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Original Research Article

CLINICAL AND ANAESTHETIC RELEVANCE OF THYROID DYSFUNCTION IN ABNORMAL UTERINE BLEEDING - A RETROSPECTIVE STUDY

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ABSTRACT

BACKGROUND

Abnormal uterine bleeding (AUB) is a prevalent reason for gynecological outpatient visits and surgical interventions. Thyroid function, assessed through T3, T4, and TSH levels, is crucial in evaluating AUB cases, particularly when there is no prior thyroid disorder. Elevated TSH levels without abnormal T3 or T4, despite the absence of hypothyroidism symptoms, are often observed.

Our study explored hypothyroidism's clinical indicators like weight gain, BMI, and ECG changes such as QTc interval, correlating them with serum TSH levels. We have extensively discussed the link between thyroid function and estrogen receptors, subclinical hypothyroidism, myxedema coma, and non-thyroidal illness.

Despite high TSH levels (6-20 µIU/ml), within a range elevated with patient age, the absence of clinical hypothyroidism symptoms raised questions about optimal treatment before anesthesia. The study underscored the body's increased thyroid hormone demand during stress responses to surgery, fearing potential myxedema from untreated thyroid hypo-function.

Research investigated physiological, biochemical, and genetic factors contributing to elevated TSH, aligning with clinical scoring criteria for hypothyroidism and non-thyroidal illness. Analysis focused on BMI, QTc interval in ECG, and thyroid profile sensitivity and specificity in detecting abnormal TSH levels.

OBJECTIVES :

• To analyse T3,T4,TSH,BMI and QTc in ECG in AUB.

• To establish clinical insignificance of abnormal TSH values.

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METHODS
A retrospective descriptive study was conducted hospital name, analyzing records of patients from September 2022 to November 2023. All patients with abnormal uterine bleeding who presented for PAC from Sep 22 to Nov 23 for laparoscopic vaginal hysterectomy.

RESULT
This retrospective analysis of 40 AUB patients from September 2022 to November 2023 examined T3, T4, TSH, BMI, and QTc intervals. Results indicated standard deviations of 20.85 for T3, 2.78 for T4, and 2.654 for TSH. Correlations between QTc interval and TSH yielded a p-value of 0.17, and between BMI and TSH an R-value of 0.17 with a p-value of 0.21.
The study aimed to establish the clinical insignificance of isolated abnormal TSH values, highlighting complexities in anesthesia management due to non-symptomatic thyroid abnormalities. It recommended further research into optimal pre-operative thyroid hormone management protocols.

CONCLUSION
In conclusion, while elevated TSH levels in AUB patients without hypothyroid symptoms raised concerns, their clinical significance in anesthesia settings remains unclear, necessitating cautious pre-operative assessment and tailored management strategies.

KEYWORDS
TSH, Hypothyroidism, Subclinical, Abnormal Uterine Bleeding, Hormone Receptor Interaction

INTRODUCTION
Thyroid dysfunction has been extensively evaluated and considered as an attributive factor or a co-existing factor in variety of obstetric and gynecological situations ranging from infertility, pregnancy, fetal growth, menorrhagia, inter menstrual bleeding and perimenopausal uterine bleeding.

It is well known that estrogen has indirect effect on thyroid economy were all kept in mind and elaborate reference were searched in endocrinology, gynecology and anesthetic textbook and journals.

Apart from absence of clinical features one physical parameters namely body mass index, one biochemical parameter namely thyroid profile and one electrophysiological parameter namely the corrected QT interval in ECG were taken up their high sensitivity and specificity.parameters we analyzed retrospectively and results were statistically evaluated.
Abnormal uterine bleeding is the commonest gynecological complaint for which patient present to the gynecology outpatient department as well as for minor and major surgical procedures for tissue diagnosis and definitive surgical management.

The thyroid biochemistry is assessed by T3, T4 and TSH levels. The abnormal uterine bleeding in the absence of preexisting organic thyroid dysfunction should raise TSH with normal T3, T4 levels. The raised TSH was not associated with any clinical signs of hypothyroidism.

In this study the sensitive and specific features of hypothyroidism namely weight gain, BMI and ECG changes as QTc interval were noted and tabulated with serum TSH levels. Any delayed recovery from anesthesia, reduced requirement of anesthetics was also noted as the specific problems in a hypothyroid patient.

The association between thyroid and estrogen receptors, clinical features of hypothyroidism, the phenomena of non- thyroidal illness, subclinical hypothyroidism and myxedema coma are all elaborately discussed in this article.

The clinical insignificance of an isolated rise in TSH and absence of any pattern in induction and recovery from general anesthesia have been statistically evaluated in forty cases in our study.

A common observation in the evaluation process was a high thyroid stimulating hormone (TSH) value in spite of normal thyroid hormones and absence of any clinical features of hypothyroidism. The TSH value ranged from 6 µIU/ml to 20 µIU/ml (normal value - 5 to 8 µIU/ml). The range was further increased proportional to the age of the patient. The query whether this should be treated optimized before anesthesia had to be answered with no direct solutions being provided in standard textbooks. As the need for 2-4 times of thyroid function demand as stress response management by the human body in the event of anesthesia and surgery was well known, an organic thyroid hypo function indicated as raised TSH was feared to precipitate myxedema if untreated.

In this study the reasons for raised TSH value were probed into the physiological, biochemical and genetic perspectives. The clinical scoring of hypothyroidism, subclinical hypothyroidism and non-thyroid illness were all kept in mind and elaborate references were searched in endocrinology, gynecology and anesthetic textbooks and journals.

