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Original Research Article

Serum Phosphorus and the Calcium–Phosphorus Balance in Newly Diagnosed Overt Hypothyroidism: A Comparative Study with Euthyroid Controls

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Abstract

Background: Phosphorus, together with calcium, forms the mineral basis of the skeleton and participates in energy transfer, membrane structure and acid–base buffering. Its serum concentration is regulated principally by parathyroid hormone and vitamin D acting on renal tubular reabsorption, processes upon which thyroid hormones exert a modulatory influence. Whereas hypocalcaemia in hypothyroidism is comparatively well documented, the behaviour of serum phosphorus remains contested, with published series variously reporting hyperphosphataemia, hypophosphataemia or no change at all.

Objectives: To compare serum phosphorus concentrations between newly diagnosed overt hypothyroid patients and age- and sex-matched euthyroid individuals, to examine the correlation between serum thyroid-stimulating hormone and serum phosphorus, and to characterise the resulting shift in the calcium–phosphorus balance.

Materials and Methods: A hospital-based comparative observational study was conducted in the Department of General Medicine, McGann Teaching District Hospital, Shivamogga Institute of Medical Sciences, Shivamogga, over 12 months. One hundred and sixty subjects aged above 18 years were enrolled after institutional ethics committee clearance and informed consent — 80 with newly diagnosed overt hypothyroidism and 80 age- and sex-matched euthyroid controls. Subjects with chronic kidney disease, hepatic disease, prior thyroid surgery or receiving mineral supplementation were excluded, since each independently disturbs phosphate handling. Two millilitres of venous blood was collected in a plain vacutainer, allowed to clot, centrifuged, and the serum stored at 4–8 °C until analysis. Serum free T3, free T4 and thyroid-stimulating hormone were estimated on a Cobas e411 analyser and serum calcium and phosphorus on a Cobas c311 analyser. The unpaired Student's t-test and Pearson's correlation coefficient were applied, with $p<0.05$ taken as significant.

Results: The two groups were identically matched for sex, each containing 63 women and 17 men, with an overall mean age of 46.49 ± 12.87 years. Group allocation was confirmed biochemically, thyroid-stimulating hormone being 24.19 ± 13.70 versus 2.89 ± 1.30 µIU/mL and free T4 0.64 ± 0.20 versus 1.18 ± 0.19 ng/dL (both $p<0.0001$). Mean serum phosphorus was significantly higher in the hypothyroid group at 4.48 ± 0.42 mg/dL compared with 3.69 ± 0.60 mg/dL in controls — an absolute rise of 0.79 mg/dL, or 21.4% above the control mean ($t = 9.12$, $p<0.0001$), corresponding to a large effect size (Cohen's $d = 1.53$). Thyroid-stimulating hormone correlated positively and significantly with serum phosphorus ($r = 0.412$, $p<0.0001$), accounting for approximately 17% of its variance. Because serum calcium simultaneously fell from 9.59 to 8.51 mg/dL, the calcium-to-phosphorus ratio derived from group means contracted markedly from 2.60 in euthyroid controls to 1.90 in hypothyroid patients.

Conclusion: Newly diagnosed overt hypothyroidism was associated with significant hyperphosphataemia that varied in proportion to the degree of thyroid-stimulating hormone elevation. Combined with the concurrent fall in calcium, this produced a substantial contraction of the calcium-to-phosphorus ratio, indicating that the two minerals move in opposite directions and that neither should be interpreted in isolation.

Keywords: Hypothyroidism; serum phosphorus; hyperphosphataemia; calcium–phosphorus ratio; thyroid-stimulating hormone; mineral metabolism.

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Introduction

Hypothyroidism is among the commonest endocrine disorders encountered in clinical practice, with a reported global prevalence of between 4% and 21% depending on iodine sufficiency, geography and genetic background [1,2]. In India, a multicentre survey across eight cities estimated adult prevalence at approximately 10.95% [3].

Because thyroid hormones influence virtually every metabolic pathway, the systemic consequences of thyroid failure extend well beyond the classical clinical syndrome, and disturbances of mineral homeostasis are prominent among them [4].

Phosphorus is indispensable to skeletal mineralisation, cellular energy transfer through adenosine triphosphate, membrane phospholipid structure, nucleic acid synthesis and acid–base buffering [5]. Its serum concentration is maintained within narrow limits by an endocrine network in which parathyroid hormone promotes renal phosphate excretion, while 1,25-dihydroxyvitamin D enhances intestinal absorption; thyroid hormones modulate this system at several points [6,7]. In consequence, thyroid dysfunction would be expected to disturb phosphate balance, and disturbances of the calcium–phosphorus axis have been linked to metabolic bone disorders including osteoporosis, osteomalacia and secondary hyperparathyroidism [8].

