Water Clear Cell Parathyroid Adenoma: A Report of Two Cases

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Abstract

Primary hyperparathyroidism is a disease characterized by hypercalcemia and excessive production of parathyroid hormone. It is the most common cause of hypercalcemia in outpatient practice and the third common endocrine disorder. The incidence of primary hyperparathyroidism is increasing with age, and it is more common in women than in men. 80-85% of cases of primary hyperparathyroidism are due to single parathyroid adenoma. The majority of parathyroid adenomas contain a mixed cell population with predominance of chief cells. Primary hyperparathyroidism with clear cell adenoma is very rare.

Here we are sharing two cases of water clear cell parathyroid adenoma associated with primary hyperparathyroidism presented with deferent clinical manifestations.

Keywords: primary hyperparathyroidism, water clear cell, parathyroid adenoma, osteoporosis, pancreatitis, hypertension diabetes mellitus, thalassemia.

Running title: Clear cell parathyroid adenoma associated with primary hyperparathyroidism, report of two cases.

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Introduction

Primary hyperparathyroidism (PHPT) is a disorder that mainly originates from the parathyroid glands itself, wherein one or more of the glands become hyperactive which results to over secretion of the parathyroid hormone (PTH). It is the third most common endocrine disorder after Diabetes Mellitus (DM) and thyroid disease, with a prevalence rate of 0.1 - 1% (Madkhali et al., 2016). It is characterized ideally as hypercalcemia with elevated serum PTH concentration; it can now be recognized extensively ranging from inappropriately high or even normal PTH in the setting of high-normal or normal calcium (Madkhali et al., 2016) .

PHPT has an incidence of 28 cases per 100,000 individuals in general population and is the highest between 50 and 60 years of age, affecting 2% of the population

aged 55 years or older. It affects females 2 to 3 times more than males (Bilezikian et al., 2016; Madkhali et al., 2016). The vast majority of cases, 90- 95%, are sporadic, in which the PHPT is attributable to a solitary parathyroid adenoma in about 80% to 85% of the cases. A double adenoma is seen in up to 4% of cases and four-gland hyperplasia makes up the remaining 10-15% of cases. While, parathyroid carcinoma is a very rare cause of PHPT, accounting for less than 1% of cases, familial parathyroid disorders are responsible for approximately 5% of the PHPT cases (Bilezikian et al., 2016; Madkhali et al., 2016).

Although, adenomas usually arise as a single lesion, the majority of functional adenomas contain a mixed cell population with predominance of chief cells. There are also existing cases where clear cells and oxyphilic cells are mixed and rare cases where the majority of cells constituting the adenoma might be oxyphilic cells (Parelkar et al., 2012).

Even though, hyperparathyroidism with clear cell adenoma is very rare (El Hussein & Poppiti, 2017; Liang et al., 2010; Parelkar et al., 2012; Yazar et al., 2017), to date, around 40 cases have been reported in the literature with only four of them being intra-thyroid (Goodman et al., 2011; Gulati et al., 2012; Muthukrishnan et al., 2007; Parelkar et al., 2012).

Water clear cell parathyroid adenoma (WCCA) is a rare cause of PHPT. Water-clear cells are not seen in normal human parathyroid glands (Ioannis et al., 2018). Usually, they are associated with hyperfunction of the parathyroid gland. They are characterized histologically based on large clear cells that might be transformed from parathyroid chief cells (ARık et al., 2017). On histopathological examinations; the cytoplasm is foamy and contains vacuoles of 0.2-2 microns in diameter. These cells represent end stage of hyperplastic chief cells (El Hussein & Poppiti, 2017).

Here we present two cases of PHPT. The first case is a water clear cell intra-thyroid giant atypical parathyroid adenoma, and the second one is a small negative scan water clear cell parathyroid adenoma. Informed consent and approval from institutional review board were obtained for publication.

Case 1:

A fifty-year-old female patient, known to have β-thalassemia trait (minor thalassemia) and long-standing type 2 DM, treated with premixed insulin, Mixtard30, 60 units in the morning and 60 units at night; presented for reevaluation on July 2016 because of uncontrolled blood sugar. One month before presentation, she experienced acute pancreatitis and was conservatively treated in a provincial hospital. No available data or medical reports were provided.

