		Oligomenorrhea	1.14	9.5	1.36		EU	NO	NO	NO	NO
64	68648	27	P1L1	6	20		Polymenorrhagia	0.45	3.1	67.4	8	HYPO	NO	NO	NO	NO
65	69208	21	P1L1	2	40		Oligomenorrhea	1.36	9.5	2.03		EU	NO	NO	NO	NO
66	69927	34	P2L2	10	90		Acyclical	0.97	10.6	0.91		EU	NO	NO	NO	NO
67	37595	32	P4L4	7	20		Polymenorrhagia	1.45	7.2	1.23		EU	NO	NO	NO	NO
68	68062	30	P2L2	8	30		Menorrhagia	1.46	7.3	1.62		EU	NO	NO	NO	NO
69	10805	34	P4L4	2	Irregular		Metrorrhagia	1.29	11.9	3.23		EU	NO	NO	NO	PRESENT
70	74955	39	P4L4	1	45		Oligomenorrhea	3.61	16.85	0.16		HYPER	NO	NO	NO	NO
71	11715	36	P2L2	6	20		Polymenorrhagia	1.38	6.2	28.76		SUB	NO	NO	NO	NO
72	75976	23	UNMARRIED	8	30		Menorrhagia	1.64	8.6	9.82		SUB	NO	NO	NO	NO
73	70285	36	P4L4	5	20		Polymenorrhagia	0.98	8.6	1.97		EU	NO	NO	NO	NO
74	932480	26	UNMARRIED	7	30		Menorrhagia	1.64	8.7	1.17		EU	NO	NO	NO	NO
75	939169	37	P4L4	2	45		Oligomenorrhoea	1.33	6.9	0.8		EU	NO	NO	NO	NO
76	75992	34	P3L3	8	30		Menorrhagia	1.08	8.8	2.29		EU	PRESENT	NO	NO	NO
77	943909	30	P1L1	7	20		Polymenorrhagia	1.35	10.8	2.9		EU	NO	NO	NO	NO
78	942952	27	UNMARRIED	7	30		Menorrhagia	0.52	2.1	58.3		HYPO	NO	NO	NO	NO
79	943491	34	P3L3	10	90		Acyclical	1.43	6.3	2.45		EU	NO	NO	PRESENT	NO
80	945362	36	P2L2	8	30	Menorrhagia		1.86	7.5	1.76		EU	NO	NO	NO	NO
81	883816	32	P2L2	6	20		Polymenorrhagia	1.54	6.7	1.23		EU	NO	NO	NO	NO
82	947196	30	P1L1	1	40	O	Oligomenorrhea	1.86	9.3	3.52		EU	NO	NO	NO	NO
83	944670	36	P4L4	8	30		Menorrhagia	2.21	14.45	0.23		HYPER	NO	NO	NO	NO
84	936947	31	P2L2	10	60		Acyclical	1.76	8.4	1.56		EU	NO	NO	NO	NO
85	748765	36	P3L3	7	30		Menorrhagia	0.86	9.6	1.67		EU	NO	NO	NO	NO
86	37431	34	P3L3	9	30		Menorrhagia	1.48	10.9	34.8		SUB	PRESENT	NO	NO	NO
87	35043	27	0	10	60		Acyclical	1.06	9.5	1.67		EU	NO	NO	NO	NO
88	42303	34	P3L3	1	45		Oligomenorrhea	1.34	6.9	2.67		EU	NO	NO	NO	NO
89	46413	43	P4L4	10	90	Acyclical		0.34	2.3	92.1		HYPO	NO	NO	NO	NO
90	47888	22	P2L2	6	30		Menorrhagia	1.78	9.7	2.36		EU	NO	NO	NO	NO
91	47893	23	P1L1	8	30		Menorrhagia	1.53	11.2	10.9		SUB	NO	NO	NO	NO
92	952137	34	P3L3	10	60	Acyclical		0.98	11.2	1.32		EU	NO	NO	NO	NO
93	44217	37	P2L2	7	20		Polymenorrhoea	1.54	7.9	2.74		EU	NO	NO	NO	NO
94	51340	38	P3L3	2	45		Oligomenorrhoea	2.34	19.2	0.31		HYPER	NO	NO	NO	NO