The mechanisms proposed for hyperphosphataemia in hypothyroidism are several and mutually reinforcing. Thyroid hormone deficiency reduces the glomerular filtration rate and renal plasma flow, diminishing filtered phosphate load clearance [4]. Reduced bone turnover, arising from suppressed osteoblastic and osteoclastic activity, lowers the incorporation of phosphate into newly formed bone matrix [9]. Altered parathyroid hormone secretion and disturbed vitamin D metabolism further modify renal tubular reabsorption of phosphate [10,11]. The net effect is retention of phosphate in the extracellular compartment.

Yet empirical findings are strikingly inconsistent. While hypocalcaemia in hypothyroidism is comparatively well documented, the direction of change in phosphorus remains contested, with published series reporting hyperphosphataemia, hypophosphataemia or no significant alteration [12,13]. Athokpham et al. found markedly raised phosphorus in hypothyroid subjects, rising from 3.49 to 5.46 mg/dL [14], and Srinivas et al. reported an increase from 3.32 to 4.71 mg/dL [15]. Sridevi et al. likewise observed elevation, though they found no significant correlation between thyroid-stimulating hormone and phosphorus, reporting a weakly negative coefficient [16]. Saxena et al. documented a more modest rise in median values [17]. Jat et al., assessing minerals across subclinical and overt disease, and Charak et al., examining 419 subjects with both hypo- and hyperthyroidism, both emphasised the value of evaluating phosphorus alongside calcium rather than in isolation [18,19].

This inconsistency is clinically consequential. Because calcium and phosphorus are reciprocally regulated, an isolated measurement of either may be uninformative or actively misleading; it is the balance between them that reflects the underlying endocrine disturbance. Yet few studies quantify this balance explicitly, and fewer still examine whether the magnitude of phosphate retention varies with the severity of thyroid failure as indexed by thyroid-stimulating hormone. Establishing a graded relationship would strengthen the case for a genuine biological effect rather than a categorical association confounded by group differences.

The present study was therefore undertaken to compare serum phosphorus concentrations between newly diagnosed overt hypothyroid patients and age- and sex-matched euthyroid controls at a tertiary care hospital, to examine the correlation between thyroid-stimulating hormone and serum phosphorus, and to characterise the resulting shift in the calcium-to-phosphorus balance, thereby providing an integrated view of mineral disturbance in overt thyroid failure.

Materials and Methods

This hospital-based comparative observational study was conducted in the Department of General Medicine, McGann Teaching District Hospital, Shivamogga Institute of Medical Sciences, Shivamogga, Karnataka, and included both outpatient attendees and admitted patients.

Study duration: The study was carried out over a period of 12 months.

Ethical considerations: Institutional Ethics Committee clearance was obtained prior to commencement. Written informed consent was taken from every participant before enrolment, and confidentiality was maintained throughout.

Study population and grouping: A total of 160 subjects were enrolled and allocated to two groups of 80 each — newly diagnosed overt hypothyroidism, and euthyroid controls matched to the cases for age and gender.

Inclusion criteria: Subjects aged above 18 years with newly diagnosed overt hypothyroidism

constituted the case group, and age- and gender-matched euthyroid individuals the control group.

Exclusion criteria: Subjects with chronic kidney disease were excluded, since impaired glomerular filtration is itself the commonest cause of phosphate retention and would confound the primary comparison. Those with hepatic disease, those receiving mineral supplementation, and those who had undergone thyroid surgery were likewise excluded.

Sample size estimation: Sample size was derived from a previous study using an anticipated between-group difference in mean serum phosphorus of 0.4 mg/dL with a pooled standard deviation of 0.73. With an alpha error of 5% and 80% power, the minimum requirement was 52 subjects per group. Eighty were recruited into each group, providing power in excess of that specified.

Sample collection and handling: After informed consent, 2 mL of venous blood was drawn from each participant into a plain vacutainer. Samples were allowed to clot, centrifuged to separate serum, and the serum stored at 4–8 °C until analysis. Identical pre-analytical handling was applied to both groups, an important consideration for phosphate estimation given its susceptibility to release from cellular elements on delayed separation.

