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ASSOCIATION OF PRIMARY HYPERPARATHYROIDISM AND PAPILLARY THYROID CARCINOMA IN A PATIENT WITH BROWN TUMOR AND PARKINSONISM: CASE REPORT

Maghakyan S.A.[1*], Aghajanova E.M.[2], Khachaturyan S.R.[1], Hrantyan A.M.[3], Melkonyan N.R.[3], Aleksanyan A.Y.[4], Barseghyan E.S.[5], Muradyan A.A.[5]

[1] Department of General Therapy, Mikaelyan Institute of Surgery, Yerevan, Armenia
[2] Department of Endocrinology, Yerevan State Medical University, Yerevan, Armenia
[3] Department of Urology, Mikaelyan Institute of Surgery, Yerevan, Armenia
[4] Department of General Surgery, Mikaelyan Institute of Surgery, Yerevan, Armenia
[5] Administration Mikaelyan Institute of Surgery, Yerevan, Armenia

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Abstract

Primary hyperparathyroidism is an endocrine disorder characterized by primary abnormality of parathyroid tissue leads to inappropriate secretion of parathyroid hormone. The inappropriately high serum concentration of parathyroid hormone in primary hyperparathyroidism, in turn, sustains excessive renal calcium reabsorption, phosphaturia, and calcitriol synthesis, as well as increased bone resorption. These actions of parathyroid hormone produce the characteristic biochemical phenotype of hypercalcemia and hypophosphatemia, loss of cortical bone, hypercalciuria, and the various clinical sequelae of chronic hypercalcemia, including renal, gastrointestinal, neuromuscular, psychological and skeletal.

We would like to present a 64-year-old female case, who had almost all manifestations of primary hyperparathyroidism, also those, which is not common like brown tumor and parkinsonism.

Brown tumor is considered a rare skeletal manifestation of hyperparathyroidism. It is a non-neoplastic, reactive bone lesion resulting from parathyroid hormone excessive secretion. The incidence of brown tumor is reported to be approximately 3-4.5% in primary hyperparathyroidism in the literature.

Hyperparathyroidism associated with hypercalcemia can worsen underlying parkinsonism. The association between hypoparathyroidism and parkinsonism have been frequently reported in the literature, while evidence of hyperparathyroidism associated with parkinsonism is rare, and further studies in this regard could be revolutionary.

In this case, there are also coexistence of primary hyperparathyroidism and papillary thyroid carcinoma. Primary hyperparathyroidism is associated with a modestly increased risk of developing various cancers, including thyroid cancer, breast cancer, and potentially other malignancies. So, maybe other studies need to discover this link and suggest more carefully examine this patient in terms of cancer risk, especially thyroid cancer.

Surgical intervention was chosen as the definitive treatment. Following surgery, the patient's overall condition, including her neurological symptoms, improved significantly.

Keywords: *primary hyperparathyroidism, calcium, brown tumor, thyroid cancer, parkinsonism*

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Address for Correspondence:

Sona A. Maghakyan, MD
Mikaelyan Institute of Surgery,
9 Ezras Hasratyan street, Yerevan, 0052, Armenia
Tel.: (+374 96) 95-09-95
E-mail: son.maghakian96@mail.ru

Introduction

Hyperparathyroidism is a disease characterized by excessive secretion of parathyroid hormone and usually subdivided into primary, secondary, and tertiary hyperparathyroidism.

Primary hyperparathyroidism, a common endocrine disorder characterized by hypercalcemia and elevated or inappropriately normal levels of parathyroid hormone (PTH), is diagnosed based upon biochemical evaluation [*Walker M, Silverberg S, 2018*].

Secondary hyperparathyroidism is the overproduction of parathyroid hormone secondary to hypocalcemia, typically as a result of vitamin D deficiency and/or chronic kidney disease.

Tertiary hyperparathyroidism is a state of excessive secretion of parathyroid hormone after longstanding secondary hyperparathyroidism, and results in hypercalcemia. Some authorities reserve the term for secondary hyperparathyroidism that persists after successful renal transplantation.

The main effects of PTH are to increase the concentration of plasma calcium by increasing the release of calcium and phosphate from bone matrix, increasing calcium reabsorption by the kidney, and increasing renal production of calcitriol, which increases intestinal absorption of calcium.