95	51385	26	P1L1	2	30		Hypomenorrhoea	1.45	7.8	2.16		EU	NO	NO	NO	NO
96	937144	36	P4L4	7	30		Menorrhagia	1.86	5.2	1.54		EU	NO	NO	NO	NO
97	52217	36	P3L3	2	Irregular		Metrorrhagia	1.56	10.9	2.34		EU	NO	NO	NO	NO
98	52403	22	P2L2	1	45		Oligomenorrhea	1.94	5.2	14.46		SUB	NO	NO	NO	NO
99	53688	24	0	2	30		Menorrhagia	0.52	2.4	63		HYPO	NO	NO	NO	NO
100	70326	31	P2L2	2	45		Oligomenorrhoea	2.79	30.5	0.19		HYPER	NO	NO	NO	NO
101	64905	32	P1L1	10	90		Acyclical	1.52	11.4	2.19		EU	NO	NO	NO	NO
102	78020	31	P3L3	6	20		Polymenorrhagia	1.67	8.3	3.72		EU	NO	NO	NO	NO
103	60820	23	UNMARRIED	8	30		Menorrhagia	1.74	11.3	2.7		EU	PRESENT	NO	NO	NO
104	34525	30	P2L2	7	30		Menorrhagia	0.84	10.1	11.32		SUB	NO	NO	NO	NO
105	81890	21	UNMARRIED	7	30		Menorrhagia	1.32	7.2	2.5		EU	NO	NO	NO	NO
106	55238	26	P2L2	2	40		Oligomenorrhoea	1.17	5.4	1.45		EU	NO	NO	PRESENT	NO
107	7774	20	UNMARRIED	6	30		Menorrhagia	1.67	8.4	1.67		EU	NO	NO	NO	NO
108	93693	28	P1L1	6	20		Polymenorrhoea	0.96	10.7	6.89		SUB	FIBRIOD	NO	NO	NO
109	50488	22	UMMARRIED	1	45		Oligomenorrhoea	0.86	8.7	1.74		EU	NO	NO	NO	NO
110	46618	37	P3L3	7	20		Polymenorrhagia	1.62	10.4	2.13		EU	NO	NO	NO	NO
111	72449	40	P5L5	9	30		Menorrhagia	1.37	5.6	1.93		EU	NO	NO	NO	NO
112	103760	22	UNMARRIED	2	45		oligomenorrhea	0.86	7.8	1.43		EU	NO	NO	NO	NO

SL NO	OP/IP	Age in years	parity	duration	cycle length in days		type of bleeding	T3 ng/ml	T4 mcg/dl	TSH mciu/ml		TDF	FIBROID	ADENOM	PCOD	POLYP
113	17256	31	P2L2	8	30		Menorrhagia	1.78	8.3	1.6		EU	NO	NO	NO	NO
114	87449	28	P1L1	7	20		Polymenorrhoea	0.85	7.2	7.92		SUB	NO	NO	NO	NO
115	94501	29	P1L1	6	20		Polymenorrhoea	1.47	5.8	1.89		EU	NO	NO	NO	NO
116	11013	35	P3L3	6	30		Menorrhagia	0.79	7.3	1.72		EU	NO	NO	NO	NO
117	109482	27	P3L3	10	70	Acyclical		1.89	8.5	3.6		EU	NO	NO	NO	NO
118	54143	32	P3L3	1	30		Hypomenorrhoea	1.98	10.8	0.3		EU	NO	NO	NO	NO
119	104740	35	P4L4	2	45	O	Oligomenorrhoea	3.61	18.45	0.21		HYPER	NO	NO	NO	NO
120	122912	37	P3L3	8	30		Menorrhagia	1.33	9.7	1.35		EU	NO	NO	NO	NO
121	76651	34	P2L2	7	20		Polymenorrhagia	1.28	7.4	1.6		EU	NO	NO	NO	NO
122	64255	36	P4L4	6	30		Menorrhagia	1.45	9.4	3.76		EU	NO	NO	NO	NO
123	48746	34	p5l5	2	45		oligomenorrhoea	1.16	7.8	2.54		EU	NO	NO	NO	NO
124	106649	37	P2L2	7	30		Menorrhagia	1.29	7.4	2.8		EU	PRESENT	NO	NO	NO