Apart from absence of clinical features one physical parameters namely body mass index, one biochemical parameter namely thyroid profile and one electrophysiological parameter namely the corrected QT interval in ECG were taken up for analysis with regard to their high sensitivity and specificity.

The parameters were analyzed retrospectively and the results were statistically evaluated.

Objectives

- To analyze T3, T4, TSH, BMI and QTc in ECG in AUB.
- To establish clinical insignificance of abnormal TSH values.

METHODS

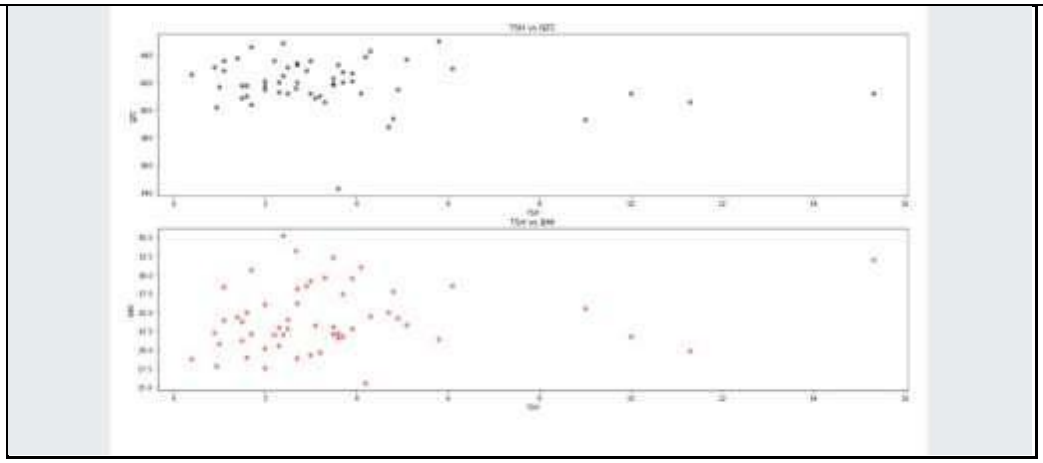
- Study: Retrospective
- Study population: All patients with abnormal uterine bleeding who presented for PAC from Sep 22 to Nov 23 for laparoscopic vaginal hysterectomy
- Sample size: 40
- Exclusion criteria: ASA 3 and 4 and known hypothyroidism

The values of BMI, QTc in ECG, T3, T4, TSH were noted for40 patients from PAC charts. All patients have successfully undergone LAVH under GA. There was no delayed recovery or difficult intubation and only 6 of them had a small goiter.

One clinical parameter, one biochemical parameter and one investigation to note a slow metabolic rate were evaluated in all the patients with isolated raised TSH with no prior thyroid illness.

The clinical parameter was taken as an indication of weight gain namely body mass index. The biochemical parameter was the serum thyroid profile in the patients. The definite and simple indication of a slow metabolic rate and a indication of impending tachy or bradyarrythymia namely the QT interval from the electrocardiogram were noted in the patients.

RESULTS



Graph 1 : TSH versus QTc and TSH versus BMI

- The standard deviation for T3 was 20.85, T4 was 2.78 and TSH was 2.654.
- On correlating QTc and TSH the p-value was 0.17 and BMI & TSH R value was 0.17 and p-value was 0.21.

S. No	Age	Ht cms	Wt kgs	BMI	TFT			Goitre	Ankle Jerk	Delayed Recovery	QTC
					T3	T4	TSH				
1	38	156	58	23.8	1.4	8.5	1.5	-	-	-	418
2	42	146	48	19.7	1.1	5.4	3.2	-	-	-	410
3	45	158	55	22.8	1.3	9.2	3.9	+	-	-	427
4	45	176	76	24.5	1.3	8.5	4.3	-	-	-	443
5	42	146	52	24.4	1.2	7.3	1.4	-	-	-	438
6	40	145	49	23.3	1.4	7.3	5.1	-	Unsustained Clonus of (Rt) LL	-	437
7	42	150	59	26.2	1.6	7.4	2.7	-	-	-	420
8	42	154	47	19.8	1.1	7.2	11.3	+	-	-	406
9	44	156	53	21.8	1.4	6.4	10	-	-	-	412
10	45	152	56	24.2	1	7.5	4.9	-	-	-	415
11	41	170	51	17.6	1.5	9.4	2	-	-	-	418
12	46	151	64	28.1	1.6	8.5	2.7	-	-	-	433
13	47	154	53	22.3	0.7	16.4	0.9	-	-	-	431

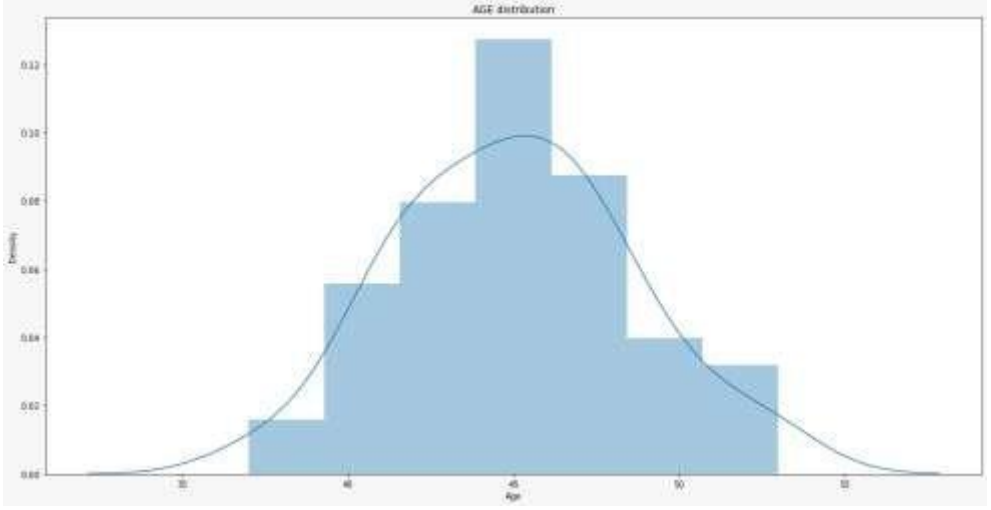
14	47	148	77	35.2	1.2	8.3	2.4	-	-	-	449
15	47	154	66	27.8	1.4	8.9	4.8	-	-	-	394
16	40	151	44	19.3	1.4	11	3	-	-	-	412
17	50	151	70	30.7	1.5	13.1	1.7	-	-	-	446

