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Imaging in Primary Hyperparathyroidism

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I. Introduction:

hyperparathyroidism is a rare disease with some metabolic presentation. Excessive production of PTH, termed hyperparathyroidism, is assessed as primary, secondary, or tertiary in form. Primary hyperparathyroidism, due to autonomous hypersecretion of PTH, usually occurs within the setting of a parathyroid adenoma (80%) but also can be seen with parathyroid hyperplasia (15%–20%) or carcinoma .Secondary hyperparathyroidism results from stimulation of the parathyroid glands as a response to hypocalcaemia or due to apparent insensitivity of the parathyroid glands to elevated serum calcium levels and dysregulation of the traditional feedback loop (pseudohypoparathyroidism). the foremost common explanation for secondary hyperparathyroidism is kidney failure , which ends up in phosphate retention, hypocalcaemia, and 1,25(OH)2D3 deficiency, resulting in a compensatory increase within the production of PTH (,3). Tertiary hyperparathyroidism is seen in cases of secondary hyperparathyroidism during which the parathyroid glands still function autonomously despite correction of the initial cause, leading to hypersecretion of PTH within the setting of normal calcium levels

CASE I

A 28 year-old woman presented in neurosurgery opd with a swelling on right parietal region andright upper quadrant pain. Few non-specific complaints such as anorexia , constipation , bilateral flank pain, chronic fatigue , joint pains and irritability. No significant medical history was given by patient. A review of systems revealed recent fatigue, weight loss, and slight burning with urination, but the results were otherwise unremarkable. Subsequent laboratory analysis demonstrated hypercalcemia with a serum calcium level of 17.6 mg/dL .A low serum phosphorus level of 2.3 mg/dL and an elevated parathyroid hormone level of 780 pg/mL.After evaluation of the imaging findings detailed in the next section, the diagnosis of primary hyperparathyroidism resulting from a solitary right parathyroid adenoma was made.The skull radiographs demonstrated a expansile lytic lesion with sclerotic margins in the right parietal region with pepper pot skull and two small osteolytic lesion ins the mandible .It give suspicious clue to hyper parathyroidism.Subsequent skeletal survey showsgeneralized osteopenia, erosion of the terminal phalangeal tufts (acro-osteolysis) and subperiosteal resorption of bone particularly the radial aspects of the 2nd and 3rd middle phalanges. End plate sclerosis is noted in the dorso lumbar spine. Abdominal ultrasound (US) demonstrated bilateral nephrocalcinosis. Thyroid US shows a well defined hypoechoic lesion ofsize 11 x 8 mmwith internal cystic changes and vascularity seen separately from right lobe thyroid capsule on posterior aspect. CT demonstrated multiple variable sized lytic lesions in ribs , mandible and calvaria.

CASEII

A 32 year-old woman presented with a swelling in right mandible since last one year. Few non-specific complaints such as anorexia , constipation , chronic fatigue , joint pains and irritability. No significant medical history was given by patient. Subsequent laboratory analysis demonstrated hypercalcemia with a serum calcium level of 14.1 mg/dL . A low serum phosphorus level of 2.1 mg/dL and an elevated parathyroid hormone level of 680 pg/mL. After evaluation of the imaging findings detailed in the next section, the diagnosis of primary hyperparathyroidism was made.The skull radiographs demonstrated a expansile lytic lesion with sclerotic margins in the right mandibular region.Subsequent skeletal survey shows osteoporosis with few small osteolytic lesion with peripheral sclerosis in middle proximal phalanx, tufting of the terminal phalangeal and subperiosteal resorption of bone particularly the radial aspects of the 3rd middle phalanges. Large osteolytic lesion with calcified internal septation seen in proximal diaphysis of right ulna and radius.On MRI face and neck a large expansile mass is seen in body of right mandible. The lesion in hypointense on T1 and hyperintense

on T2. Post GD shows patchy enhancement. There is bony destruction in right mandible involving the alveolus of canine and molar tooth (floating tooth appearance) . Thyroid and Parathyroid glands are normal. Patient was advised scintigraphy but the patient refused.

