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BONE FORM OF PRIMARY HYPERPARATHYROIDISIS IN SURGERY PRACTICE

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Abstract:

Aim: *To study the possible clinical masks of primary hyperparathyroidism encountered in the practice of the surgeon.*

Materials and methods: *From our clinical practice, we selected 2 of the most striking clinical cases of primary hyperparathyroidism with bone form. Both patients were not immediately referred to an endocrine surgeon and suffered a large number of complications and falsely necessary surgical interventions.*

Results. As a result of the analysis of the clinical cases described in the article, we see that the poor awareness of doctors about the possible clinical manifestations of primary hyperparathyroidism leads to a false diagnosis and many unjustified medical manipulations. As a result, when the diagnosis was made, the patients underwent parathyroid adenoma removal with a positive clinical effect.

Conclusions: *Thus, surgical treatment of hyperparathyroidism is effective, but the most rational is the naturally specific visualization of the glands using scintigraphy. In some cases, standard research methods need to be supplemented with genetic analysis, for the differentiated diagnosis of hereditary and acquired forms of hyperparathyroidism.*

Key words: *hyperparathyroidism, diagnosis, treatment.*

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INTRODUCTION:

Primary hyperparathyroidism (PHPT) is the most common endocrinopathy, is the third most common endocrine pathology after thyroid disease and diabetes, and is the main cause of hypercalcemia. The disease is 2 times more common in women than in men (in the postmenopausal period - 5 times more often), it is observed at the age of 20-70 years, the peak incidence occurs in 40-60 years. A single adenoma of one of the parathyroid glands occurs in 80-90% of cases of the disease that is the cause of this disease. The clinical forms of hyperparathyroidism include: 1) renal (50% of patients with hyperparathyroidism have nephrolithiasis); 2) bone; 3) digestive (gastric ulcer, cholecystitis, pancreatitis); 4) cardiovascular; 5) mixed [1, 2, 3]. Raising the educational level of a wide range of doctors, and especially traumatologists, urologists, neuropathologists, maxillofacial surgeons, and the possibility of hormonal confirmation of the diagnosis, make it possible to radically change the idea of the prevalence of the disease, to identify and treat PHPT at the stage of initial laboratory and clinical manifestations. In Russia, only 5% of patients operate at the preclinical stage of PHPT, that is, with laboratory changes [6,8].

In our article, we want to focus on the bone form of PHPT. The most common and early clinical manifestations of PHPT are bone changes. Most patients associate the onset of the disease only with the appearance of the first clinically significant symptoms (pathological fractures, the appearance of bone tumors) [7]. With PHPT due to the increased concentration of parathyroid hormone, diffuse demineralization of the skeleton bones occurs. Local pronounced absorption of bone tissue with radiological signs of cystic degeneration can simulate a primary bone tumor. Filling a bone defect with fibroblastic structures that change the contour of the bone creates an x-ray symptom complex that is as similar as possible to the tumor process (osteitis fibrosa cystica). The x-ray picture of subperiosteal resorption of the bones of the hand is an important additional feature that helps to identify the true cause of bone changes. Skeletal changes in parathyroid osteodystrophy may be mistaken for a primary bone tumor or secondary metastatic lesion. Moreover, it should be noted that the lesion in hyperparathyroidism is devoid of characteristic histological signs. In resorption zones of bone trabeculae, foci of proliferation of fibroblastic elements of connective tissue with abundant inclusions of groups of giant cells appear, which, first of all, suggests a giant cell tumor [2]. Often, hyperparathyroid osteodystrophy is mistaken for metastases of a malignant tumor without a primary focus. In 20% of cases, hyperparathyroidism can be asymptomatic. Specific changes in soft tissues (ovoid calcification) are found only in secondary and tertiary hyperparathyroidism in patients receiving hemodialysis [9]. Given the clinical and radiological picture, 3 types of parathyroid osteodystrophy are distinguished: 1) osteoporotic; 2) fibrocystic - with the formation in the bones of the so-called brown tumors and cysts; 3) “pagetoid”, in which, against the background of osteoporosis in flat bones, there are areas of bone tissue remodeling with spotted osteosclerosis, which resembles the picture of Paget's disease [3]. There are descriptions of a number of clinical cases in the literature when, due to an erroneously established diagnosis of osteoblastoclastoma, patients were repeatedly operated on unsuccessfully and only after a diagnosis of PHPT and removal of the parathyroid adenomas, the clinical picture was regressed [1, 4, 5, 10, 11]. Removal of hyperhormone-producing tissue of the parathyroid gland is considered to be a generally accepted method of treatment for PHPT [1-10], however, a number of studies claim that genetic testing for possible mutations that determine the development of hereditary forms of hyperparathyroidism (MEN-1) should also be referred to the choice of therapeutic tactics for laboratory studies in primary hyperparathyroidism. , MEN-2a, PHT-JT syndrome, familial hyperparathyroidism syndrome) and pathology variants of the gene encoding the calcium receptor [12].