Most patients with primary hyperparathyroidism have single adenomatous (80%) or multiply hyperplastic (15% to 20%) parathyroid tissue. Multiglandular disease can also be manifest as two and, very rarely, three adenomas. Parathyroid carcinoma is rare, accounting for <1% of all cases of primary hyperparathyroidism [*Bilezikian J et al., 2016*].

Primary hyperparathyroidism manifests with symptoms from many organs and systems, mainly kidney stones or osteoporotic fractures. Therefore, patients with primary hyperparathyroidism are usually first diagnosed by non-endocrinologists (including urologists, nephrologists, orthopedists, osteoporosis specialists and rheumatologists), who should know its symptoms, diagnostic criteria, and current evaluation.

The clinical picture of primary hyperparathyroidism depends on disease severity and chronicity. The classical manifestations of primary hyperparathyroidism are bone pain, proximal myopathy, osteoporotic fractures, bone cysts, brown tumors, nephrolithiasis and/or nephrocalcinosis. It has been clearly established that primary hyperparathyroidism causes skeletal and renal manifestations and that they are at least partially reversed with parathyroidectomy [*Bilezikian J et al., 2022*]. There are also many nonclassical symptoms, which may occur in patients with mild, moderate and severe hypercalcemia. They include gastrointestinal, cardiovascular, neurobehavioral and neurocognitive features [Marques O, Moreira C, 2020]. Some of these symptoms are the consequence of hypercalcemia (e.g. polyuria, polydipsia, dehydration, nausea, vomiting, constipation, anorexia, headache, altered cognitive function and mental status, acute kidney injury or nephrocalcinosis), while others are exclusively caused directly by PTH (skeletal manifestations) [*Hannan F et al., 2019, Goltzman D, 2018*].

Brown tumors, an exceptional bone complication of severe primary or secondary hyperparathyroidism, are caused by long-standing, elevated PTH-induced osteoclast activation causing multinucleated giant cell conglomerates with hemosiderin deposits in addition to the local production of cytokines and growth factors [*Carsote M et al.*, 2024]. The brown tumor is also referred to as "osteitis fibrosa cystica" [*Hong W et al.*, 2011]. The incidence of brown tumor is reported to be approximately 3-4.5% in primary hyperparathyroidism in the literature [*Karaca M et al.,* 2024]. Treatment of brown tumors frequently focuses on managing the underlying primary hyperparathyroidism, which can often lead to regression and resolution of the lesion, without the need for surgical intervention. However, in refractory cases or when dealing with large symptomatic lesions, surgical treatment may be necessary [*Tengg M et al., 2024*].

One of the nonclassical symptom of primary hyperparathyroidism is neuropsychiatric dysfunction.

Parkinson's disease is a degenerative disorder that affects the neuromotor system and is characterized by extrapyramidal symptoms including bradykinesia, rigidity, tremor, postural instability, and the freezing phenomenon. The loss of dopamine-secreting neurons in the substantia nigra induces a relative deficiency of dopamine and imbalance of dopamine and other various neurotransmitters including acetylcholine, glutamate, and GABA in the basal ganglia.

Secondary parkinsonism is a disease state in which actions of dopamine in the basal ganglia is

blocked or interfered by drugs, toxicants, cerebrovascular diseases, metabolic disorders and various comorbidities [*Ohya Y et al., 2018*].

Drug-induced parkinsonism, tardive dyskinesia, tardive dystonia, akathisia, myoclonus, and tremor are drug-induced motility disorders. Among these adverse drug reactions, drug-induced parkinsonism is the most widespread movement disorder caused by drugs that antagonise dopamine receptors, after Parkinson's disease, which has similar clinical manifestations [*Sethi K, 2001*]. One of the causes of antipsychotic induced parkinsonism is a disruption of neurotransmitter interactions that regulate the signaling pathways of the dopaminergic, cholinergic, GABA-ergic, adenosinergic, endocannabinoid and other neurotransmitter systems.

The monoamine (dopamine, serotonin, and norepinephrine) systems of the brain play an important role in normal behavior, and disturbances in these circuits are thought to be involved in the development of a number of neurological and psychiatric disorders. The dopamine system is involved in the implementation of such brain functions as locomotion, affect and cognition [*Lauder J, Bloom F, 1974*]. The association between hypoparathyroidism and parkinsonism have been frequently reported in the literature, while evidence of hyperparathyroidism associated with parkinsonism is rare.

