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Case report

Severe hypercalcemia complicated by acute pancreatitis revealing generalized bone lysis metastasis: Case report and review ☆,☆☆

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ABSTRACT

Malignant hypercalcemia is a frequent metabolic complication of osteophilic tumors, exceptionally revealing cavitary cancer, but its prognosis remains poor despite early and adequate management. We report the case of a young patient, smoker without any previous history, admitted for the management of a digestive symptoms made of abdominal pain with food vomiting. The patient had an electrocardiogram withchest computed tomography scan and BBC evoking PA on malignant hypercalcemia. An etiological investigation was conducted to confirm the tumoral origin of the hypercalcemia. We put the patient on hyperhydration with corticotherapie and biohosphonates with a good clinical and biological improvement. Malignant hypercalcemia affects about 10%-20% of patients with cancer including nasopharyngeal carcinoma. Its clinical presentation varies according to the extent and speed of onset, responsible for multivisceral involvement including kidney, heart, neuropsychiatric system, which may engage the patient's vital prognosis. The therapeutic management is based on 4 main principles; hyperhydration, increase of urinary calcium excretion by loop diuretics, decrease of bone resorption by biphosphonates and extrarenal purification which remains the ideal choice in case of life-threatening severe hypercalcemia. Acute hypercalcemic pancreatitis as a mode of revelation of cavum cancer has almost never been described in the literature

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Introduction

Malignant hypercalcemia can lead to acute pancreatitis in less than one percent of cases provided the threshold of 3 mmol/L is exceeded. Among its etiologies, we note solid osteophilic tumors and rarely the cancer of the cavum. The objective of our work is to report the case of a patient who presented an acute pancreatitis complicating malignant hypercalcemia revealing a thickening of the cavum most likely related to neoplasia.

Case report

We report the case of a 55-year-old patient, active smoker, without any pathological history, admitted for the management of diffuse abdominal pain which began 3 days ago, resistant to the usual antispasmodic treatment, increased by food intake associated with food vomiting, with the notion of a recent weight loss of 10 kg in 2 months.

On admission, the patient was conscious, Glasgow Coma Scale 15/15, polypneic with a FR = 25 cpm and SpO2 = 95% on room air, tachycardia at 120 bpm, normotensive, T° 37.4 with a correct blood sugar level. The clinical examination was unremarkable except for epigastric tenderness.

ECG showed sinus tachycardia with QT segment shortening. Chest radiography showed osteolytic costal images. The biological workup on admission showed a corrected hypercalcemia of 194 mmol/L (normal value 88-105 mmol/L), a lipasemia of 7 times normal, a cholestasis and cytolysis workup without any particularity including GGT. Triglycerides, total cholesterol is normal. Abdominal ultrasound did not reveal any signs of intra- or extra-hepatic biliary dilatation or biliary lithiasis.