Quite rarely, patients with hyperparathyroidism and hypercalcemia come, but the diagnosis of PHPT is questionable. Due to the lack of the possibility of genetic research and the refusal of patients to go to more specialized centers with greater diagnostic capabilities due to disability and the possibility of transportation or psychological refusal to stay in another medical institution, it is necessary to help patients situationally. We present our clinical case of hyperparathyroidism in all likelihood associated with a hereditary form, but for the above reasons, the diagnosis cannot be finally established.

MATERIAL AND METHODS:

From our clinical practice, we selected 2 of the most striking clinical cases of primary hyperparathyroidism with bone form. Both patients were not immediately referred to an endocrine surgeon and suffered a large number of complications and falsely necessary surgical interventions.

RESULTS AND DISCUSSION:

Clinical case 1. Patient A., born in 1963, was admitted to the clinic of general surgery with a diagnosis of Primary Chronic Intrastitial Jade.

Complications of the main one: CKD C5 (D) ST., Prolonged renal replacement therapy with hemodialysis since 11/11/2010, Implantation of a Tenkhoff peritoneal catheter into the abdominal cavity. Secondary arterial hypertension 3st, 4risk. Secondary cardiomyopathy CH-2Ast. Severe secondary hyperparathyroidism, large chin calcine. Chronic iron deficiency anemia.

Demonstration of clinical material is carried out with the written permission of the patient, taking into account ethical standards.

Anamnesis of the disease: According to the patient, he has been ill since childhood. He was repeatedly examined and treated at the Endocrinology Center of Kazakhstan (no extracts), according to his parents, the diagnosis was not established, there were episodes of hypercalcemia and hyperglycemia, obesity. Upon receipt of a complaint of malnutrition, thirst, polyuria. Headache. Enuresis for a long time. In 2005, he was operated on for varicocele on the left. Since 2005, there has been an increase in blood pressure to 170/90 mm Hg. Art. In 2009 in the nephrology department, the doctor found: chronic glomerulonephritis. In 2010, a sharp deterioration in the state, was hospitalized in the nephrology department of the switchgear of the Design Bureau named after N.A. After a consultation of nephrologists, Semashko recommended renal replacement therapy. From 11.11.10g. - after the installation of the temporary vascular access of the AVF, situational hemodialysis on the right started. 13.01.11g operation Implantation of the Tenkhoff peritoneal catheter into the abdominal cavity and peritoneal dialysis started. Given the compensation of the general condition, the patient's refusal from further treatment with programmed hemodialysis 08/01/2015. by a decision of the consultation, a change of modality was carried out - a return to permanent outpatient peritoneal dialysis.

Upon admission to the surgical clinic, complaints of bone pain, pathological formation of the lower jaw (Fig. 1), duck gait, decrease in growth by 6 cm over the past six months. Upon examination, parathyroid hormone 5800 pg/ml, calcium 2.75 mmol/L, Ca * P coefficient 1.74, phosphorus 0.97 mmol/L, alkaline phosphatase increased by 5 times.

Fig. 1. The appearance of the patient's chin before and after the disease.

An X-ray examination (Fig. 2) revealed destruction of the bones of the lower jaw, calcification in the soft tissues of the chin.