Decreased or increased levels of serum calcium can lead to metabolic disorders known as hypocalcemia or hypercalcemia, respectively [*Augusto C et al., 2019*]. It is known that calcium is necessary for neuromuscular stimulation and also in neurotransmission [*Emad E et al., 2022*]. Therefore, altered calcium levels affect the transmission of nerve impulses, which could lead to partial or total damage of dopaminergic receptors [*Augusto C et al., 2019*]. Hyperparathyroidism associated with hypercalcemia can worsen underlying parkinsonism. The association of hyperparathyroidism with parkinsonism is rare [*Parks K et al., 2017*]. Hypercalcaemia can lead to many neuropsychiatric symptoms from fatigue, lethargy, anxiety, irritability, and insomnia to impaired concentration and memory, depression, delirium, and psychosis.

Neuropsychiatric disorders, often referred to as the "moans" of primary hyperparathyroidism, have long been acknowledged alongside "bones" (osteoporosis and decreased bone mineral density) and "stones" (kidney stones) as hallmark features of primary hyperparathyroidism. Among patients undergoing parathyroidectomy, the most prevalent neuropsychiatric symptoms include depression (33%–62.1%), anxiety (43.1%–53%), anger and irritability (51.9%), cognitive impairment (37.3%–46.5%), suicidal ideation (22%), and hallucinations and delusions (5%–20%) [*Serdenes R et al., 2021*].

Hypercalcemia, stemming from primary hyperparathyroidism, is thought to bidirectionally influence monoamine neurotransmission, thereby leading to either manic or depressive manifestations [*Seib C et al., 2021*]. Parathyroidectomy was associated with a reduced incidence of cognitive impairment, somnolence, and schizophrenia [*Song Z et al., 2024*].

Primary hyperparathyroidism is associated with several cancer types, including papillary thyroid carcinoma. Larger population studies are required to determine if hyperparathyroidism is a significant risk factor for papillary thyroid carcinoma. At this time, we suggest that patients with primary hyperparathyroidism should be regarded as extra-suspicious for papillary thyroid carcinoma. Evaluation of thyroid nodules by preoperative ultrasound may be advisable in patients with primary hyperparathyroidism, particularly for females and patients with modestly elevated serum parathyroid hormone levels [*Cinamon U et al., 2015, Luchuan L et al., 2021*].

The classical surgical management of primary hyperparathyroidism consists of exploration and identification of all four parathyroid glands through a transverse low cervical incision. This approach allows effective treatment of not only single gland but also the detection of multiple adenomas and diffuse parathyroid hyperplasia involving all four glands. The localized approach focusing on a single parathyroid gland was developed in order to reduce the operative morbidity associated with multi-gland exploration (hematoma, recurrent laryngeal nerve palsy), to decrease the duration of surgery and hospitalization, and to minimize the cosmetic impact of the cervical incision. In addition, it can be performed under local anesthesia in fragile patients. Mini-invasive approach can be proposed for laboratory-proven primary hyperparathyroidism where there is no family history of endocrine disease, no associated thyroid disease, and a single well-defined parathyroid adenoma lo-

calized by at least two concordant imaging studies (ultrasonography, scintigraphy, CT scan). The effectiveness of the surgery can be confirmed by frozen section histopathologic examination of the resected specimen and by serial intraoperative measurement of serum PTH levels. A fall of at least 50% in the PTH level after removal of the parathyroid gland confirms excision of the pathologic gland [*Taieb M et al., 2013*].

MATERIALS AND METHODS

A 64-year-old female was admitted to the Urology Clinic of Mikaelyan Institute of Surgery, complaining of general weakness, pain in the right lumbar region and fever. The complaints had presented for about 10 days, and a CT scan performed. The patient was diagnosed Ureterolithiasis, right-ureteral stone obstruction, right-sided ureter hydronephrosis, pyelonephritis and was preparing for surgery. A right-sided nephrectomy and right-sided ureterolithotomy were performed.

In a past medical history, the patient had a Schizophrenia for about 25 years, and was receiving treatment, underwent a hemimaxillectomy for a giant cell tumor in the upper jaw 10 years ago. The patient also had pathological fractures.

During the initial examination, the patient exhibited reduced interaction and appeared very indifferent. Her responses were brief, with significant delays. According to her caregivers, she experienced anxiety, delusions, and hallucinations, which were attributed to her primary diagnosis of schizophrenia. The patient has been on Haloperidol 10*mg* for more than 25 years and Trihexyphenidyl 2*mg* for the past 7 years. However, she occasionally adjusts the dosage on her own.

