



Asian Journal of Science and Technology Vol. 10, Issue, 03, pp.9540-9543, March, 2019

RESEARCH ARTICLE

PARATHYROID ADENOMA - A CASE REPORT

¹Kuldeep Nahar and ^{2,*}Sunil Kumar Sharma

¹Orthopedic department, The Gujarat Research and Medical Institute, Rajasthan Hospital, Shahibaug, Ahmedabad, Gujarat, India 380004

²Otorhinolaryngology and Head and Neck surgery, The Gujarat Research and Medical Institute, Rajasthan Hospital, Shahibaug, Ahmedabad, Gujarat, India 380004

ARTICLE INFO

Article History:

Received 18th December, 2018 Received in revised form 29th January, 2019 Accepted 18th February, 2019 Published online 30th March, 2019

Key words:

Parathyroid adenoma, Parathyroid hormone.

*Corresponding author: Sunil Kumar Sharma

ABSTRACT

A parathyroid adenoma is a benign tumor on one of your parathyroid glands. These are four very small glands located near or at the back of your thyroid gland. They produce parathyroid hormone (PTH). This hormone helps control the amount of calcium and phosphorus in your blood. A parathyroid adenoma causes the affected gland to release more PTH than it should. This disrupts your calcium and phosphorus balance. This condition is called hyperparathyroidism. Primary hyperparathyroidism is a common endocrine disorder mostly associated to parathyroid adenomas. Although those tend to be small in size, rare cases of giant parathyroid adenomas may be present.

Citation: Kuldeep Nahar and Sunil Kumar Sharma, 2019. "Parathyroid adenoma - A Case Report", Asian Journal of Science and Technology, 09, (03), 9540-9543

Copyright © 2019, Kuldeep nahar and Sunil Kumar Sharma. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

INTRODUCTION

The incidence of primary hyperparathyroidism (PHPT) is increasing with rate of 42:100,000 per year. While in women over 60 years of age the average annual incidence rate approaches 190:100,000 per year. Whether this gradual rise reflects a true increase in the incidence of PHPT, a greater use of routine testing of serum calcium or an altered referral pattern for surgery is not known. Primary hyperparathyroidism is usually seen in females above the age of 50 years, with a prevalence of 21/1000,1 whereas the incidence in patients aged 12-28 years is less than 5%.2 A solitary adenoma is responsible for 80% of cases of primary hyperparathyroidism. Primary hyperparathyroidism is most commonly asymptomatic. The incidence of acute pancreatitis associated hyperparathyroidism is less than 10%. The incidence of hyperparathyroidism associated with a Brown tumour is less than 5%.

CASE REPORT

A 16 year old male Patient was admitted in our hospital with multiple fracture of leg bones, weakness and vague abdominal pain for the past 1 years. Patient operated 3 times for multiple femur or tibia fracture in both legs which occurs during routine activity.

Preoperative exams indicated primary hyperparathyroidism as a cause to his symptomatology, with elevated values of parathormone and high level of serum calcium. Ultrasound scan and MRI of her cervical region uncovered a giant 3 × 2 cm parathyroid adenoma, located in the lower left thyroid lobe. Complete investigative workup revealed a solitary parathyroid adenoma causing hyperparathyroidism. Surgical exploration with excision of the parathyroid adenoma was performed, following which the patient recovered uneventfully. Despite its size, the gland was successfully removed through implementation of Microscopic parathyroidectomy. He was uneventfully discharged on the 5th postoperative day.

DISCUSSION

Although common reason for developing hyperparathyroidism, parathyroid adenomas may rarely present with exaggerated dimensions and weight. Physical examination is usually unremarkable, while patients may present with symptomatology associated with elevated calcium levels. Treatment of this medical condition consists of surgical removal of the pathologic parathyroid gland either by bilateral exploration or through minimal invasive parathyroidectomy. Preoperative localization plays important role in the second case, since the method focuses on resection of a pre-op marked hyperactive parathyroid gland, through a small incision.





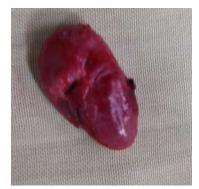


Fig. 1. Pre-op marking

Fig. 2. Intra op adenoma

Fig. 3. Post op parathyroid adenoma

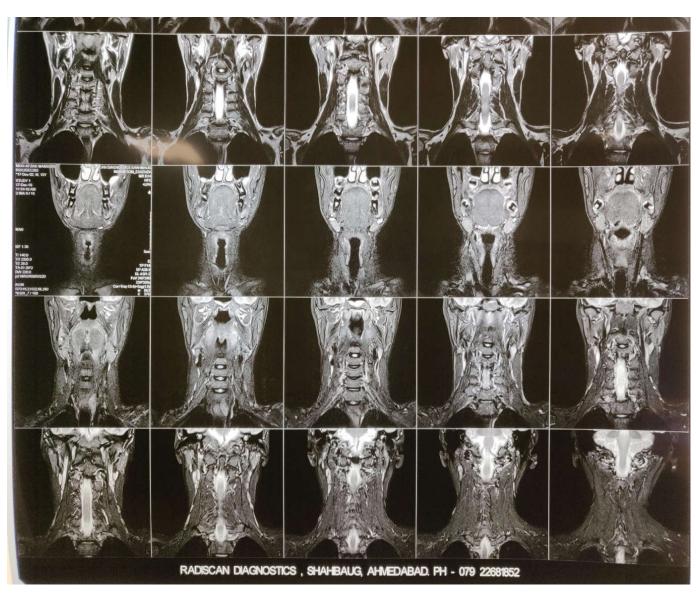


Fig. 4. Imagine: left inferior adenoma

DIAGINOS THE NEW STANDARDS IN DIAGNOSTICS www.radiscandiagnostics.com

NAME: MOH. AFZAL MANSURI DATE: 17/12/18 M/16 YRS

REF BY: DR. KULDEEP NAHAR (MS, ORTHO)

1.5T MRI OF NECK:

1.51 WINT C. MR imaging of the neck performed and high resolution T1 and T2-weighted serial sections obtained in the MR imaging of a large serial serial serial serial and axial planes on a 1.5Tesla scanner. Coronal images were also obtained.