Table 1A : Age Distribution

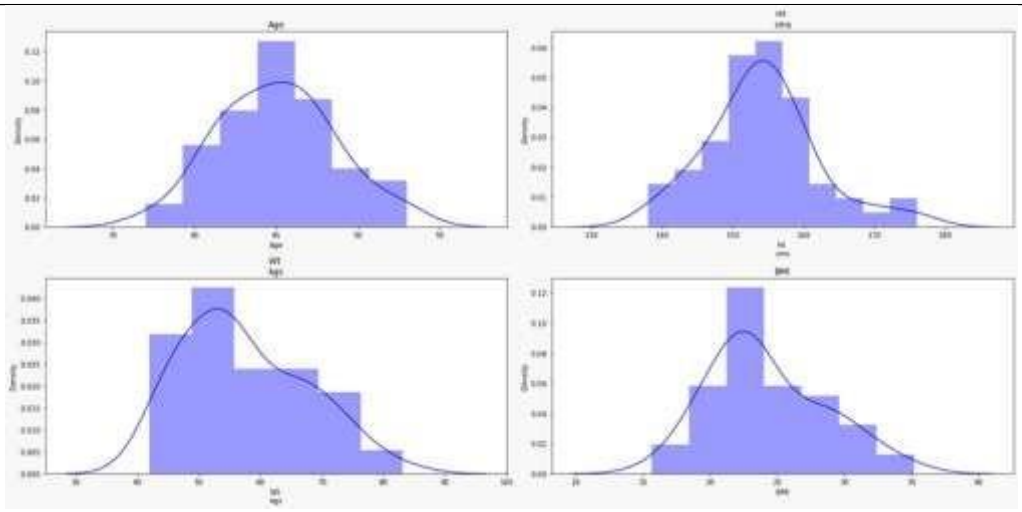
S. No	Age	Ht cms	Wtkgs	BMI	TFT			Goitre	Ankle Jerk	Delayed Recovery	QTC
					T3	T4	TSH				
1	46	155	53	22.1	3.1	2.2	3.5	-	-	-	423
2	44	153	75	32	1.7	7.9	15.3	-	-	-	412
3	47	154	57	24	1.5	9.9	2.5	-	-	-	431
4	41	148	45	20.5	1.4	8	2.3	-	-	-	420
5	37	140	50	25.5	1.2	6.3	9	-	-	-	393
6	49	159	45	17.8	1.1	1.17	0.94	-	-	-	402
7	45	161	54	20.8	1.4	8.6	1	-	-	-	417
8	45	153	56	23.9	1.4	7.3	1.1	-	-	-	436
9	47	160	48	18.8	1.3	10.3	0.4	-	-	-	426
10	47	168	65	23	2.75	1.2	2.3	-	-	-	413
11	43	138	42	22.1	1.6	13	1.7	-	-	-	404
12	46	150	64	28.4	1.7	13	1.1	-	-	-	429
13	44	143	45	22	1.3	9.6	2.4	-	-	-	425
14	44	145	45	21.4	1.2	9.2	5.8	-	-	-	450
15	48	153	54	23.1	1.3	9.2	3.5	-	-	-	419
16	51	158	65	26	1.2	10.1	2	-	-	-	415
17	50	159	72	28.5	1.3	9	2.9	-	-	-	429
18	41	143	66	32.3	1.6	9.7	3.5	-	-	-	419
19	52	154	70	29.5	1.6	11.2	3.9	-	-	-	421
20	47	162	72	27.4	1.5	10.7	3.7	-	-	-	428
21	47	155	51	21.2	1.3	6.2	1.5	-	-	-	409
22	53	156	71	29.2	1.2	5.8	3	-	-	-	436
23	48	148	68	31	1.4	9	4.1	-	-	-	412
24	42	160	64	25	1.2	7.2	4.7	-	-	-	388
25	45	153	67	28.6	1.5	10.3	6.1	-	-	-	430
26	49	152	44	19	1.2	8	1.6	-	-	-	418
27	43	152	50	21.6	1.3	7.8	3.6	-	-	-	433
28	43	156	54	22.2	1.2	7.8	3.6	-	-	-	343
29	49	158	55	22	1.2	6.9	2.2	-	-	-	436
30	53	147	47	21.8	1	12.3	3.7	-	-	-	420
31	41	158	83.1	33.2	156	11.9	2.67	-	-	-	416
32	42	162	53	20.2	1.4	8.2	2	-	-	-	421
33	43	168	44	15.6	1.2	7.8	4.2	-	-	-	439