II. Discussion

A product of the chief cells of the four parathyroid glands, parathormone (PTH) may be a key modulator of calcium homeostasis. Complex in its metabolic function, PTH helps maintain the serum calcium level by mobilizing calcium at the bone surface, stimulating osteoclast-mediated bone resorption (,1). PTH also promotes renal hydroxylation of 25-hydroxy–vitamin D, which, additionally to being instrumental in intestinal absorption of calcium, binds to intranuclear receptors within bone, producing mediators of calcium mobilization and mineralization of organic matrix (,2). Other effects of PTH include promotion of renal tubular reabsorption of calcium and lowering of serum phosphate level by inducing phosphaturia (,3).

Excessive production of PTH, termed hyperparathyroidism, is assessed as primary, secondary, or tertiary in form. Primary hyperparathyroidism, due to autonomous hypersecretion of PTH, usually occurs within the setting of a parathyroid adenoma (80%) but also can be seen with parathyroid hyperplasia (15%–20%) or carcinoma .

Secondary hyperparathyroidism results from stimulation of the parathyroid glands as a response to hypocalcemia or due to apparent insensitivity of the parathyroid glands to elevated serum calcium levels and dysregulation of the traditional feedback loop (pseudohypoparathyroidism). the foremost common explanation for secondary hyperparathyroidism is kidney failure , which ends up in phosphate retention, hypocalcemia, and 1,25(OH)2D3 deficiency, resulting in a compensatory increase within the production of PTH (,3). Tertiary hyperparathyroidism is seen in cases of secondary hyperparathyroidism during which the parathyroid glands still function autonomously despite correction of the initial cause, leading to hypersecretion of PTH within the setting of normal calcium levels (,5). Although most patients with primary hyperparathyroidism today are asymptomatic due to the widespread availability of laboratory screening for hypercalcemia and earlier detection, the clinical presentation is variable (,6). Previously encountered overt symptoms including dementia, depression, peptic ulceration disease, pancreatitis, constipation, renal calculi, and diffuse bone and joint pain are not any longer common. Patients now often complain of mild subjective symptoms like weakness and straightforward fatigability (,7).

The classic imaging features of advanced primary hyperparathyroidism also are less frequently seen today. Generalized osteopenia is that the commonest imaging finding in primary hyperparathyroidism. Only very rarely encountered in primary hyperparathyroidism, diffuse or localized osteosclerosis may be a more common finding in secondary hyperparathyroidism. When involving the spine, sclerosis can produce a striped appearance, the so-called "rugger jersey" spine (,4).

Subperiosteal bone resorption may be a common finding of advanced hyperparathyroidism, most frequently seen involving the hands and therefore the feet. Although classically most pronounced at the radial aspects of the second and third middle phalanges, subperiosteal resorption also can be seen involving the medial aspects of the metaphyses of the long bones also the ribs and lamina dura of the teeth. Terminal tuft resorption also can be seen, and hyperparathyroidism should be considered within the medical diagnosis for acro-osteolysis (,2). As in our case, the digits may appear spatulous or clubbed, presumably due to soft-tissue collapse resulting from marked terminal tuft erosion (,8). Other areas of bone resorption are seen at sites of high bone area and include subchondral locations, often best appreciated at the sacroiliac, acromioclavicular, sternoclavicular, and temporomandibular joints, the symphysis pubis, and therefore the patella. Resorption of subligamentous and subtendinous bone occurs most frequently at the femoral trochanters, the ischial tuberosities, the calcaneal insertions of the plantar aponeurosis and Achilles tendon , the inferior margin of the distal clavicle, and therefore the tuberosities of the humerus (,4). Intracortical, endosteal, and trabecular bone resorption also can be present. Brown tumors, also referred to as osteoclastomas, are eccentrically located and sometimes expansile lesions resulting from amassing osteoclasts and. Chondrocalcinosis may be a nonspecific finding that features a higher prevalence in primary instead of secondary hyperparathyroidism (,2). Although also having a better prevalence in primary hyperparathyroidism, brown tumors are more frequently encountered within the setting of secondary hyperparathyroidism due to the greater prevalence of this condition (,2). Soft-tissue calcification and periostitis are among the radiographic findings more commonly seen in secondary hyperparathyroidism (,2).

Although directed US examination, CT of the neck, and resonance imaging are useful within the complete evaluation of primary hyperparathyroidism, 99mTc-sestamibi parathyroid scintigraphy is now considered the simplest preoperative localizing modality for the detection of parathyroid adenomas

(,9). As parathyroid scintigraphy are often limited by the coexistence of thyroid nodules or other metabolically active tissues like lymph nodes, diffuse hyperplasia, or metastatic thyroid cancer, it's often correlated with CT results to yield functional and anatomic localization (,5).