A well defined heterogeneous soft tissue intensity lesion involving infrathyroid visceral space on left side. It appears separate from thyroid gland. Lesion appears hyperintense on T2 and fat suppressed images and hypo to isointense on T1

The lesion measures 16 x 12 x 29 mm (AP x TR x CC).

There is no invasion of trachea or major vessels noted.

The lesion is situated antero-medial to carotid vessels and lesion is postero-lateral to the trachea.

Craniocaudally, the lesion is extending from C7-D1 level. There is small tail like extension into prevertebral space.

The oral floor muscles are normally developed and bilaterally symmetrical. The spaces of the oral cavity and neck are clear and well defined.

Parotid and submandibular glands show no abnormalities.

The pharynx and larynx show normal boundaries and normal wall thickness.

The thyroid gland shows reasonable symmetry and normal size. The thyroid lobes display normal internal structure.

Cervical vessels have normal appearance.

The muscular structures of the neck are normal.

There are no signs of cervical lymphadenopathy.

No abnormalities are seen in the cervical spinal cord or cervical plexus.

Visualized cervical spine shows endplate sclerosis with central lucent areas (giving Rugger Jersey spine).

IMPRESSION:

- A well defined heterogeneous soft tissue intensity lesion involving infrathyroid visceral space on left side, separate from thyroid gland.
- No invasion of trachea or major vessels noted.
- Craniocaudally, the lesion is extending from C7-D1 level.
 - --- Possibility of parathyroid adenoma likely.

Adv: Clinical correlation, S.PTH and S.CA/PO4.

No definite thyroid mass lesion or cervical lymphadenopathy.

DR. RAJENDRA SOLANKI M.D.

IN GAJJAR

DR. RUCHIT PATEL DR. NIKUNJ BANKER M.D. M.D.

DR. BRIJESH GAJJAR M.D.

DR. YASHPAL RANA M.D.

A Noor SRI Bank, Ghoda Camp Road, Shahibaug, Ahmedabad-380004.

Treatment

The patient was electively posted for surgery. Excision of the parathyroid adenoma with exploration of the remaining parathyroid glands was done. Neck was accessed via a transverse skin crease incision. Subplatysmal flaps were raised and strap muscles retracted laterally. Strap muscles of the right side were divided horizontally to expose the right lobe of the thyroid. The thyroid was retracted medially to expose the posteromedial border, thus enabling visualization of the parathyroid adenoma. It was seen as a smooth, oval, brown to caramel color structure near the lower pole of the right lobe of the thyroid, seen to be arising from the left inferior parathyroid gland, measuring approximately 3 ×2 cm in size. The adenoma was then dissected free from the surrounding thyroid gland, finally isolating its blood supply. It was then excised after ligating the feeding vessel, and sent for frozen section. Frozen section report confirmed the mass to be a parathyroid adenoma. The rest of the parathyroid glands were explored and were found to be normal, hence were left in situ.

Histopathology

The excised parathyroid adenoma was then sent for histopathological examination. The diagnosis of parathyroid adenoma was confirmed on histopathological examination by presence of normal parathyroid glandular tissue, markedly reduced amount of adipose tissue and an intact capsule.

REFERENCES

- Adami S., Marcocci C., Gatti D. 2002. Epidemiology of primary hyperparathyroidism in Europe. *J Bone Miner Res: Off J Am Soc Bone Miner Res.*, 17(2):N18–N23.
- Biondi A., Persiani R., Marchese M., Cananzi F., D'Ugo D. 2011. Acute pancreatitis associated with primary hyperparathyroidism. *Updates Surg.*, 63(June (2)):135–138. [PubMed].
- Chowdhury S., Aggarwal A., Mittal N., Shah A. 2013. Brown tumor of hyperparathyroidism involving craniomaxillofacial region: a rare case report and literature review. *Minerva Stomatol.*, 62(September (9)):343–348. [PubMed].
- Lee C.C., Chao A.S., Chang Y.L., Peng H.H., Wang T.H., Chao A. 2014. Acute pancreatitis secondary to primary hyperparathyroidism in a postpartum patient: a case report and literature review. *Taiwan J Obstet Gynecol.*, 53(June (2)):252–255. [PubMed]
- Udelsman R. 2002. Six hundred fifty-six consecutive explorations for primary hyperparathyroidism. *Ann Surg.*, 235:665–670. discussion 70–2. [PubMed]
- Yeh Michael W., Ituarte Philip H.G., Zou Hui Cynthia, Nishimoto Stacie, Liu In-Lu Amy, Harari Avital. 2013. Incidence and prevalence of primary hyperparathyroidism in a racially mixed population. *J Clin Endocrinol Metab.*, 983:1122–1129. [PubMed].
- Zhao Lin, Liu Jian-min, He Xiao-Yan, Zhao Hong-yan, Sun Li-hao, Tao Beim, 2013. The changing clinical patterns of primary hyperparathyroidism in Chinese patients: data from 2000 to 2010 in a single clinical center. *J Clin Endocrinol Metab*, 98(February (2))