34	48	175	58	18.9	1.8	11.9	2.7	-	-	-	434
35	44	154	54	22.8	0.9	6.5	2.5	-	-	-	412
36	46	155	60	25	1.1	3.6	1.6	-	-	-	410
37	45	158	58	23.2	1.3	7.9	3.1	-	-	-	409
38	40	140	58	29.6	1.6	8.4	3.3	-	-	-	406

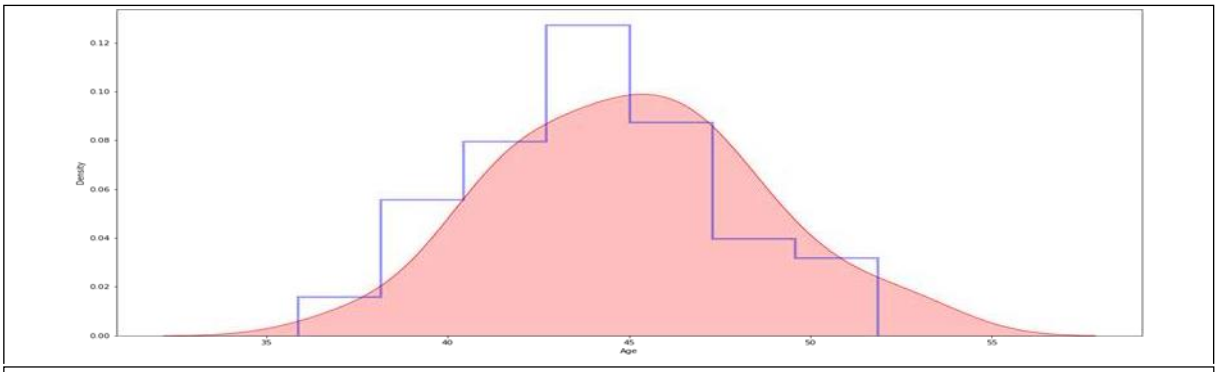
Table 1B : Age Distribution



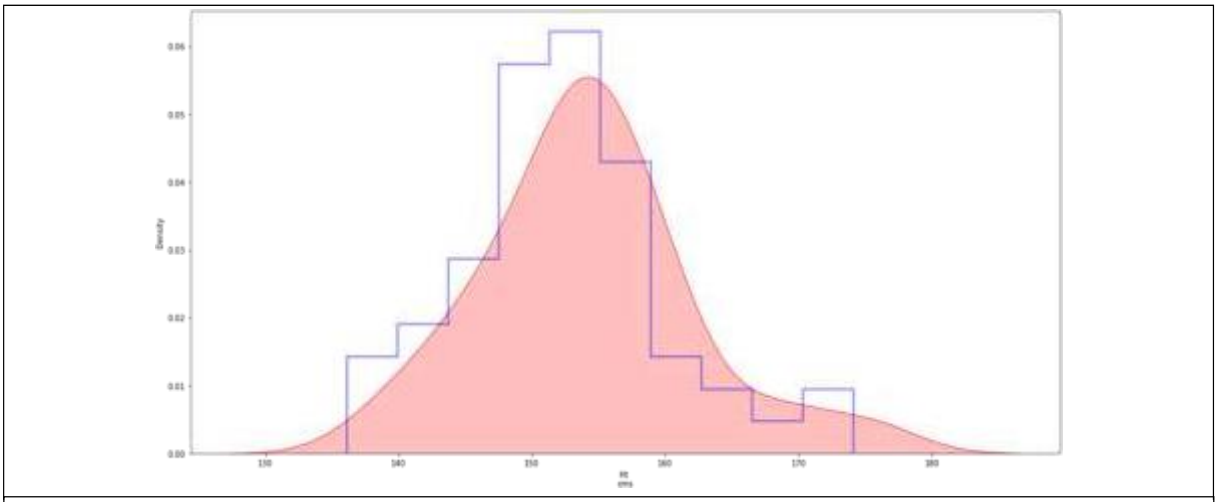
Graph 2 : Age Distribution



Graph 3 : Age, height, Weight and BMI Distribution

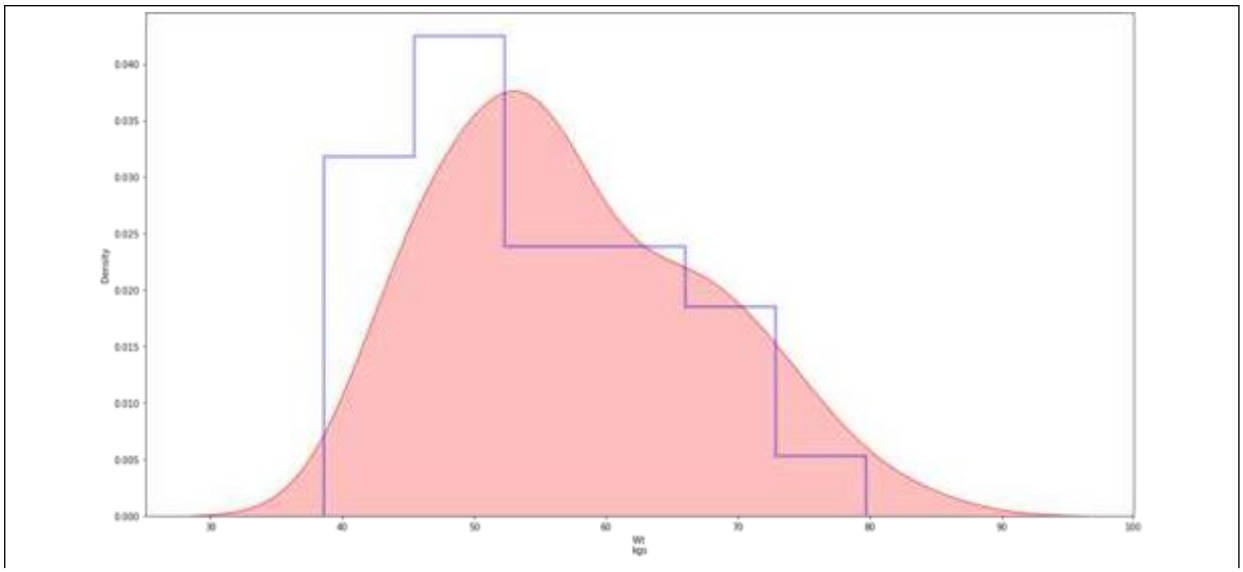


Graph 4 : Age Distribution

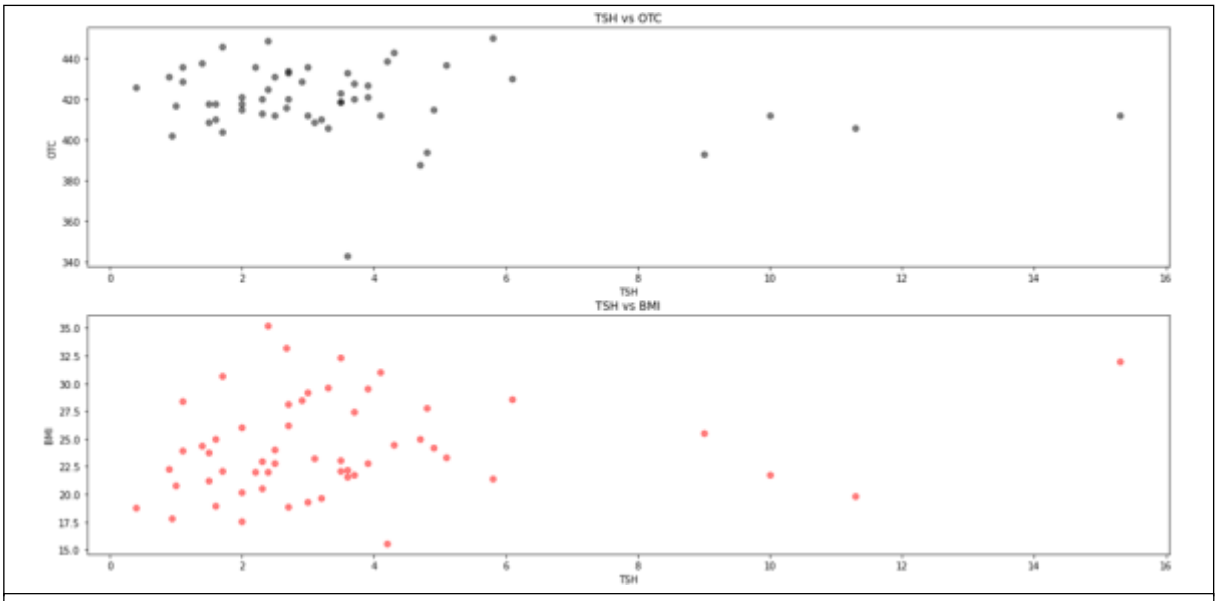


Graph 5 : Height Distribution

Weight Distribution



Graph 6 : Weight Distribution



Graph 7 : TSH versus QTc and TSH versus BMI

P AND R
QTc TSH
R-Value ---- -0.1848 P-Value ------0.1768
- - - - - - -
BMI TSH
R-Value ---- 0.1701 P-Value ------0.2145
- - - - - - -

DISCUSSION

In this discussion some points and explanation regarding thyroid gland, regulation of thyroid dysfunction including hypothyroidism and myxedema coma, subclinical hypothyroidism and mechanism involving various clinical features of hypothyroidism and non-thyroidal illness syndrome are all elaborated

- Thyroid hormone is involved in neuro endocrine control of reproduction.
- Estrogen increases the amount of TBG.
- There is molecule interaction between estrogen and thyroid receptor isoforms.
- Estrogen alpha receptor is present in thyroid gland.
- Estrogen dominance affects thyroid gland. Due to the above reasons TSH levels could be altered even without clinical significance.[1]