Reviewing the history of the patient, she had a long-standing insulin treated type2 DM, hyperuricemia, diabetic peripheral neuropathy and chronic anemia. Additionally, she was diagnosed with β-thalassemia a year before presentation. Furthermore, a previous history of nonspecific abdominal pain and epigastric tenderness since 2012 that was associated with alternating diarrhea and constipation were reported. Patient was examined in a gastroenterology (GI) clinic without a definite diagnosis. Although panendoscopy was suggested but it was not performed at that time.

On presentation, the patient complained of tiredness, easy fatigability, all over muscle and joints pain, numbness in both hands and lower limbs as well. On examination, the patient is overweight with a BMI of 40. Vital signs showed high systolic blood pressure 196/76, tachycardia, pulse was regular and high random blood sugar (319 mg/dL). Palpation of the neck revealed a right thyroid lobe enlargement. Chest auscultation: vesicular breathing

and normal heart sounds. Abdomen was soft, lax, with non-specific tenderness and no organomegaly. Lower limbs: pulses are present and no edema was detected, however a decreased vibration sensation was noted.

Laboratory investigations showed uncontrolled blood sugar with high HbA1c, mixed dyslipidemia, low vitamin D levels, hyperuricemia and high serum calcium. Once severe vitamin D deficiency and high serum calcium with right thyroid lobe enlargement were observed, further laboratory and radiological investigations were ordered. These investigations revealed and confirmed a picture of hyperparathyroidism with severe vitamin D deficiency (Table 1).

Table 1: Laboratory Investigations Pre and Post-Surgery for case 1

	Pre-Surgery	Post-Surgery	Normal range
Fasting Blood Sugar	327	130	80-100 mg/dl
HbA1c	11.41	7.5	4.6-5.7%
Calcium	12.07	10	8.5-10.5 mg/dl
TSH	2.3	2.4	0.4-4.0 mU/l
Vitamin D	17	40	50-150 nmol/L
ALP	135	149	35-147 IU/L
PTH	45.5	6.6	1.0-6.5 pmol/L

Additionally, neck ultrasound (U/S) revealed right thyroid lobe enlargement measuring 3.3x3.5x5 cm and a large cystic solid nodule was seen in the right lobe measuring 4.2x3.2x2.8 cm, showing surrounding vascularity and mild internal vascularity. The left lobe measured 1.2x1.7x3 cm and appeared normal in size, shape and texture. Multiple reactive lymph nodes enlargement in the left parotid and submandibular region were observed (Figure 1).

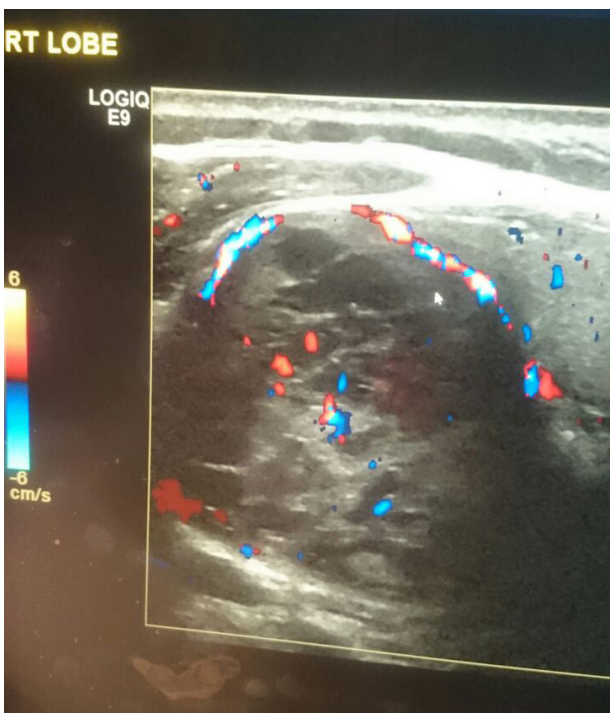

Figure 1: Neck ultrasound showing right thyroid lobe enlargement measuring 3.3x3.5x5 cm and a large cystic solid nodule in the right lobe.

Abdominal U/S showed fatty liver, left renal simple cyst, tiny focus of cholesterolosis in the gall bladder and the visualized part of the pancreas appeared normal, but the tail of pancreas was not clearly visualized due to excessive bowel feces. Skeletal bone survey showed an overall decreased bone density, mild subperiosteal tunneling and absorption was seen in the proximal phalanges of both hands (Figure 3), small foci of lucency were observed along the skull bones, giving salt and pepper appearance (Figure 2).