As most patients with primary hyperparathyroidism are asymptomatic, treatment is controversial. Given the potential unpredictable long-term complications including recurrent urolithiasis and osteoporosis, some clinicians believe that surgical management is actually always appropriate (,7). The National Institutes of Health (NIH) consensus statement in 1990 concluded that while surgical intervention is that the acceptable treatment of primary hyperparathyroidism, "conscientious surveillance could also be justified in patients whose calcium levels are only mildly elevated and whose renal and bone status are on the brink of normal." NIH criteria identified for choosing patients for parathyroidectomy include the following: (a) typical parathyroid-related symptoms involving the skeletal, renal, or gastrointestinal systems; (b) a sustained elevation of serum calcium level quite 1–1.6 mg/dL (0.25–0.40 mmol/L) above the upper limits of normal; (c) a considerable decline in bone mass; (d) a decline in renal function by 30% or more; (e) nephrolithiasis or worsening of calciuria; (f) severe neuromuscular or psychological problems; or (g) unwillingness of the patient to continue under medical supervision (,10)

Illustrations

CASE 1

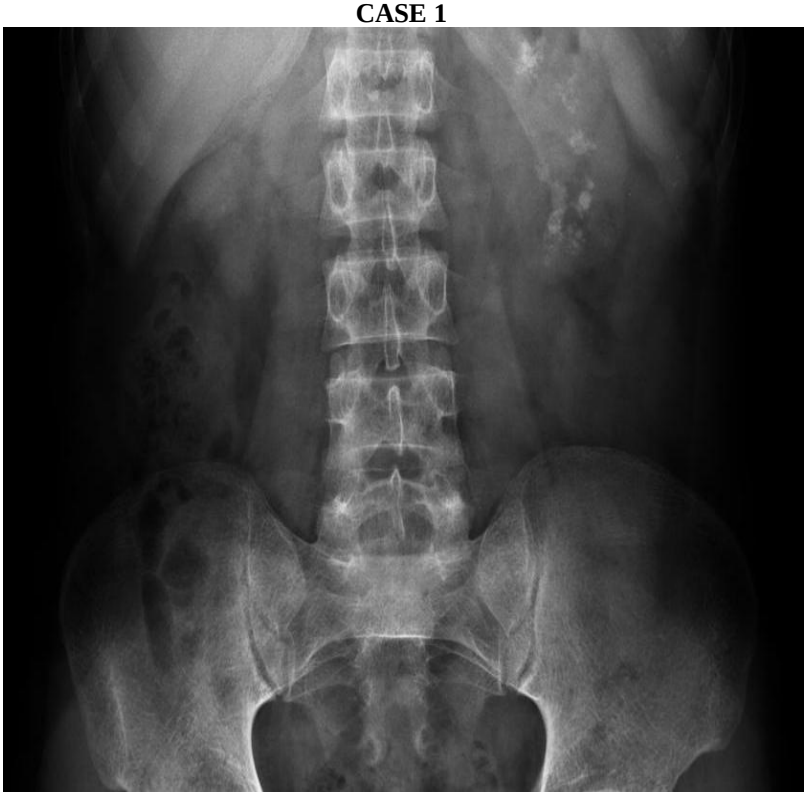

Fig 1 shows fine granular opacities over renal parenchymal outlines s/o nephrocalcinosis