Thyroid dysfunction has been extensively evaluated and considered as an attributive factor or a coexisting factor in a variety of obstetric and gynecological situations ranging from infertility, pregnancy, fatal growth, menorrhagia, intermenstrual bleeding and postmenopausal uterine bleeding. It is well known that estrogen has indirect effect on thyroid economy.

The expression and ratios of two estrogen receptors alpha and beta that mediate genomic effects of estrogen on normal and abnormal thyroid tissue has been elaborately reviewed. Estrogen increases the thyroid binding globulin and the need for thyroid hormone in hypothyroid women.

Estrogen binding to estrogen alpha promotes cell proliferation and growth and that with estrogen beta promotes apoptotic actions and suppressive functions in thyroid tissue. 17-beta estradiol which is natural occurring binds to estrogen receptors to initiate genetic transcription on the domains.[2]

Significant interactions between thyroid and estrogen receptor exist. Estrogens are critical in the control of reproduction. The central role of estrogen in mammalian reproduction is reflected in neuroendocrine control of gonadotropin production from anterior pituitary and feedback regulation of gonadotrophin releasing hormone in the hypothalamus.[2]

Thyroid

Main regulator of thyroid function is TSH - a glycoprotein synthesized from thyrotrophic cells in anterior pituitary in turn under control of thyrotrophin releasing hormone (TRH) by hypothalamus.