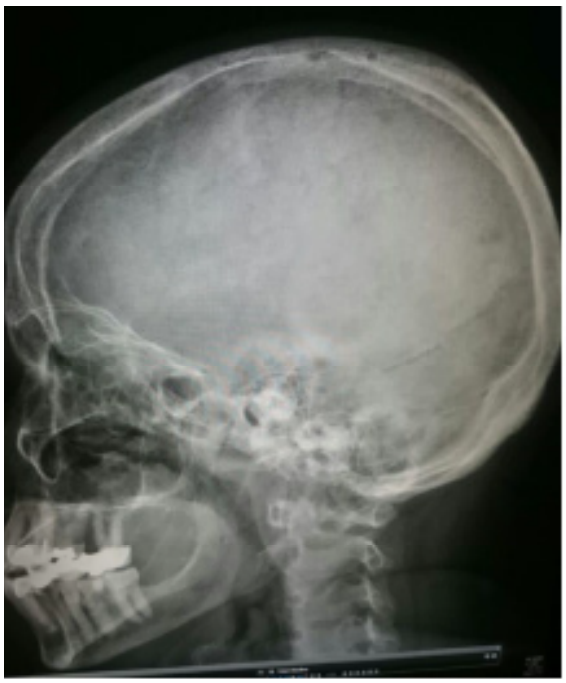

Figure 2: Small foci of luceny seen along the skull bones giving salt and pepper appearance

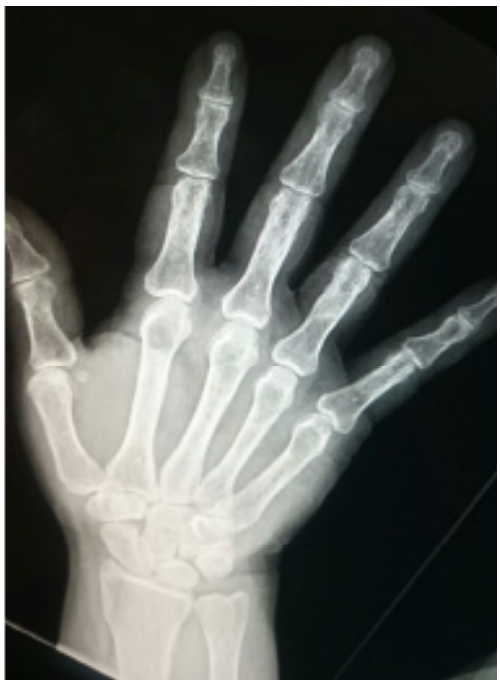

Figure 3: Mild subperiosteal tunneling and absorption seen in the proximal phalanges

Reviewing previous 2012 X-rays, bones appeared grossly osteopenic with vertebral endplate sclerosis. Vascular calcifications were observed and both sacroiliac joints were slightly sclerosed. Total bone mineral density (BMD) was 0.759/cm2 which is consistent with T-score of -3.2 and Z-score of -4.6 (Table 2).

Table 2: Bone Mineral Density for case 1

	T-score	Z-score
L1	-3.1	-4.2
Right femur	-2.8	-3.5
Left femur	-2.6	-3.3
Total body	-3.2	-4.6

Parathyroid imaging with technetium-99m Sestamibi showed a large tracer-avid right thyroid nodule that could be an aggressive thyroid nodule or a large hyperfunctioning intra-thyroid parathyroid adenoma (Figure 4). Fine needle aspiration (FNA) revealed focally cellular foci where cohesive sheets that were formed by small and uniform cells, with rounded homogenous nuclei. Rare granulomas and focal Hurtle cell changes were noted in these samples and the pathology report was a follicular lesion (Figure 5).

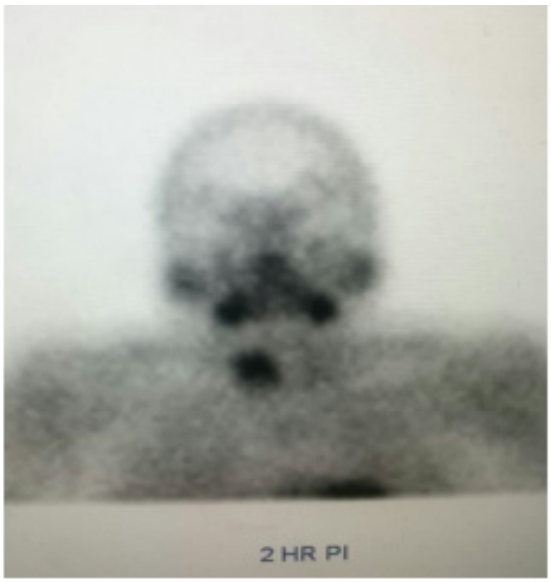

Figure 4: Parathyroid technetium 99 Sestamibi scan showing large tracer avid right thyroid nodule that could represent an aggressive thyroid nodule or a large hyperfunctioning intra-thyroid parathyroid adenoma