Objective neurological examination revealed the following findings: combined rest and action tremor, which became more pronounced during speech and eating. Gait characterized by wide-based steps with shuffling. Tremor observed in the chin, accompanied by noticeable oral dyskinesias. The patient examined by a neurologist and was diagnosed Drug-induced Parkinsonism.

Blood tests results showed that Hb-76*g/l* (normal range 115-152), Creatinine-128*umol/l* (44-100), Ca ionized-2.20*mmol/l* (1.13-1.32), PTH-409.6*pg/ml* (15-65), Vitamin D-10.6*ng/mL* (30-100), TSH-3.85 *uIU/mL* (0.3-4.5).

Neck ultrasound found a perifocal, abundantly vascularized tissue mass with macrocalcifications in the left parathyroid gland, measuring 1.6*1.2 cm, with wavy and well-defined borders. It also found multiple thyroid nodules (TI-RADS 4), but the patient refused to do the fine needle biopsy.

The results of dual-energy X-ray absorption assay found the T-score at L1-L4 was -5.1, and T-score at the femoral neck was -5.4. Wrist X-ray found severe osteoporosis of the wrist bones.

Full-Body CT Scan (Fig. 1a, b, c) showed multiple lytic lesions involving bone probable Brown tumor, chronic cholecystitis, fibrous formations in the uterine fundus and body.

Esophagogastroduodenoscopy found picture of atrophic gastritis.

The patient started treatment with Vitamin D_3, rehydration and correction of anemia. After 1 months, she underwent to left parathyroidectomy and total thyroidectomy by classical approach.

The histology showed parathyroid adenoma and papillary microcarcinoma of the left lobe of the thyroid gland. PTH level decreased after surgery immediately (from 551.2 to 69.98).

The patient's neurological condition also improved after surgery. She had a mood lift, positive changes in speech and gait. After surgery the pa-

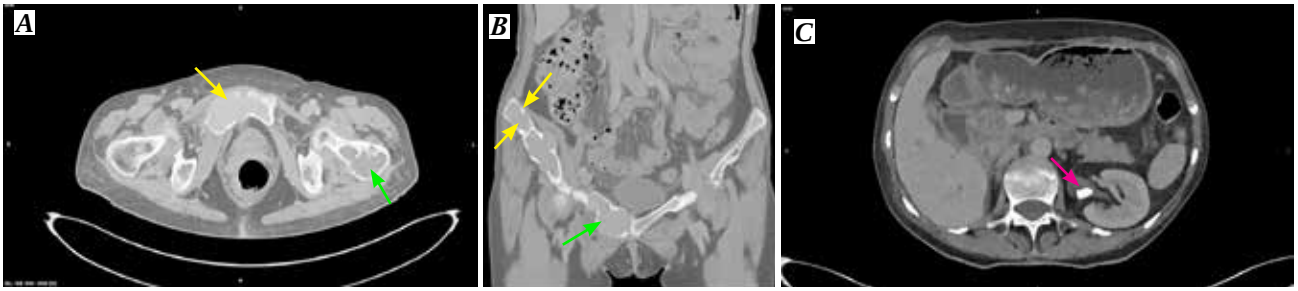

FIGURE 1. *Lytic lesion of the upper ramus of the right iliac bone (yellow arrow) and the left proximal femur (green arrow) (A), the sternal wings on both sides (yellow arrow) and the upper ramus of the right iliac bone (green arrow) (B) and stones in the left kidney (arrow) (C).*

tient was given Vitamin D_3 supplementation, Calcium gluconate and Levothyroxine. The patient is currently under the supervision of an endocrinologist, oncologist and neurologist.

Discussion

This patient had long disease duration and many manifestations of hypercalcemia. Approximately 90% of cases of hypercalcemia are caused by hyperparathyroidism or malignancy [*Walker M, Shane E, 2022*]. In our case we had both.

In Primary hyperparathyroidism patients, several studies reported the prevalence of papillary thyroid cancer to range from 2% to 15% [*Jeong C et al., 2020*].

Except, common manifestations of hypercalcemia and Primary hyperparathyroidism, the patient had also these rare manifestations. Brown tumor is considered a rare skeletal manifestation of hyperparathyroidism. It is a non-neoplastic, reactive bone lesion resulting from excessive PTH secretion. Histologically, brown tumors are characterized by masses of giant cells surrounding the fibrovascular stroma, cystic spaces in connective tissue, foci of hemorrhage associated with microfracture, and subsequent release of hemosiderin, which gives them their name.