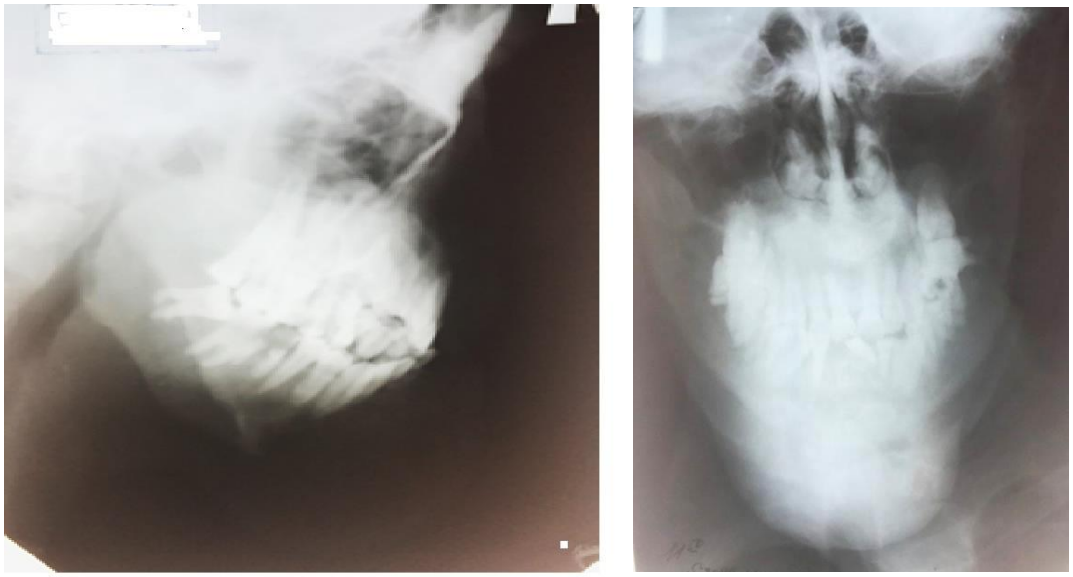

Fig. 2. Pathological changes in the bones of the skull with hyperparathyroidism.

MRI revealed a destruction of the lower jaw bone plate with cystic bone degeneration - osteoblastoclastoma? (Fig. 3).

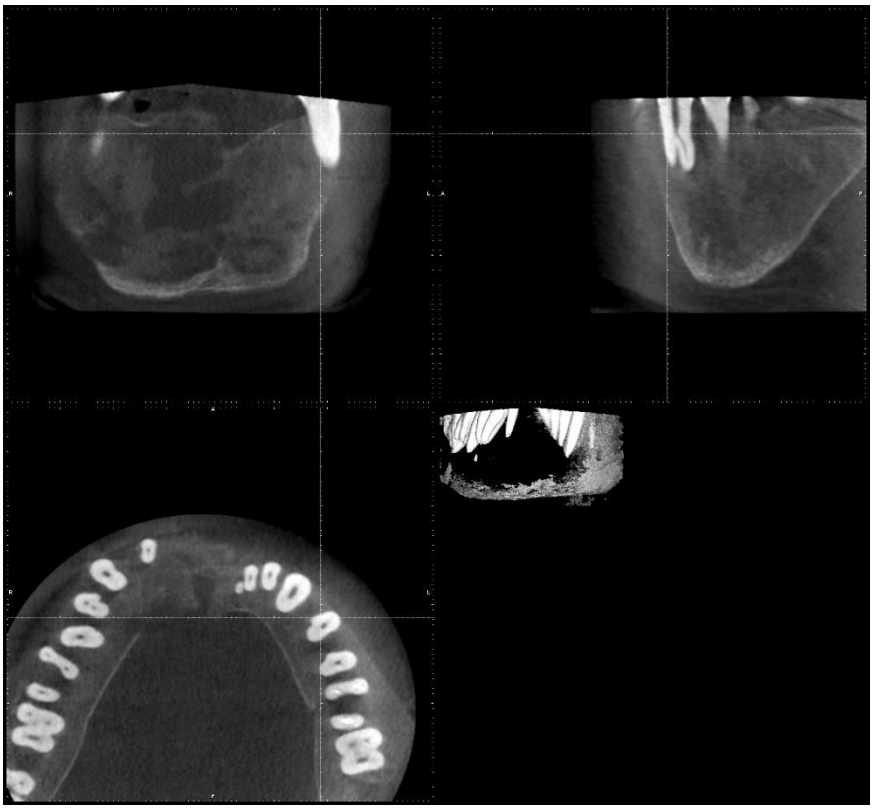

Fig. 3. MRI cystic restructuring of the lower jaw, in some areas complete destruction of the bone with pathological thinning of the lower jaw, free sagging of the roots of the teeth of the lower jaw.