Biochemical estimation: Serum free T3, free T4 and thyroid-stimulating hormone were estimated using the fully automated Cobas e411 analyser. Serum calcium and serum phosphorus were estimated using the fully automated Cobas c311 analyser. Overt hypothyroidism was defined biochemically by elevated thyroid-stimulating hormone with reduced free T4, and euthyroidism by thyroid function within the reference range.

Study variables: The primary outcome variable was serum phosphorus concentration. Secondary variables comprised serum calcium, thyroid-stimulating hormone, free T3 and free T4, together with age and sex, recorded to verify adequacy of matching. Two derived indices were computed from the group means to characterise mineral balance: the calcium-to-phosphorus ratio and the calcium × phosphorus product. Because individual-level data were not available for these derivations, both indices were calculated from group mean values and are presented as descriptive summaries without inferential testing.

Statistical analysis: Data were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. An unpaired Student's t-test was used to compare parameters between cases and controls, with the t statistic and p value reported. Pearson's correlation coefficient assessed the relationship between the thyroid profile and serum phosphorus. A p value below 0.05 was considered statistically significant and below 0.0001 highly significant. Standardised mean differences (Cohen's d) were derived from the reported means and standard deviations to express the magnitude of between-group differences independently of sample size, and the coefficient of determination was calculated to quantify the proportion of variance shared between thyroid-stimulating hormone and serum phosphorus.

Results

One hundred and sixty subjects were studied, comprising 80 with newly diagnosed overt hypothyroidism and 80 euthyroid controls.

Table 1: Baseline characteristics of the two study groups

Characteristic	Euthyroid (n = 80)	Hypothyroid (n = 80)	Total (n = 160)
Female, n (%)	63 (78.75)	63 (78.75)	126 (78.75)
Male, n (%)	17 (21.25)	17 (21.25)	34 (21.25)
Mean age of whole cohort (years)	—	—	46.49 ± 12.87
Aged 40–62 years, n (%)	—	—	83 (51.88)
Aged 18–29 years, n (%)	—	—	24 (15.00)

The two groups were identically constituted with respect to sex, each containing 63 women and 17 men, and were matched for age by design.

Any between-group difference in serum phosphorus therefore cannot be attributed to differing age or sex composition — a methodological point of some importance, since both variables independently influence phosphate handling, phosphorus being higher in younger subjects and in men.

The cohort as a whole was predominantly middle-aged, with 83 subjects (51.88%) between 40 and 62 years and a mean age of 46.49 ± 12.87 years, and overwhelmingly female at 78.75%.

Table 2: Thyroid function profile confirming group allocation

Parameter	Euthyroid (n = 80) Mean ± SD	Hypothyroid (n = 80) Mean ± SD	Change	t statistic	p value
TSH (μIU/mL)	2.89 ± 1.30	24.19 ± 13.70	+737.0%	13.84	<0.0001
Free T3 (pg/mL)	2.91 ± 0.42	1.96 ± 0.69	−32.6%	10.52	<0.0001
Free T4 (ng/dL)	1.18 ± 0.19	0.64 ± 0.20	−45.8%	17.50	<0.0001

The biochemical distinction between groups was unambiguous, satisfying the definition of overt rather than subclinical hypothyroidism, since both the thyroid-stimulating hormone elevation and the free T4 reduction were substantial. Thyroid-stimulating hormone rose more than eightfold while free T4 fell by almost half.

The wide standard deviation of thyroid-stimulating hormone in the hypothyroid group (13.70 against 1.30 in controls) reflects a broad range of disease severity within that group, which is what makes the correlation analysis in Table 5 meaningful rather than merely restating the group difference.

Table 3: Comparison of serum phosphorus between groups

Parameter	Euthyroid (n = 80) Mean ± SD	Hypothyroid (n = 80) Mean ± SD	Mean difference	t statistic	p value
Serum phosphorus (mg/dL)	3.69 ± 0.60	4.48 ± 0.42	+0.79 (+21.4%)	9.12	<0.0001
Standardised effect size (Cohen's d)	—	—	**1.53**	—	—

This table addresses the primary objective. Mean serum phosphorus was 4.48 ± 0.42 mg/dL in the hypothyroid group against 3.69 ± 0.60 mg/dL in controls, an absolute increase of 0.79 mg/dL representing a 21.4% rise above the control mean, highly significant at p<0.0001. The standardised effect size of 1.53 places this in the large category, indicating limited overlap between the two distributions. A subtler observation concerns the standard deviations: variability was appreciably *narrower* in the hypothyroid group (0.42) than in controls (0.60). This suggests that phosphate retention in overt hypothyroidism produces a consistent upward shift affecting essentially the whole group rather than a scattered response confined to the most severely affected individuals.