Hypercalcemia is known to cause neuropsychiatric dysfunction too. In the literature, the association between hypoparathyroidism and parkinsonism have been frequently reported, while evidence of hyperparathyroidism associated with parkinsonism is rare. In our patient parathyroidectomy provided a significant remission of parkinsonism. Therefore, in this case, Parkinsonism may not only be drug-induced, but also hypercalcemia-induced.

Conclusion

It is known that a lot of endocrine diseases have neurological and psychiatric manifestations and often they appear at first. It is interesting that our patient at first had psychiatric complaints and was referred to psychiatrist. The diagnosis was made Schizophrenia and treatment was started. Then was appeared bone manifestations and the patient was operated. The last manifestation for the hospitalization was ureterolithiasis, when during hospitalization diagnosis primary hyperparathyroidism was made.

So, the patient had long and complicated anamnesis of disease. Now it is difficult to know Schizophrenia was one of the manifestations of primary hyperparathyroidism or concomitant disease.

Therefore, it is very important to deny underling endocrine diseases before making some psychiatric or neurological diagnosis.

REFERENCES

1. *Augusto CMG, de Morais NS, Santana RP, de Almeida MOP. (2019).* Parkinsonism as an atypical manifestation of primary hyperparathyroidism AACE Clin Case Rep. 2019;5: e244–6
2. *Bilezikian, J., Cusano, N., Khan, A. et al.* (2016). Primary hyperparathyroidism. Nat Rev Dis Primers 2, 16033 (2016). https://doi.org/10.1038/nrdp.2016.33
3. *Carsote M, Ciobica ML, Sima OC, Valea A, Bondor CI, Geleriu A, et al., (2024).* Brown Tumors: The Hidden Face of Primary and Renal Hyperparathyroidism Amid Real-Life Settings. J Clin Med. 2024 Jun 29;13(13):3847. doi: 10.3390/jcm13133847. PMID: 38999413; PMCID: PMC11242279.
4. *Cinamon U, Levy D, Marom T. (2015).* Is primary hyperparathyroidism a risk factor for papillary thyroid cancer? An exemplar study and literature review. Int Arch Otorhinolaryngol. 2015 Jan;19(1):42-5. doi: 10.1055/s-0034-1396520. Epub 2014 Dec 8. PMID: 25992150; PMCID: PMC4392524.
5. *Emad, E.M., Elmotaym, A.S.E., Ghonemy, M.A. et al. (2022).* The effect of hypocalcemia on motor symptoms of Parkinson's disease. Egypt J Neurol Psychiatry Neurosurg 58, 76 (2022). https://doi.org/10.1186/s41983-022-00499-1
6. *Goltzman D. (2018).* Physiology of Parathyroid Hormone. Endocrinol Metab Clin North Am 2018; 47: 743–758, https://doi.org/10.1016/j.ecl.2018.07.003
7. *Hannan F.M., Kallay, E.,Chang, W. et al. (2019).* The calcium-sensing receptor in physiology and in calcitropic and noncalcitropic diseases. Nat Rev Endocrinol 15, 33–51(2019). https://doi.org/10.1038/s41574-018-0115-0
8. *Hong W.S., Sung, M.S., Chun, KA. et al.*

(2011). Emphasis on the MR imaging findings of brown tumor: a report of five cases. Skeletal Radiol 40, 205–213 (2011). https://doi.org/10.1007/s00256-010-0979-0