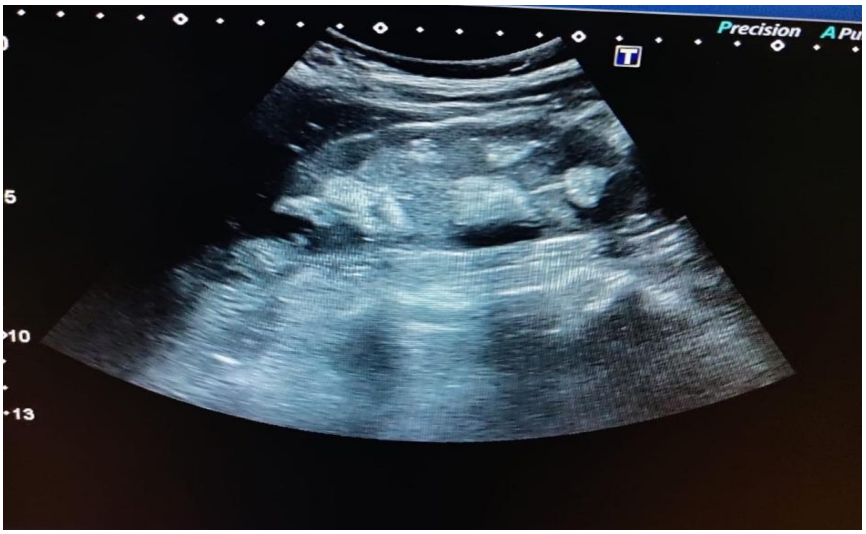

Fig 2 show US corelation showing multiple medullary caliculi suggestive of nephrocalcinosis.

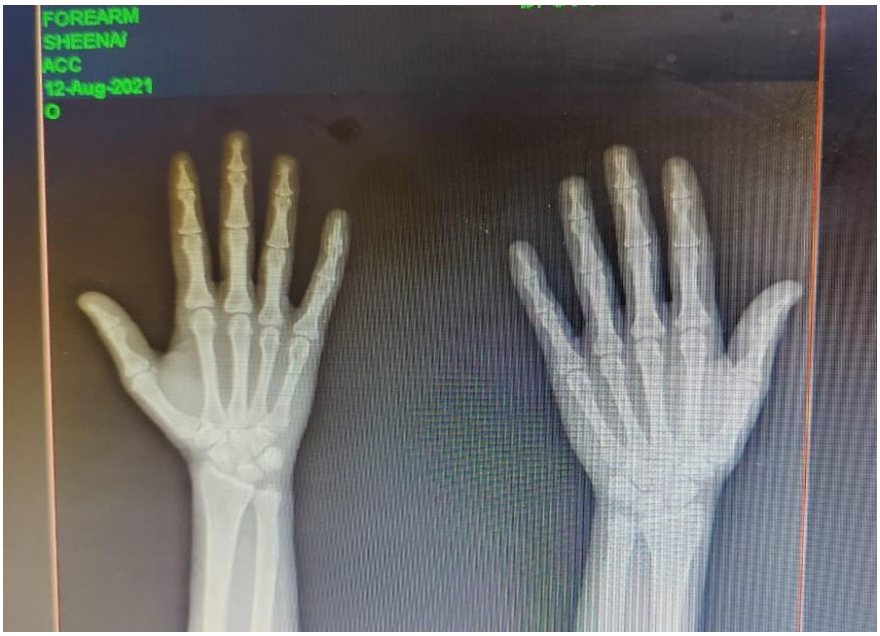

Fig 3 shows generalized osteopenia, erosion of the terminal phalangeal tufts (acro-osteolysis) and subperiosteal resorption of bone particularly the radial aspects of the 2nd and 3rd middle phalanges.