Hypothylmo - Pituitary - Thyroid Axis

- Hypothalamus is <1% of brain volume emerges from ventral caudal prosencephalon or diencephalon during 6th week of gestation.
- Hypothalamic neuronal bodies - paraventricular, supraoptic and arcuate nuclei.
- Median eminence of hypothalamus is the link between hypothalamus and pituitary, a two different neurosecretary systems.
- Magnocellular neurons - posterior pituitary
- Parvocellular neuron - anterior pituitary
- TRH regulating TSH - thyroid axis.
- Functions of pituitary are marked out by 6 cell types located in anterior and intermediate lobes pituitary gland.
  1. Corticotropes - adrenocorticotrophic hormone
  2. Thyrotropes - thyroid stimulating hormone
  3. Somatotropes - growth hormone
  4. Lactotropes - leutenising hormone, follicle stimulating hormone
  5. Melanotropes - melanocyte stimulating hormone.[3]

Hypothyroidism and Myxoedema Coma

The first step in the spontaneous development of hypothyroidism is a slight decrease in T4 levels that cause increased release of TSH. The decreased T4 results in a modest decrease in free T4 which still remains in the normal reference range but serum TSH increases to values above the upper normal limit because of the exquisite sensitivity of pituitary thyrotroph for circulating thyroid hormone. This is the subclinical hypothyroidism. The increase in TSH induces preferential thyroid secretion of T3 by stimulating the synthesis of T3 more than T4 by increasing thyroidal 5' mono iodination of T4 into T3. The fractional conversion rate of T4 to T3 in extra thyroidal tissues (notably brain) increases. These result in a relative over production of T3 compared to T4 and serve (in view of greater biological potency of T3 than T4) to restrict the impact of thyroid hormone deficiency in peripheral tissues. The preferential

T3 production explains the increased T3 in subclinical hypothyroidism. Later as the disease progresses, there is further rise in TSH but T3 becomes normal.[3,4]

Grades	Disease	Tsh	Free t4	T3
Grade I	Subclinica l hypothyroidism	Slight increase	Normal	Slight increase
Grade Ii	Mild hypothyroidism	Moderate increase	Low	Normal
Grade Iii	Overt hypothyoidism	Very high	Low	Low

Table 2 : Grades of Hypothyroidism

Grades of Hypothyroidism History

Hypothyroidism as a clinical syndrome was described in 1874 by Gull under the name myxedema (myx - mucin).

Causes of Hypothyroidism

Reduced T4 in central hypothyroidism is due to insufficient stimulation of thyroid by TSH. An absent TSH response to exogenous TRH suggests pituitary cause. A delayed TSH response to TRH denotes hypothalamic cause. Abnormal sialylation of TSH is related reduced biological activity of TSH leading to normal or raised TSH in central hypothyroidism. There is a usual normal nocturnal TSH surge which lost in central hypothyroidism. There is a loss of increase in TSH pulse amplitude not in pulse frequency.

In critically ill patients receiving dopamine, serum TSH and T4 production decrease by 60% and 56% respectively due to direct inhibition of pituitary TSH. Transient functional inhibition of TSH release is observed after withdrawal of long term levothyroxine suppressive therapy which may last for 6 weeks.

Glucocorticoid excess tampons pulsatile TSH release. Octreotide therapy inhibits TSH secretion but does not cause clinical hypothyroidism. High doses of bexarotene specific retinoid X receptor agonist used in cutaneous T cell lymphoma, strongly inhibits TSH secretion causing central hypothyroidism.[3]

Hypothyroidism - genetic and environmental factors direct the evolution of autoimmune reaction. Smoking decreases risk of developing thyroid peroxidase antibodies and hypothyroidism. Chronic autoimmune thyroiditis due to TSH receptor blocking antibodies. Spontaneous disappearance of such antibodies causes the hypothyroidism to revert spontaneously. Silent or painless thyroiditis and post party thyroiditis are variants of chronic autoimmune thyroiditis. Incidence being 4-6% for postpartum thyroiditis.[3]

Subclinical Hypothyroidism Other Causes

1. Infiltrative and infectious disease - cystinosis, progressive systemic sclerosis, amyloidosis, immunosuppression, previous chronic viral infections.
2. Congenital - loss of functional thyroid tissue (thyroid dysgenesis), functional defects in thyroid hormone synthesis - loss of function mutation in gene encoding for TSH receptor, sodium - iodine transporter, thyroglobulin, thyroperoxidase.
3. Nutritional iodine deficiency
4. Excess inorganic iodide (dietary) inhibits organification of iodide (Wolff-Chaikoff effect).

Thyroid gland has auto regulatory mechanism which inhibit thyroid transport in the presence of excess iodide.

Subacute /postpartum thyroiditis, chronic autoimmune thyroiditis prevents the auto regulation and patient land up with hypothyroidism.[5,6]

Drug Induced Hypothyroidism

Lithium, flavonoids, resorcinol, amiodarone play the etiology by inhibiting thyroidal iodide transport and release of T4 and T3.

Tyrosine kinase inhibitors like sunitinib also cause depressed thyroid function.

Hepatic and Cutaneous Hemangiomas

These express high levels of type 3 iodothyroxine 5'-deiodinase catalysing conversion of T4 and T3 to biologically inactive T3 and 3, 3'-T2. Thus 3-iodothyroxine-5'-deiodinase induced degradation of thyroid hormone causes hypothyroidism.