Table 4: Derived calcium–phosphorus balance indices

Index	Euthyroid	Hypothyroid	Change
Serum calcium (mg/dL)	9.59 ± 0.52	8.51 ± 0.55	−1.08 (−11.3%)
Serum phosphorus (mg/dL)	3.69 ± 0.60	4.48 ± 0.42	+0.79 (+21.4%)
Calcium-to-phosphorus ratio*	2.60	1.90	−0.70 (−26.9%)
Calcium × phosphorus product* (mg^2/dL^2)	35.4	38.1	+2.7 (+7.7%)

***Derived from group mean values, not from individual paired measurements; presented descriptively without inferential testing.**

This table integrates the two minerals and reveals what neither shows alone. Calcium fell by 11.3% while phosphorus rose by 21.4% — movements in opposite directions that compound one another.

The calcium-to-phosphorus ratio consequently contracted from 2.60 to 1.90, a relative reduction of 26.9%, more than twice the proportional change in calcium alone. The ratio is thus a considerably more sensitive descriptor of the mineral disturbance than either constituent measured in isolation. By contrast, the calcium × phosphorus product rose only modestly, by 7.7%, because the fall in calcium partly offsets the rise in phosphorus; this index is therefore comparatively insensitive to the reciprocal shift characteristic of hypothyroidism, in contrast to its established utility in renal bone disease where both minerals may rise together.

Table 5: Correlation between thyroid-stimulating hormone and serum phosphorus (n = 160)

Correlation	Pearson's r	Coefficient of determination (r^2)	Shared variance (%)	p value
TSH versus serum phosphorus	+0.412	0.170	17.0	<0.0001
TSH versus serum calcium (for comparison)	−0.539	0.291	29.1	<0.0001

Thyroid-stimulating hormone correlated positively and significantly with serum phosphorus (r = 0.412, p<0.0001), confirming that phosphate retention is graded with the severity of thyroid failure rather than being simply a categorical feature of the hypothyroid state.

The relationship is of moderate strength, thyroid-stimulating hormone sharing approximately 17% of

the variance in phosphorus. Notably, this is weaker than the corresponding inverse correlation with calcium (r = −0.539, 29.1% shared variance), indicating that calcium tracks the degree of thyroid failure more closely than phosphorus does.

The 83% of phosphorus variance left unexplained is consistent with the dominant influence of renal function, parathyroid hormone and dietary phosphate intake, none of which were measured here.

Table 6: Comparison of serum phosphorus findings with published studies

Study	Euthyroid phosphorus (mg/dL)	Hypothyroid phosphorus (mg/dL)	Difference	p value
Present study	**3.69 ± 0.60**	**4.48 ± 0.42**	**+0.79**	**<0.0001**
Srinivas et al. (2021) [15]	3.32 ± 0.61	4.71 ± 0.97	+1.39	<0.001
Athokpham et al. (2020) [14]	3.49 ± 0.69	5.46 ± 0.45	+1.97	<0.001
Sridevi et al. (2016) [16]	3.62 ± 0.62	4.39 ± 0.38	+0.77	<0.0001
Saxena et al. (2020) [17]	3.8 (3.5–4.1)*	4.3 (1.7–6.2)*	+0.5	0.00

***Median with range.**

All five studies report phosphorus to be elevated in hypothyroidism, and the present increase of 0.79 mg/dL corresponds almost exactly to the 0.77 mg/dL of Sridevi et al. It is more conservative than the 1.39 mg/dL of Srinivas et al. and considerably smaller than the 1.97 mg/dL of Athokpham et al., whose hypothyroid mean of 5.46 mg/dL is the highest reported. Euthyroid control values cluster tightly between 3.32 and 3.8 mg/dL across all series, so the divergence lies almost entirely in the hypothyroid groups and most plausibly reflects differing severity of thyroid failure between cohorts. The consistent direction of effect across geographically and methodologically distinct studies establishes hyperphosphataemia as a reproducible feature of overt hypothyroidism.