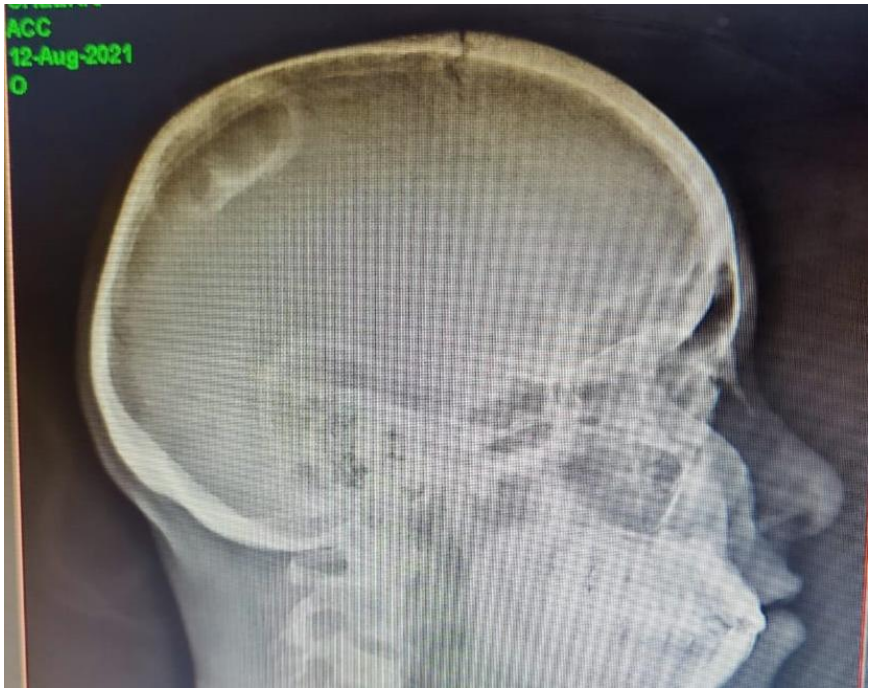

Fig 4 shows "Salt-and-pepper-skull." Lateral skull X-ray with salt-and-pepper appearance from trabecular bone resorption depicted as fine areas of lucency mixed with sclerotic radiopaque-denser-dot-like foci.

Expansile lesion is also noted in parieto-occipital region with sclerotic margins S/o Brown's Tumour.

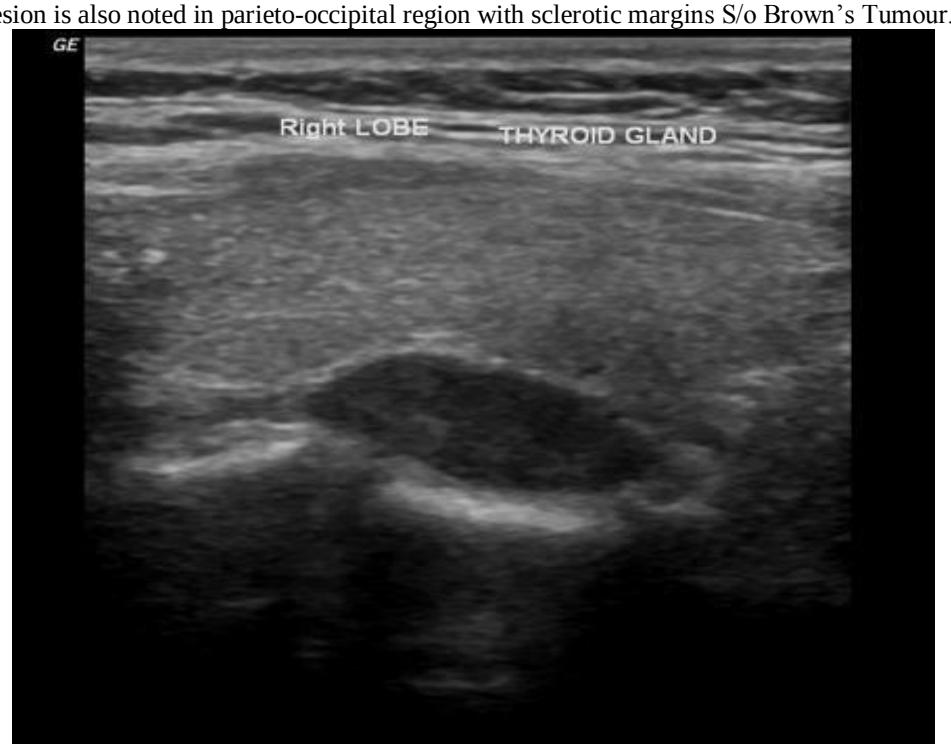

Fig 5 shows Findings – A well defined hypoechoic lesion with internal cystic changes and vascularity seen separately from right lobe thyroid capsule on posterior aspect.