After preoperative preparation and further examination, according to the MRI of the neck and ultrasound, there are adenomas of the lower left parathyroid gland with a diameter of up to 2 cm, the rest is not revealed pathology. The patient underwent removal of adenoma of the lower parathyroid gland on the left, a slightly enlarged parathyroid lower gland on the right. In the postoperative period, parathyroid hormone decreased to 1170 pg / ml, the histological conclusion was clear cell-adenoma of the parathyroid gland, the patient's bone pain disappeared. The patient in satisfactory condition the patient was discharged from the department, recommendations are given. In the postoperative period, the patient uncontrollably took calcimimetics, calcium preparations. However, after 5 months, the patient returned with a relapse - parathyroid hormone increased to 2800 pg / ml, with ultrasound - in the thyroid gland tissue a nodular formation up to 1.5 cm in diameter is visualized, parathyroid adenoma? The patient categorically refused further treatment and examination, consultations in specialized centers, trying to achieve transfer to peritoneal dialysis.

Clinical case 2. Patient D., born in 1938, was hospitalized at the Department of Bone Oncology since 11/14/11. on December 13, 2011, with a diagnosis of Hyperparathyroid osteodystrophy. A pathological fracture in the right tibia. Hypertonic disease 2st. (slowly progressing course). IHD: Atherosclerotic cardiosclerosis H1.

Upon admission, he complained of pain in the right lower leg, impaired function of the right lower limb.

An.morbi: He considers himself ill for about 10 months when there were pulling and bursting pains in his left leg. Gradually the pain grew, night pains joined. Radiography was performed - a neoplasm of the diaphysis of the left tibia, a pathological fracture. 06/15/11-performed trepan biopsy of education: pathological conclusion №6756-64 dated 06/22/2011. osteoblastoclastoma, lytic form. 06/24/2011 - performed the operation: Segmental resection with upper third of the left tibia, removal of the tumor with a soft tissue block. Intramedullary metallopolymer plastic defect. Pathological conclusion from 07/06/2011, No. 7200-14: giant cell tumor of the bone,

lytic form. The patient was discharged from the ward in satisfactory condition for further rehabilitation at the place of residence. In everyday life he moved with the help of a cane, fully loading the operated leg. 11/10/2011 during the exercise, the "bicycle" felt a sharp pain in the right lower leg and then could not lean on the right lower limb.

An.morbi: Extremity in a plaster cast. The area in upper third of the right tibia is painful on palpation. It is noted spindle-shaped thickening of it at this level. Inguinal lymph nodes are not palpable, b / painful. The skin above him is warm without changes. Joint movements in full. Pulsation in the peripheral arteries of the foot is satisfactory, the sensitivity of the foot is not impaired. On the lower extremities are varicose nodes.

An X-ray on 10.11.2011 shows a neoplasm in upper third of the right tibia, a pathological fracture in this area. Pathological rearrangement in lower third of the right b \ b bone.

Dz. neoplasm in upper third and lower third of the right tibia, pathological fracture at the level of upper third.

An examination in the blood revealed: parathyroid hormone -1192ppg/ml, calcium 3.56mmol/l, phosphorus-1.09mmol/l, alkaline phosphatase 245 units/l., Magnesium-0.91 mmol/l.

In order to stabilize the pathological fracture to the patient 11/18/2011, the operation was performed: Intramedicular fixation of the pathological fracture with a blocked rod, filling the cavity around the blocked screws with bone cement.

In the department received dressings, antibiotics, analgesics. The wound healed by primary intention. Sutures were removed on the 14th day.

01/20/12 the patient was admitted for inpatient treatment to the department of abdominal surgery and polytrauma. An additional examination on CT revealed neck formation located on the left side of the trachea, under the thyroid gland, extending retrosternally into the anterior mediastinum, 12 * 10 * 4cm in size. (Fig. 4), inhomogeneous density with an uneven contour. Conclusion: a parathyroid tumor?