Discussion

The present study demonstrates significant hyperphosphataemia in newly diagnosed overt hypothyroidism, serum phosphorus rising from a control mean of 3.69 ± 0.60 to 4.48 ± 0.42 mg/dL ($p<0.0001$). This is a 21.4% increase with a large standardised effect size of 1.53, indicating a substantial and consistent shift rather than a marginal statistical finding.

The direction of effect accords with the majority of published work. Srinivas et al. reported an increase from 3.32 to 4.71 mg/dL [15] and Athokpham et al. a larger rise from 3.49 to 5.46 mg/dL [14], while Sridevi et al. observed an increment of 0.77 mg/dL almost identical to that found here [16]. Saxena et al. documented a more modest elevation in median values [17]. Jat et al. and Charak et al. similarly emphasised the value of assessing phosphorus alongside calcium in thyroid dysfunction [18,19]. The mechanistic basis is well described: thyroid hormone deficiency reduces glomerular filtration and renal plasma flow, impairing phosphate clearance, while suppressed bone turnover lowers phosphate incorporation into matrix, and altered parathyroid hormone and vitamin D signalling modify tubular reabsorption [4,9,10].

The positive correlation between thyroid-stimulating hormone and phosphorus ($r = 0.412$, $p<0.0001$) confirms a graded relationship. Srinivas et al. reported a far stronger coefficient of 0.8755 [15] and Athokpham et al. 0.927 [14]. Notably, Sridevi et al. found no significant relationship, reporting a weakly negative r of −0.08 [16] — a discordance that may reflect their smaller sample of 80 and different distribution of disease severity, and which illustrates why replication across cohorts matters.

The integrated finding is arguably the most instructive. Because calcium fell by 11.3% while phosphorus rose by 21.4%, the calcium-to-phosphorus ratio contracted by 26.9%, from 2.60 to 1.90. This reciprocal movement amplifies the signal: the ratio changes proportionally more than either mineral alone, making it a more sensitive descriptor of the underlying endocrine disturbance. The calcium × phosphorus product, by contrast, rose only 7.7%, since the opposing movements largely cancel — a reminder that an index validated in renal bone disease, where both minerals may rise together, does not transfer straightforwardly to hypothyroidism.

That thyroid-stimulating hormone shared 29.1% of the variance in calcium but only 17.0% in phosphorus suggests calcium is the more closely coupled of the two to thyroid status, with phosphorus subject to stronger competing influences from renal function and dietary intake. This asymmetry has not been widely commented upon and merits examination in larger cohorts.

Limitations: Parathyroid hormone, 25-hydroxyvitamin D and creatinine were not measured, so the relative contribution of reduced glomerular filtration, altered parathyroid activity and vitamin D status could not be apportioned. Serum albumin was unavailable, leaving calcium uncorrected.

The derived ratio and product were computed from group means rather than individual paired values

and are therefore descriptive only, without confidence intervals or significance testing. The correlations were calculated across the pooled cohort, so the group difference itself contributes to the coefficients. Finally, no post-treatment reassessment was performed, so reversibility with levothyroxine remains untested.

Conclusion

In this comparative study of 160 subjects — 80 with newly diagnosed overt hypothyroidism and 80 age- and sex-matched euthyroid controls — serum phosphorus was significantly higher in the hypothyroid group at 4.48 ± 0.42 mg/dL against 3.69 ± 0.60 mg/dL ($p<0.0001$). The absolute rise of 0.79 mg/dL represents a 21.4% increase with a large standardised effect size of 1.53.

Variability was narrower in the hypothyroid group than in controls, indicating a consistent upward shift across the group rather than a response confined to the most severely affected.

Phosphate retention was graded with the severity of thyroid failure, thyroid-stimulating hormone correlating positively with serum phosphorus ($r = 0.412$, $p<0.0001$) and accounting for approximately 17% of its variance. This is appreciably weaker than the corresponding inverse correlation with calcium ($r = −0.539$, 29.1% shared variance), indicating that calcium is the more tightly coupled of the two minerals to thyroid status while phosphorus is subject to stronger competing influences from renal handling and dietary intake.

The integrated picture is the principal message. Calcium fell by 11.3% while phosphorus rose by 21.4%, so that the calcium-to-phosphorus ratio contracted by 26.9%, from 2.60 to 1.90 — a proportional change more than twice that of calcium alone. The two minerals move in opposite directions in overt hypothyroidism, and neither should be interpreted in isolation. The calcium × phosphorus product, rising only 7.7%, proved comparatively insensitive here because the opposing movements largely offset one another.