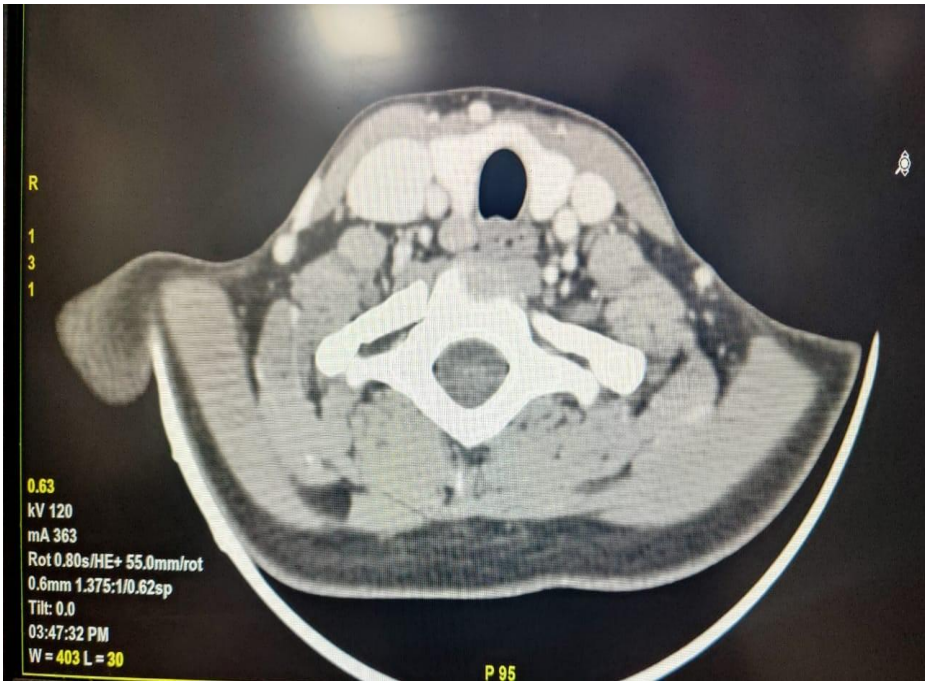

Fig 6 shows a lesion seen just inferior to lower pole of right thyroid gland and appears slightly hypodense to thyroid on venous phase likely right parathyroid mass lesion.

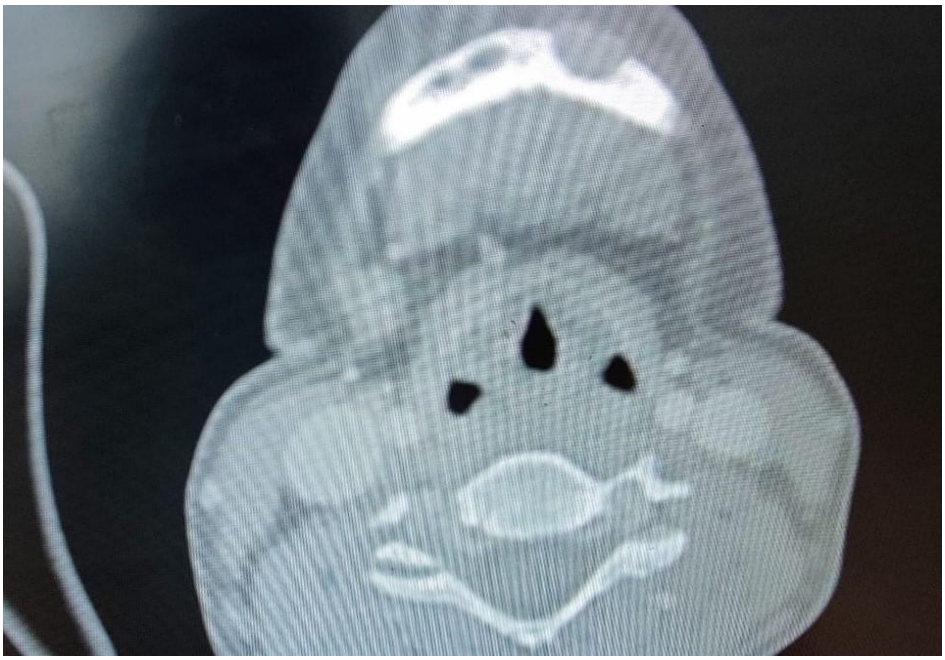

Fig 7 shows two expansile lesion are also seen in mandible s/o Brown's tumour.