Clinical Features of Hypothyroidism and Underlying Mechanisms[7,8]

Clinical Features	Mechanism
Slowing of physical and mental activity and organ function	Loss of inhibitory effect of thyroid hormone on synthesis of hyaluronate, fibronectin and collagen leading to their deposition in tissues
Non pitting pedal oedema	Hydrophilic properties of glycosaminoglycans
Slow metabolism	Reduced energy consumption
Cold intolerance	Reduced thermogenesis
Increase in >10% of body weight	Water and salt retention with increase in body fat. Low adipocytokines such as leptin, adeponectin and resistin.
Glucose intolerance	Delayed glucose absorption from intestine insulin secretion
No hypoglycaemia in hypothyroidism except pituitary causes	Hepatic gluconeogenesis and glycolysis are normal
Impaired growth	Reduced synthesis and degradation of proteins
Increase in total cholesterol	Increase in LDL due to decreased expression of T3 responsive liver LDL receptor that is involved in LDL clearance
Atherogenic lipid profile	Increase oxidation of LDL , HDL is increased modestly
Dry, scaly rough thick skin	Reduced sebaceous and sweat gland
Pallor	Reduced skin blood flow
Yellow discoloration of palms and soles	Carotene deposition
Slow mentation	Reduced regional cerebral blood flow. Reduced cerebral glucose metabolism
Slow responsiveness (speech and motor	Low voltage EEG, prolonged central motor

actions), cognitive impairment	conduction time. Decreased SEP and long latency
Depressive illness	Reduced 5 HT - synthesis and turnover
Cerebellar ataxia, paresthesia, carpal tunnel syndrome	Increased TPO antibodies
Myalgia, cramps, fatiguability	Decrease phosphocreatine and increase serum creatine kinase
Slow DTJ (Peudomyotonic Effect)	Prolonged relaxation of Achillies tendon reflexes due to defective transition from fast white type II to slow red type I fibre in muscle bioenergetics.
Macroglossia	Type I fibre hypertrophy and type II fiber atrophy. Sarcoplasmic degeneration and interstitial edema.
Increase diastolic BP, decrease systolic BP, normal MAP, decrease CO, bradycardia	1. Endothelial dysfunction and impaired vascular relaxation. 2. T3 dependent myocardial gene expression is affected. 3. Genes coding calcium regulatory proteins are defective.
Bradypnoea, sleep apnoea, reduced ventilatory drive, airway obstruction	T3 dependent myogenic changes and obesity
Hyponatremia, increased body water, edema	Increased vascular permeability, extravascular accumulation of hydrophilic glucose aminoglycans, decreased free water clearance and increased serum AVP
Anovulatory cycles, menorrhagia, abnormal uterine bleeding	Action of TRH on gonadotrophs. Action of TSH on FSH receptors are abnormal

Table 3 : Clinical Features of Hypothyroidism and Underlying Mechanisms

Nonthyroidal Illness Syndrome

NTIS is a neuroendocrine response to illness, trauma and stress and is a part of general adaptation to the precipitating factors. NTIS is also known as sick euthyroid syndrome or low T3 syndrome. The fall in T3 and rise in reverse T3 are consistent feature.[9] The receptors for TSH, FSH and LH/CG are members of rhodopsin like G-protein coupled receptor family. TSH receptor has a serpentine domain containing seven transmembrane regions with many features typical of this receptor family. The relatively high sequence identity between the hormone binding domain of TSH, LH, CG receptors open the possibility of spillover phenomenon during pregnancy (normal or abnormal) and gynecological dysfunctions.

The glycoprotein hormone receptor of TSH dimerize in vivo and these dimers interact functionally in LH, CG receptors knockout situation.

Both the beta subunit of glycoprotein hormone and glycoprotein hormone receptors are encoded by paralogous genes. The gene coding for the human TSH receptor is located on the long arm of chromosome 14. Triplication of the ancestral glycoprotein hormone receptor gene and subsequent divergence lead to the receptor of TSH, FSH, LH, CG.

10 exons exist in both TSH and FSH receptor genes. TSH secretion is inhibited by high dose of glucocorticosteroids and endogenous hypercortisolemia. Glucocorticoids act in the paraventricular nucleus to inhibit TRH gene expression in vivo. But even prolonged

administration of high dose of glucocorticoids does not cause hypothyroidism. The reason is that the potent effect of low serum T3 T4 to raise TSH secretion overcomes glucocorticoid induced inhibition of TSH secretion.

Zulewski Muller Eher et al clinical score of hypothyroidisms. Weight increase had a specificity of 78% and positive predictive value of 71%. Delayed ankle jerk had a specificity of 94% and positive predictive value of 92% and sensitivity of 77% whereas other signs and symptoms such as constipation, hoarseness, slow movements, coarse skin lacked sensitivity and only mild to moderate specificity.

TSH	**Free T4**	**Diagnosis**
Normal	NORMAL	Euthyroid
Increased	Decreased	Primary Hypothyroidism
Increased	Normal	Subclinical Hypothyroidism
Increased	Increaesd	Thyroid Hormone Resistance
Decreased	Decreased	Central Hypothyroidism
Decreased	Normal or Increased	Subclinical Hyperthyroidism

Table 4 : Diagnosis with Free T4 and TSH levels

Thus the parameters T3,T4, TSH and BMI have been substantiated. The significance of QTc interval is discussed further.