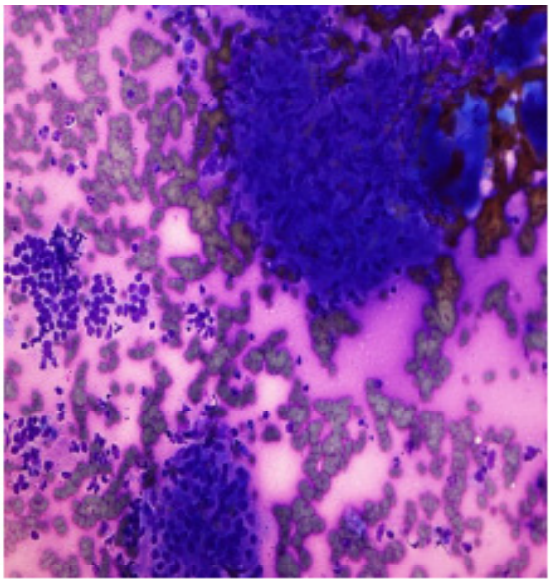

Figure 5: Fine need aspiration of patient number 1 neck mass showing cellular clusters (white arrow) of small uniform cells, with rounded homogenous nuclei. DQx200arrows

The patient underwent hemithyroidectomy, parathyroidectomy and hospital course was smooth without any complications. Even though, parathyroid hormone (PTH) dropped from 51 to 0.8 pmol/L following surgery, she did not develop hypocalcemia, which is frequently observed after a parathyroidectomy due to hungry bone syndrome. Laboratory investigations before and after surgery are presented in (Table1).

Gross description of hemithyroidectomy and parathyroidectomy specimens (Figure 6) revealed parathyroid adenoma of 5 cm in maximum dimension and weighing 36.3g. Microscopic examination revealed predominantly water cell pattern with atypical features and rim of benign thyroid tissue. The atypical features included fibrotic areas and fibrous septae formation and clusters of tumor cells in the fibrous capsule (Figure 7). During follow up visits postoperatively; the patient is much better clinically with no significant complaints and good control of blood sugar, her most recent HbA1c was 7.5% (Table 1).

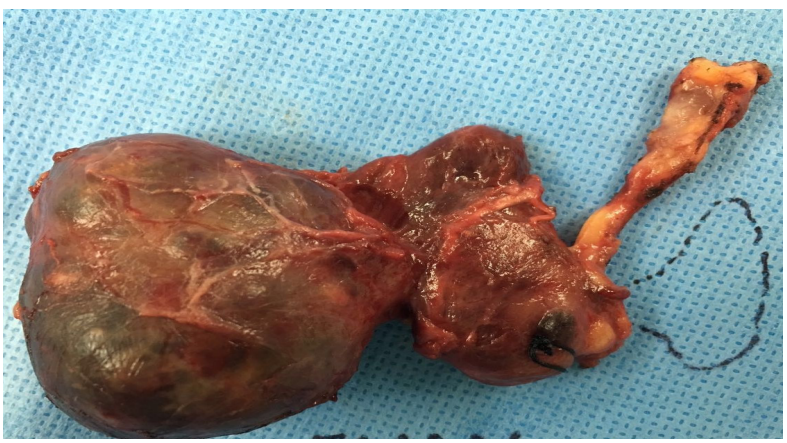

Figure 6: Hemi-thyroidectomy-parathyroidectomy specimen.