CASE II

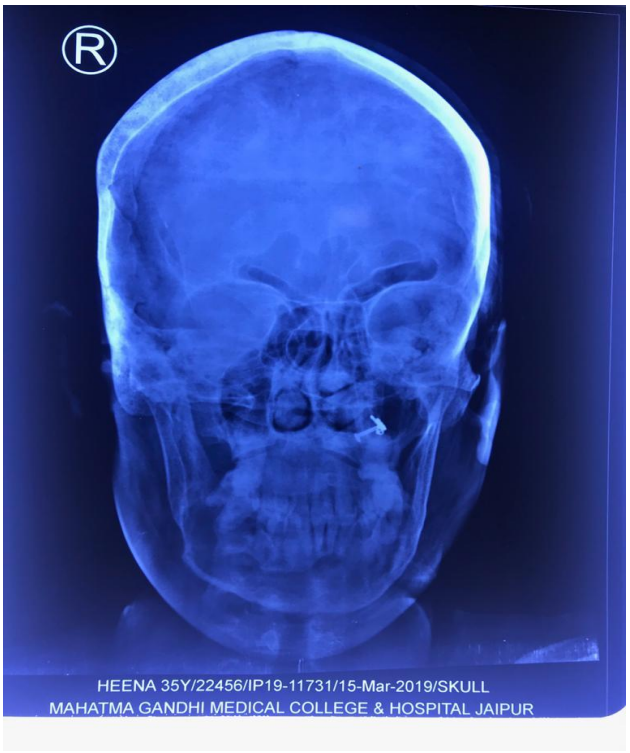

Fig 1. shows The skull radiographs demonstrated a expansile lytic lesion with sclerotic margins in the right mandibular region.

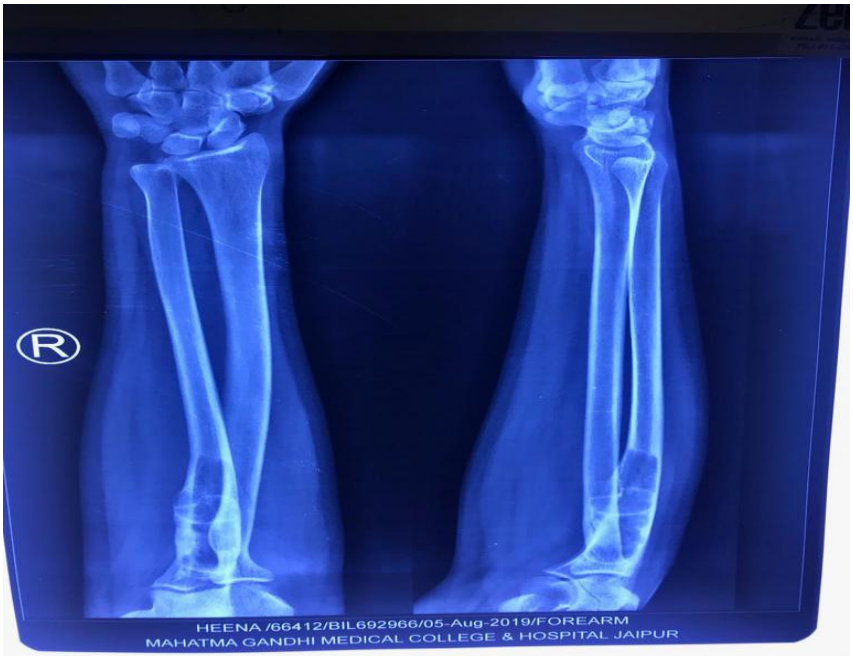

Fig 2 shows large osteolytic lesion with calcified internal septation seen in proximal diaphysis of right ulna and radius.