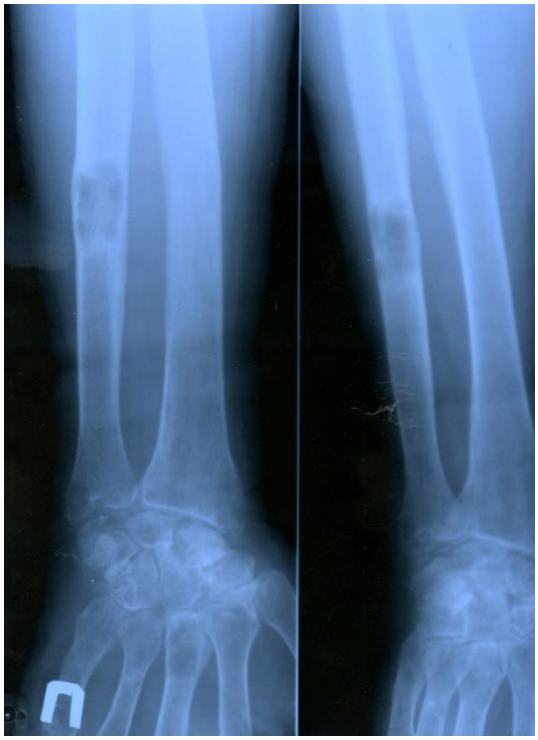

Fig. 4. Cystic bone remodeling right forearm

An X-ray examination of the bones of the upper extremities and skull revealed multiple cystic changes in the tubular bones, swelling of bone tissue, osteomalacia, pathological fracture of the humerus (Fig. 5) and uneven focal resorption and osteolysis of the temporal bone to the left of a rounded shape up to 3 cm in diameter (Fig. 6).

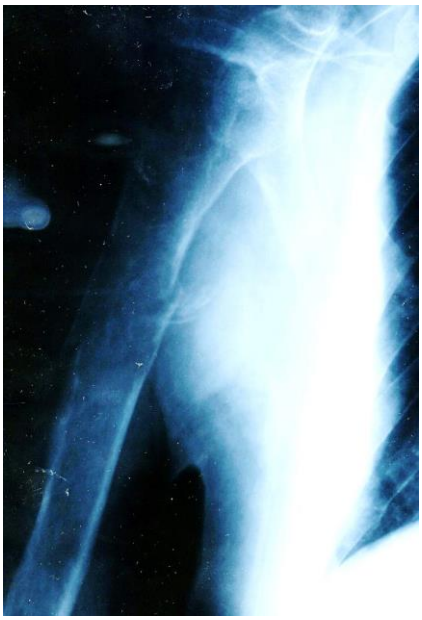

Fig. 5. A pathological fracture of the middle third of the left humerus at the site of cystic reconstruction (a picture through gypsum)

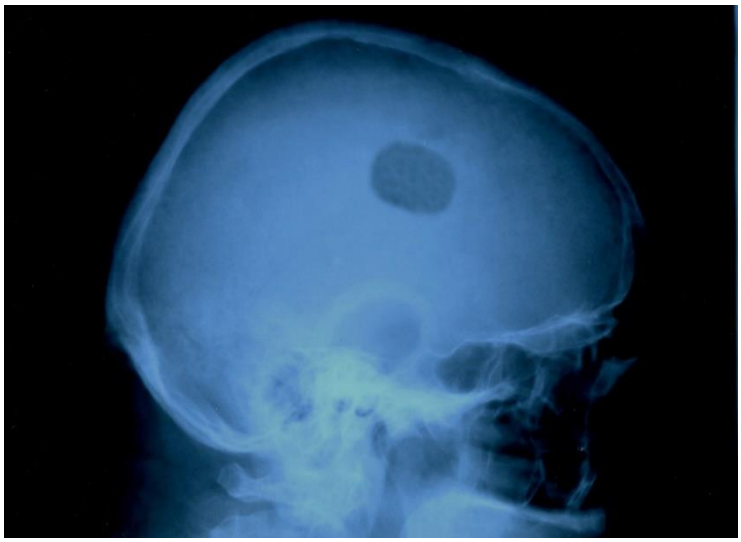

Fig. 6. X-ray signs of skeletal damage in primary hyperparathyroidism (osteolysis of the temporal bone on the left rounded to 3 cm, with relatively clear contours).

01/21/12 under general anesthesia under mechanical ventilation, from a Kocher incision without sternotomy, a parathyroid tumor was removed, emanating from the lower left parathyroid gland and extending 4 cm retrosternally. Urgent pathological conclusion of the removed drug: trabecular adenoma of the parathyroid gland.

The final histopathological diagnosis confirmed that the parathyroid adenoma was removed; in the postoperative period, the patient returned normal

levels of parathyroid hormone, blood calcium levels and alkaline phosphatase. Fractures consolidated, the patient feels satisfactory, leads an active lifestyle.

CONCLUSION:

Thus, surgical treatment of hyperparathyroidism is effective, but the most rational is the naturally specific visualization of the glands using scintigraphy. In some cases, standard research methods need to be supplemented with genetic analysis, for the differentiated diagnosis of hereditary and acquired forms of hyperparathyroidism.

List of symbols and Abbreviations:

AVF – arteriovenous fistula
CT – computer tomography
IHD – ischemic heart disease
MRI – magnetic resonance imaging
PHPT - primary hyperparathyroidism
X-ray – x-ray examination

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