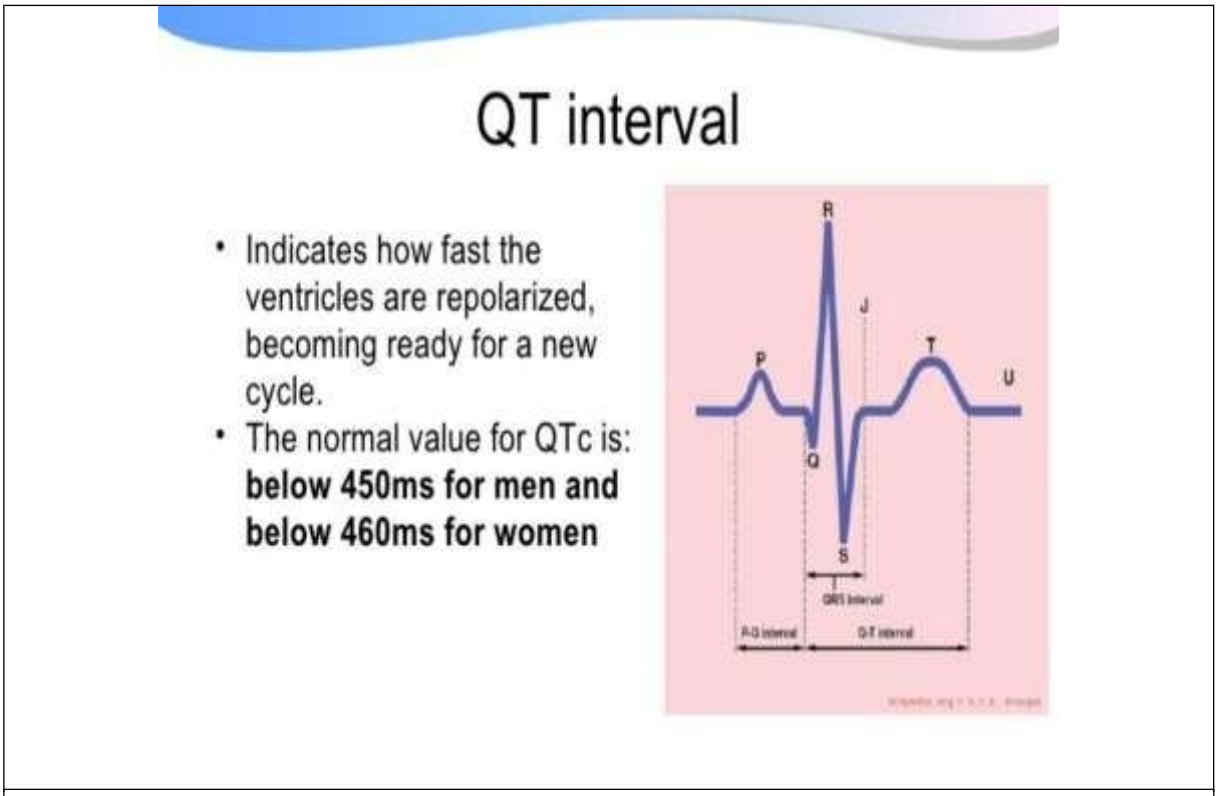
The QT interval is from the beginning of Q to the end of T wave and is approximately 0.4 seconds. This interval is variable with the heart rate; hence a corrected QT interval namely QTc has been devised and indicated in all the routine cardiograms.

Commonly three of several formulae have been used by several computerized electrocardiography machines and multi parameter monitors namely Bazett, Fridericia Framingham, Hodges or Rautaharju formulae have been used to devise correction factors,

QTc= QT/RR e, where e = 0.5 for the Bazett correction and 0.33 for Fridericia correction.

Commonly Bazett QTc correction factor is used in the software of the machines to give us the QT correction or QTc as it is denoted in our ECG reports. The QTc reduces the error in the interval caused because of a rapid or slow heart rate (R-R interval) on the QT interval.

This QTc has been included in our study. In this retrospective analysis the QTc from the ECGs of the sample population (using the Bazette formula) were noted, tabulated and analyzed.



Graph 8: QT Interval

CONCLUSION

Isolated rise of TSH values may not be clinically relevant and rarely need thyroid supplementation. The suspicious of hypothyroidism did not affect conduct and recovery of standard general anaesthesia for minor and major surgical procedures.

In the event of profuse menorrhagia, patient may be treated surgically (when medical management fails) at the earliest instead of waiting to optimize thyroid function for 6-8 weeks and prevent further anaemia and avoid further blood transfusions and subsequent reactions.

Safe and early definitive management is assured by standard and safe anaesthesia even in the scenario of raised TSH levels which are left to regress in spontaneous fashion.

REFERENCES

[1] Santin AP, Furlanetto TW. Role of estrogen in thyroid function and growth regulation. J Thyroid Res. 2011; 2011:875125.
[2] Vasudevan N, Ogawa S, Pfaff D. Estrogen and thyroid hormone receptor interactions: physiological flexibility by molecular specificity. Physiol Rev. 2002 Oct; 82(4):923-44.
[3] Werner & ingbar's THE THYROID A fundamental and clinical text Lewis e braverman & David s cooper 10th edition 2013 Causes of hypothyroidism pg. 523/ David s cooper Straub RH the complex role of estrogen in inflammation endocrine review 2007:28:521
[4] Leonard Wartofsky Myxedema coma p600:44
[5] Surks MI Ortiz E Daniels GH et al Subclinical thyroid disease JAMA 2004:291 228-238
[6] Douglas s. Ross Subclinical hypothyroidism 47:628
[7] Michael T. Mc Dermatt Overview of Clinical Manifestation of hypothyroidism chapter

39 p569
[8] Irwin L Klein - Sara Danzi Chapter 40 Cardiovascular system in hypothyroidism p 575.
[9] Leslie J. De Groot Nonthyroidal Illness syndrome 86:1623 Endocrinology 6th Edition 2010.