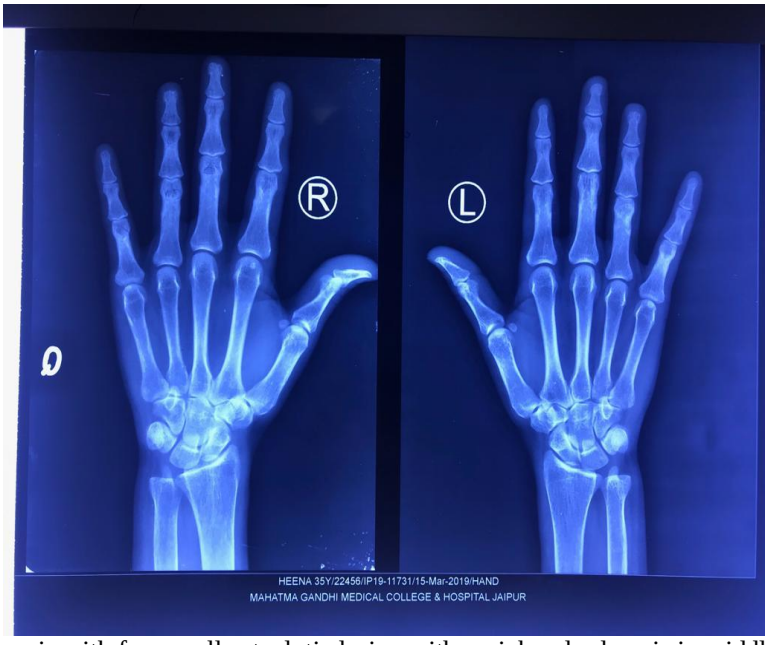

Fig 3 shows osteoporosis with few small osteolytic lesion with peripheral sclerosis in middle proximal phalanx, tufting of the terminal phalangeal and subperiosteal resorption of bone particularly the radial aspects of the 3rd middle phalanges.

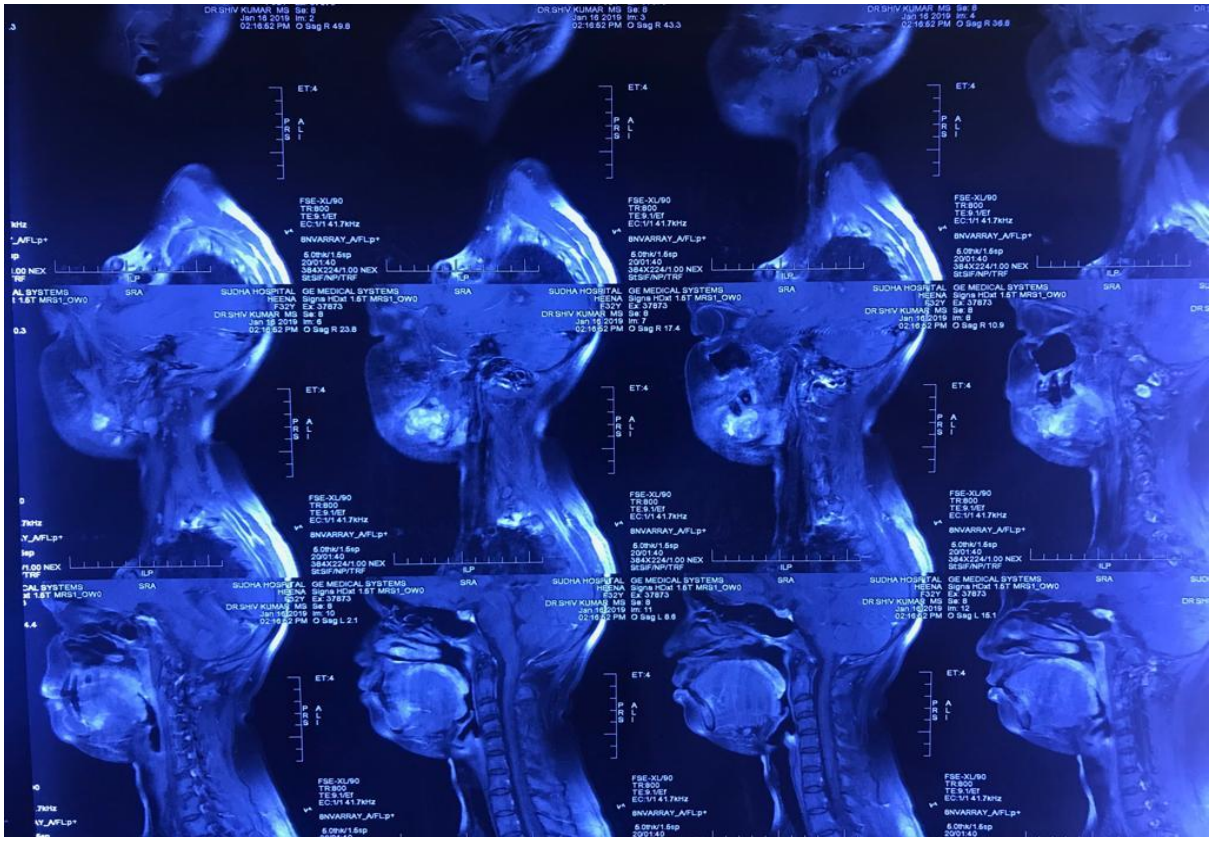

Fig 4 shows a large expansile mass is seen in body of right mandible. The lesion in hypointense on T1 and hyperintense on T2.

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