9. *Jeong C, Kwon HI, Baek H, Kim HS, et al., (2020)*. Association of Hyperparathyroidism and Papillary Thyroid Cancer: A Multicenter Retrospective Study. Endocrinol Metab (Seoul). 2020 Dec;35(4):925-932. doi: 10.3803/EnM.2020.725. Epub 2020 Dec 10. PMID: 33297604; PMCID: PMC7803618.
10. *Bilezikian JP, Khan AA, Silverberg SJ, Fuleihan GEH, Marcocci C, Minisola S et al., (2022)*. On behalf of the International Workshop on Primary Hyperparathyroidism, Evaluation and Management of Primary Hyperparathyroidism: Summary Statement and Guidelines from the Fifth International Workshop, Journal of Bone and Mineral Research, Volume 37, Issue 11, 1, 2022, Pages 2293–2314, https://doi.org/10.1002/jbmr.4677
11. *Karaca, M.O., Özyıldıran, M., Savran, M.D. et al. (2024)*. Brown tumors: Retrospective analysis of 26 cases. Arch Orthop Trauma Surg 144, 2927–2934 (2024). https://doi.org/10.1007/s00402-024-05372-9
12. *Lauder, J.M.; Bloom, F.E. (1974)*. Ontogeny of monoamine neurons in the locus coeruleus, Raphe nuclei and substantia nigra of the rat. I. Cell differentiation. J. Comp. Neurol. 1974, 155, 469–481.
13. *Li, L, Li B, LvB, Liang W, Zhang B, Zeng O, Andrew G Turner, and Sheng L (2024)*.. "Increased thyroid malignancy in patients with primary hyperparathyroidism", Endocrine Connections 10, 8 (2021): 885-893, accessed Dec 23, 2024, https://doi.org/10.1530/EC-21-0217
14. *Majic Tengg A, Cigrovski Berkovic M, Zajc I, Salaric I, Müller D, Markota I. (2024)*. Expect the unexpected: Brown tumor of the mandible as the first manifestation of primary hyperparathyroidism. World J Clin Cases 2024; 12(7): 1200-1204 [PMID: 38524508 DOI: 10.12998/wjcc.v12.i7.1200]
15. *Oberger Marques JV, Moreira CA. (2020)*. Primary hyperparathyroidism. Best Pract Res Clin Rheumatol 2020; 34: 101514, https://doi.org/10.1016/j.berh.2020.101514.
16. *Ohya Y, Osaki M, Sakai S, Kimura S, Yasuda C, Ago T, et al., (2018)*. A case of hyperparathyroidism-associated Parkinsonism successfully treated with cinacalcet hydrochloride, a calcimimetic BMC Neurol. 2018;18:62.
17. *Parks, K. A., Parks C. G., Onwuameze O. E., and Shrestha S.. (2017)*. "Psychiatric Complications of Primary Hyperparathyroidism and Mild Hypercalcemia." American Journal of Psychiatry 174(7): 620–622.https://doi.org/10.1176/appi.ajp.2017.16111226
18. *Seib, C. D., MenT. g, Suh I., A. HarrisH. S. , Covinsky K. E., Shoback D. M., et al. (2022)*. "Risk of Fracture Among Older Adults with Primary Hyperparathyroidism Receiving Parathyroidectomy vs Nonoperative Management." JAMA Internal Medicine 182(1): 10–18. https://doi.org/10.1001/jamainternmed.2022.
19. *Serdenes R., M. Lewis, and S. Chandrasekhara. (2021)*. "A Clinical Review of the Psychiatric Sequelae of Primary Hyperparathyroidism." Cureus 13(10): e19078.https://doi.org/10.7759/cureus.19078.
20. *Sethi K.D. (2001)*. Movement disorders induced by dopamine blocking agents. Semin. Neurol. 2001, 21, 59–68.
21. *Song Z, Balachandra S, Wu C, Wang R, Zmijewski P, Gillis A, et al., (2024)*. Risk of neuropsychiatric disorders in primary hyperparathyroidism: Parathyroidectomy versus nonoperative management. World J Surg. 2024 Jul 14. doi: 10.1002/wjs.12285. Epub ahead of print. PMID: 39004613.
22. *Taieb, M. Seman, F. Menegaux, C. Trésallet, (2013)*. Surgical technique parathyroidectomy through a minimally invasive gland-centered localized approach for primary hyperparathyroidism, Journal of Visceral Surgery, Volume 150, Issue 6, 2013, Pages 403-406, ISSN 1878-7886, https://doi.org/10.1016/j.jviscsurg.2013.09.013.(https://www.sciencedirect.com/science/article/pii/S1878788613001306)
23. *Walker MD, Shane E. (2022)*. Hypercalcemia: A Review. JAMA. 2022 Oct 25;328(16):1624-1636. doi: 10.1001/jama.2022.18331. PMID: 36282253.
24. *Walker, M., Silverberg, S. (2018*) Primary hyperparathyroidism. Nat Rev Endocrinol 14, 115–125 (2018). https://doi.org/10.1038/nrendo.2017.104

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Address for correspondence:

Yerevan State Medical University
2 Koryun Street, Yerevan 0025,
Republic of Armenia

Phones:

(+37410) 582532 YSMU
(+37493 588697 Editor-in-Chief
Fax: (+37410) 582532
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