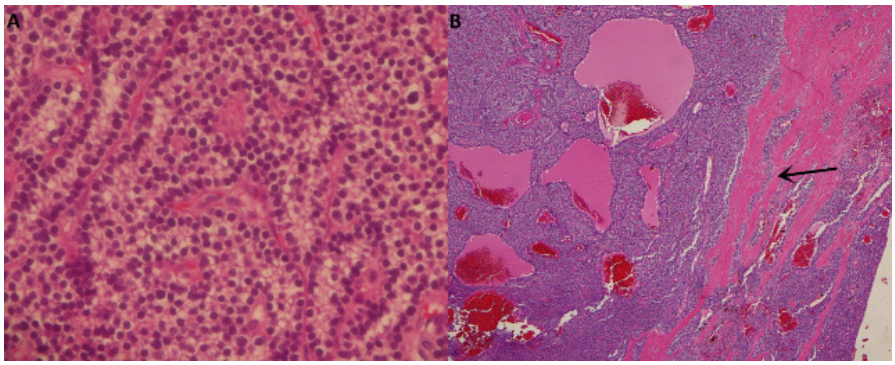

Figure 7: A. High power view showing trabeculae of cellular parathyroid tissue with clear white cytoplasm and round monotonous nuclei (H&E, X 400). B. Low power microscopic view of the mass revealing trabeculae and sheets of cellular parathyroid tissue with microfollicles formation and fibrous septae in the stroma is seen (black arrow).

Case 2:

Thirty-three-year-old female patient, presented to endocrine clinic for reevaluation in 2015. She complained of tiredness, easy fatigability, bone and joint pain. For the last 2 years before presentation, she was labeled as a case of polycystic ovary syndrome (PCOS) and treated with metformin for short periods. She is clinically stable, asymptomatic, with normal vital signs and normal body mass index (BMI). No palpable goiter. On examination, neck, heart, abdomen and limbs were normal. Her laboratory investigations showed low vitamin D level 16 (15-150), high-normal calcium 2.56 (2.15-2.55 mmol/L), and high PTH 16.56 (1.6-6.9 pmol/L). A diagnosis of PHPT was suggested.

Neck U/S in 2014 revealed a small hypoechoic area in the left lobe of the thyroid measuring 0.9x0.6 cm with increased peripheral vascularity. The possibility of parathyroid adenoma could not be entirely excluded and scintigraphy was warranted for further work up (Figure 8).

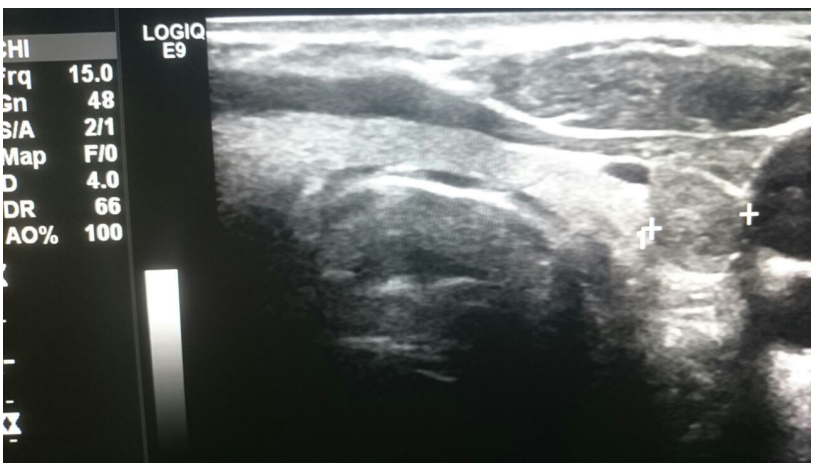

Figure 8: Neck US showing left inferior intra-thyroid oval shaped hypoechoic lesion, 1.3x0.4 cm.

Over the last 2 years, the patient continued to have high PTH, high calcium, and high alkaline phosphatase (ALP) with normal phosphorus levels (Table 3). Parathyroid adenoma was not considered at that time because BMD was normal and nuclear medicine investigations failed to detect parathyroid adenoma.

Table 3: Laboratory Investigations for case 2

	Pre-surgery	Post-surgery	Normal range
Albumin	45	44	40-49 g/l
Vitamin D	22	17	50-150 nmol/L
Calcium	10.7	10.4	8.5-10.5 mg/dl
Corrected Ca	10.5	8	8.5-10.5 mg/dl
Urine, Calcium	7.22		8.5-10.5 mg/dl
PTH	16.56	2.4	1.0-6.5 pmol/L
ALP	186	105	44-147 U/l
Phosphorus	3.63	3.66	3.4 to 4.5 mg/dl
HbA1c	5.1	4.5	4.6-5.7%
FBS	94	88	80-100 mg/dl
PTH	16.56	2.4	1.6-6.9 pmol/L
TSH		1.6	

Upon reviewing medical records, the patient had nonspecific abdominal pain that was investigated in the GI clinic. No significant abnormalities were detected and therefore, no definite diagnosis was established. Meanwhile, at that time, in 2012, when she was diagnosed with PCOS, her laboratory investigations failed to show high normal corrected calcium of 2.55 (2.2-2.60) mmol/L with severe vitamin D deficiency of 12 (50-150 nmol/L).