Clinically, these findings support paired measurement of calcium and phosphorus at the time of diagnosis of overt hypothyroidism, with attention to the ratio between them rather than to either value alone. Future studies should incorporate parathyroid hormone, 25-hydroxyvitamin D, serum creatinine and albumin to apportion mechanism, should compute mineral indices from individual paired measurements to permit formal inference, and should reassess patients after restoration of euthyroidism to establish whether the disturbance is reversible with adequate levothyroxine replacement.

References

1. Chaker L, Razvi S, Bensenor IM, Azizi F, Pearce EN, Peeters RP. Hypothyroidism. Nat Rev Dis Primers. 2022;8(1):30.
2. Canaris GJ, Manowitz NR, Mayor G, Ridgway EC. The Colorado thyroid disease prevalence study. Arch Intern Med. 2000;160(4):526–34.
3. Unnikrishnan AG, Kalra S, Sahay RK, Bantwal G, John M, Tewari N. Prevalence of hypothyroidism in adults: an epidemiological study in eight cities of India. Indian J Endocrinol Metab. 2013;17(4):647–52.
4. Dhanwal DK. Thyroid disorders and bone mineral metabolism. Indian J Endocrinol Metab. 2011;15(Suppl 2):S107–12.
5. Ciosek Z, Kot K, Kosik-Bogacka D, Lanocha-Arendarczyk N, Rotter I. The effects of calcium, magnesium, phosphorus, fluoride, and lead on bone tissue. Biomolecules. 2021;11(4):506.
6. Anandkumar S, Chacko J, Theertha CK, Usha M. Thyroid disorder: an overview. Res J Pharmacol Pharmacodyn. 2020;12(1):1–4.
7. Bilezikian JP, Khan A, Potts JT, Brandi ML, Clarke BL, Shoback D, et al. Hypoparathyroidism in the adult: epidemiology, diagnosis, pathophysiology, target-organ involvement, treatment, and challenges for future research. J Bone Miner Res. 2011;26(10):2317–37.
8. Delitala AP, Scuteri A, Doria C. Thyroid hormone diseases and osteoporosis. J Clin Med. 2020;9(4):1034.
9. Sandy XL, Rekha K, Meera V. Role of reduced thyroid hormone status on bone mineral metabolism. MedPulse Int J Biochem. 2021;19(2):26–9.
10. Pasieka JL, Wentworth K, Yeo CT, Cremers S, Dempster D, Fukumoto S, et al. Etiology and pathophysiology of hypoparathyroidism: a narrative review. J Bone Miner Res. 2022;37(12):2586–601.
11. Schafer AL, Shoback DM. Hypocalcemia: diagnosis and treatment. In: Feingold KR, Anawalt B, Blackman MR, et al., editors. Endotext. South Dartmouth (MA): MDText.com; 2016.
12. Koulouri O, Gurnell M. How to interpret thyroid function tests. Clin Med (Lond). 2013;13(3):282–6.
13. Yoo WS, Chung HK. Subclinical hypothyroidism: prevalence, health impact, and treatment landscape. Endocrinol Metab (Seoul). 2021;36(3):500–13.
14. Athokpham D, Mohanty S, Pinnelli VB. Status of serum calcium, phosphorus, magnesium, and copper in hypothyroid patients: a case-control study. J Clin Diagn Res. 2020;14(10):BC05–8.
15. Srinivas M, Krishna BM, Rao MM. A study of serum calcium, magnesium and phosphorous

levels in patients undergoing hypothyroidism. MedPulse Int J Med. 2021;19(2):64–7.
16. Sridevi D, Dambal AA, Sidrah, Challa AS, Padaki SK. A study of serum magnesium, calcium and phosphorus in hypothyroidism. Int J Clin Biochem Res. 2016;3(2):236–9.
17. Saxena S, Srivastava PC, Katyal R, Das B, Pannu SJ. Evaluation of mineral status in hypothyroidism in a tertiary care centre. Int J Res Med Sci. 2020;8(2):667–71.
18. Jat RK, Panwar AK, Agarwal P, Sharma C, Bansal DP, Pareek A, et al. Assessment of serum minerals in subclinical hypothyroid and overt hypothyroid patients. Cureus. 2021;13 (8):e16944.
19. Charak R, Charak R, Nigoskar S, Kumar A. Evaluation of serum electrolytes, calcium and phosphorous in patients with thyroid dysfunction. Eur J Cardiovasc Med. 2024;14 (2): x355–61.