Repeated neck U/S reported left inferior intra-thyroid oval shaped hypoechoic lesion of 1.3x0.4 cm (Figure 8). However, a year later, when the patient was reevaluated, biochemical picture was consistent with and confirmed the diagnosis of PHPT.

Furthermore, repeated neck Computed Tomography scan (CT scan) (Figure 9) and Magnetic Resonance Imaging (MRI) were suggestive of left lower parathyroid adenoma with similar picture as was observed earlier by neck U/S. In spite of the negative Sestamibi scan, minimal neck invasive surgery was performed and 0.3 gm left lower parathyroid adenoma measuring 1.2x0.7x0.6 cm was resected. Post-operative course was uneventful.

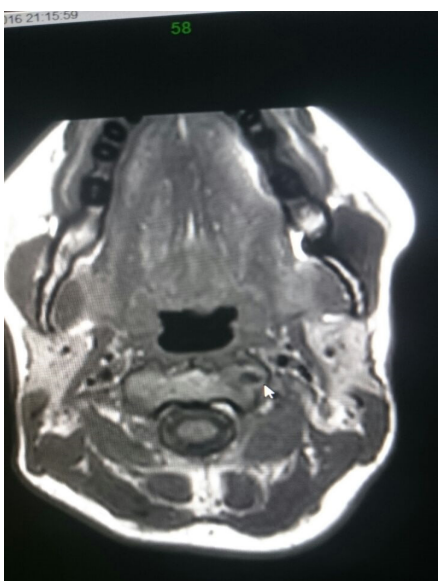

Figure 9: Neck computed tomography scan (CT scan).

Histopathological examination of the mass demonstrated water clear cell parathyroid adenoma with a rim of non-neoplastic parathyroid tissue (Figure 10). Patient did not develop hypocalcemia, her ionized calcium was normal during hospitalization and PTH dropped significantly, from 11.1 to 2.4 (1.6-6.9 pmol/L). The patient became asymptomatic with no further complaints.

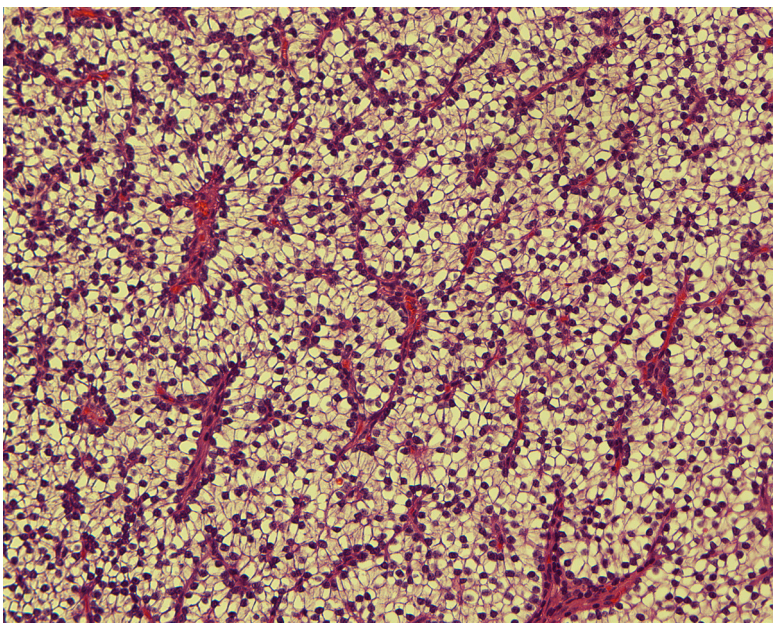

Figure 10: The mass similarly consists of trabeculae of cellular parathyroid tissue with clear white pale cytoplasm. Delicate capillary network can be seen in the background

Discussion

The diagnosis of PHPT is usually established biochemically, based on serum calcium, PTH, and phosphate levels (Bilezikian et al., 2016; Madkhali et al., 2016). While elevated levels of serum calcium and PTH are associated with 95% of classical cases of PHPT, patients however may present with normal levels of phosphate, calcium or PTH (Bilezikian et al., 2016; Madkhali et al., 2016). Twenty-four-hour urine collection for calcium measurement is therefore necessary to rule out familial hypocalciuric-hypercalcemia. Even though, 24-hour-urine collection was not performed for our patients, random calcium in urine was high in the first case. While parathyroidectomy success rate is high, persistent and recurrence still represent clinical challenge (Bilezikian et al., 2016; Madkhali et al., 2016). Therefore, pre-operative localization techniques should be considered, not only to confirm the diagnosis of PHP, but also to guide the endocrine surgeons intraoperatively, as well as to evaluate for any ectopic glands, and to assist in planning of minimally invasive parathyroidectomy (Agarwal & Pradhan, 2012; Bilezikian et al., 2016; Madkhali et al., 2016).

Usually ultrasonography, CT scan and MRI are common techniques used for parathyroid adenoma workup. Nevertheless, Sestamibi scan with single photon emission computerized tomography (SPECT) is superior due to the high sensitivity (Bilezikian et al., 2016; Madkhali et al., 2016).

Sestamibi scan was performed for both patients. In the first case, it was localized as a persistent increased area of activity in the right thyroid lobe that was seen on the neck U/S. In the second patient, ultrasonography and CT scan were suggestive of localized adenoma with negative Sestamibi scan. However, negative scan could be due to several factors including

small size of adenomas, thyroid nodules, superior position and the paucity of oxyphillic cells in the parathyroid adenoma (Bilezikian et al., 2016; Grenko et al., 1995; Kojidi et al., 2016; Madkhali et al., 2016). Water-clear cell adenoma (WCCA) is extremely rare condition, only 40 cases has been reported in the literature with variant clinical presentations (Bai et al., 2012; Kojidi et al., 2016).

While water-clear cell hyperplasia (WCCH) is another rare condition, it should be distinguished from WCCA, where in the case of WCCH, all four parathyroid glands have water clear cells on their histological examination (El Hussein & Poppiti, 2017; Ezzat et al., 2013; Liang et al., 2010; Parelkar et al., 2012; Yadav et al., 2017; Yazar et al., 2017). In fact, recent studies reported 0.3% incidence of water clear cell parathyroid disease, which supports the decline in the incidence of WCCH from 13% in 1930 to less than 1% (El Hussein & Poppiti, 2017; Ezzat et al., 2013; Parelkar et al., 2012). On the other hand another study showed the prevalence of WCCA of 46% in 66 patients with PHPT with an overt clinical picture (Varshney et al., 2013). Parathyroid clear cell carcinoma is very rare and only few cases were reported recently (El Hussein & Poppiti, 2017; Naganuma et al., 2013; Parelkar et al., 2012).

In the two cases reported here, the first one was an ectopic intra-thyroid adenoma, which is still uncommon, even though few cases have been previously reported (Abboud et al., 2007; Mondal, 2013; Parelkar et al., 2012; Prasad et al., 2004; Shi et al., 2016). Parathyroid adenoma in the first case weighted 36,5 gm which is considered a giant adenoma.Currently, fine needle aspiration (FNA) is usually performed prior to surgery for thyroid nodules as well as for suspicious parathyroid adenomas. Previously, it has been reported that FNA might be misleading and/or challenging for the diagnosis of parathyroid adenoma and therefore, other neoplasms should be considered in the differential diagnosis (Abboud et al., 2007; Heo et al., 2013; Papanicolau-Sengos et al., 2013; Parelkar et al., 2012)Interestingly, FNA biopsy for the first patient was reported as thyroid follicular lesion.

Histopathological examination of the specimen of the first case, revealed atypical features of clear cell adenoma, which should be differentiated from parathyroid carcinoma clinically and histopathologically (Ioannis et al., 2018; LiVolsi et al., 2014). In our case, the diagnosis of WCCA was confirmed by both clinical and histopathological examinations. Even though, atypical adenomas are defined as adenomas that exhibit some features of malignancy, such as broad fibrous bands crossing the tumor or pseudocapsular invasion (clusters of parathyroid cells trapped within the capsule), they do not show vascular invasion, metastases or apparently increased mitotic activity. To best of our knowledge, this observation has not been reported previously for a water-clear clear cell adenoma.

The first case, parathyroid adenoma was located ectopically intra-thyroid which accounts for 1.4-6% of parathyroid adenomas of ectopic location. Parathyroid ectopia occurs in 4-20% of patients because of abnormal migration during embryogenesis or secondary to acquired migration (Issoufou et al., 2015; Parelkar et al., 2012; Roy et al., 2013).

Currently, clinical presentation of PHPT is either asymptomatic, with minor symptoms or it could be with an overt clinical picture and radiological manifestations (Bilezikian et al., 2016; Madkhali et al., 2016; Parelkar et al., 2012). Nonetheless, our first patient developed acute pancreatitis a month prior to presentation and hyperparathyroidism could be the cause of pancreatitis excluding other causes (Bilezikian et al., 2016; Madkhali et al., 2016; Parelkar et al., 2012). However acute pancreatitis was reported as initial manifestation of PHPT that was due to WCCA10 and the first case reported here should be the second in literature. Bone radiological manifestations in our case is a well-documented clinical picture reported in other

cases of PHPT (Varshney et al., 2013; Zanocco & Yeh, 2017).

Furthermore, our first case of WCCA has β-Thalassemia trait. This might be the first case of hyperparathyroidism associated with β-Thalassemia, as it is well known for β-Thalassemia to be associated with hypoparathyroidism (Shetty & Shenoy, 2014). Additionally, the first patient has type 2 DM and hypertension, however blood sugar control became possible following parathyroidectomy, which has been reported previously (Jena et al., 2016; Kumar & Singh, 2015; Reddy et al., 2009)..

The second case underwent minimal invasive surgery and parathyroidectomy and the adenoma was small weighing 0.3 gm, a clear cell parathyroid adenoma that might explain the negative Sestamibi scan as well as the low functional activity.

The differential diagnosis of WCCA includes, WCCH, clear cell changes in a thyroid tumor, paraganglioma, parathyroid chief cell adenoma with focal clear cell changes, clear cell lung tumors, clear cell sarcoma, Primary clear cell carcinoma of the thymus, parathyroid carcinoma, and metastatic renal cell carcinoma (Zinovkin et al., 2020). The distinctive histopathologic features of WCCA include optical clear cytoplasm, large clear cells with fine cytoplasmic vacuolization, high levels of cytoplasmic glycogen and fat, and absence of vascular invasion, metastases or apparently increased mitotic activity.

Conclusion

PHPT is a common endocrine disorder. Clinical presentation of PHPT varies from asymptomatic to insignificant, it might present with other complications such as pancreatitis. The most common cause of PHPT is parathyroid adenoma. Therefore, any suspected clinical condition associated with calcium abnormalities should be further investigated with biochemical and imaging workup to confirm the diagnosis of PHPT. The aim of this report is to highlight this rare variant of parathyroid adenomas, especially with WCCA could be the underlying cause of the clinical picture.

Conflict of Interest

All authors declare that they do not have any conflict of interest of any type.

Informed consent

Informed consent and approval from institutional review board were obtained for publication from Arrayan Hospital, Sulaiman Habib Medical Group, Riyadh, Saudi Arabia.

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Ethical approval

Informed consent and approval from institutional review board were obtained for publication from Arrayan Hospital, Sulaiman Habib Medical Group, Riyadh, Saudi Arabia.

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ورم الغدة الجار درقية ذو الخلايا الصافية: تقرير عن حالتين

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المُستَخلَص

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فرط نشاط جارات الدرقية الأولي هو مرض يتميز بفرط كالسيوم الدم والإفراط في إنتاج هرمون الغدة الجار درقية. وهو السبب الأكثر شيوعًا لفرط كالسيوم الدم في العيادات الخارجية وثالث الامراض الاكثر شيوعما من اضطرابات الغدد الصماء. يزداد معدل الإصابة بفرط نشاط جارات الدرقية الأولي مع تقدم العمر وهو أكثر شيوعًا عند النساء منه عند الرجال. 80-85٪ من حالات فرط نشاط جارات الدرق الأولي ناتجة عن ورم الفدة الجار درقية. تحتوي غالبية الأورام الغدية جارات الدرقية على مجموعة خلايا مختلطة مع غلبة الخلايا الرئيسية. فرط نشاط جارات الدرقية الأولي مع الورم الحميد الخلوي الصافي نادر جدًا.

نحن هنا نشارك حالتين من الورم الحميد جارات الدرقية ذو الخلايا الصافية المرتبط بفرط نشاط جارات الدرقية الأولي مع عرض المظاهر السريرية مختلفة.

الكلمات الدالة: فرط نشاط جارات الدرق الأولي، خلية صافية للماء، ورم الغدة الجار درقية، هشاشة العظام، التهاب البنكرياس، ارتفاع ضغط الدم، داء السكري، الثلاسيميا.