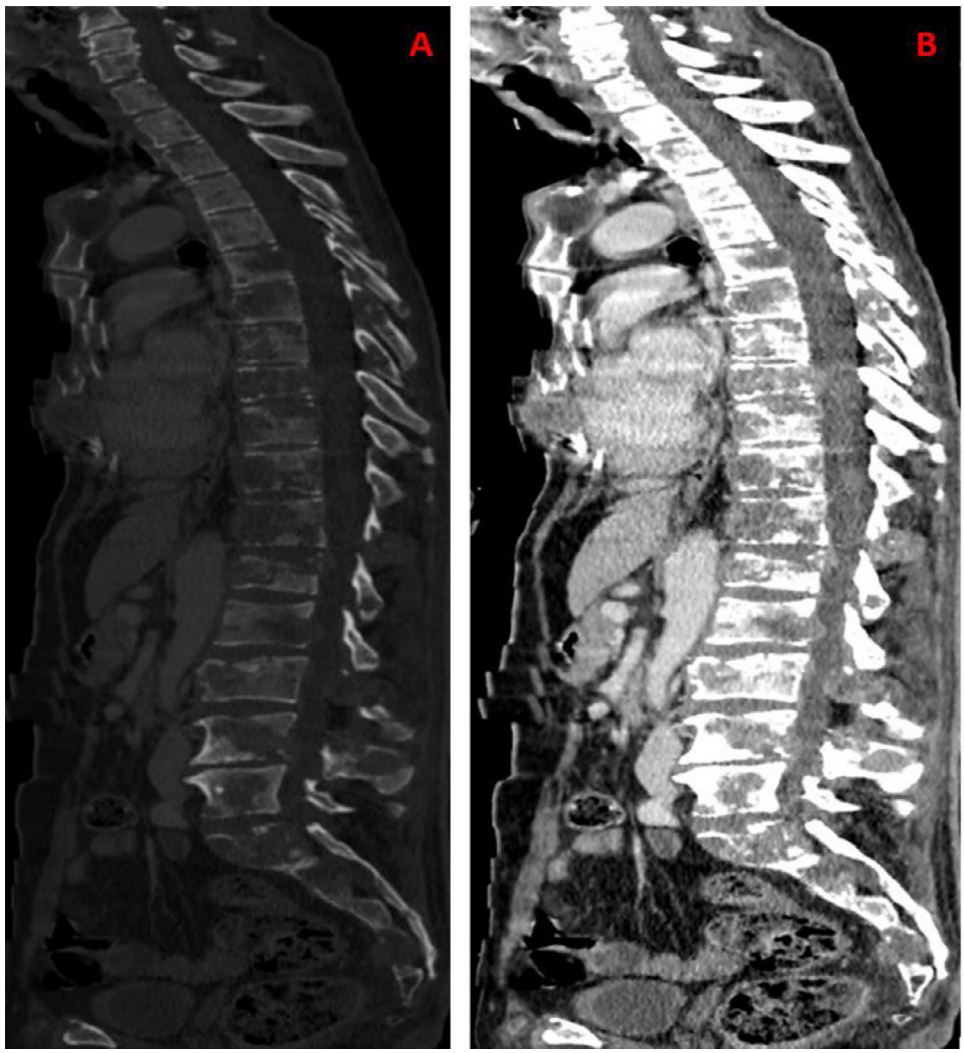

Fig. 1 – Computed tomography (CT) scan in sagittal spine reconstruction showed multiple osteolytic lesions in bone (A) and parenchymal windows (B).

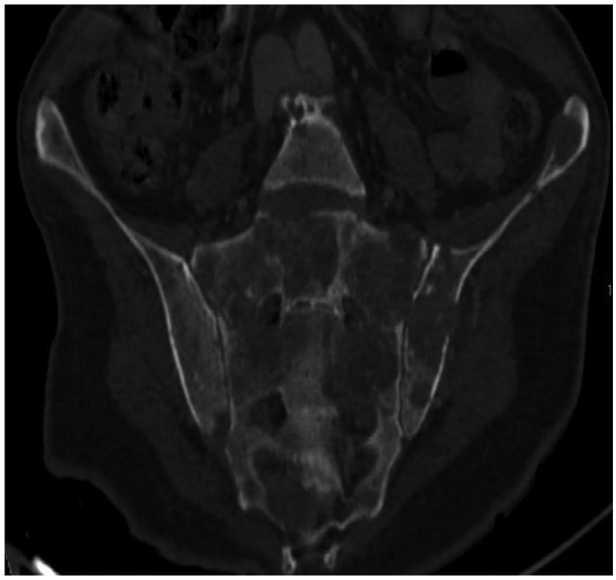

Fig. 2 – CT scan of the pelvis showing osteolytic lesions.

The diagnosis of acute pancreatitis on malignant hypercalcemia was made. The patient was put on a codeine-based analgesic treatment, with a rehydration based on ringer lactate, with a switch to parenteral nutrition. For the management of hypercalcemia, the patient received continuous hydration with isotonic saline, administration of diuretics, and a corticosteroid therapy.

As part of the etiological assessment. A clinical examination was carried out, showing no adenopathy, the rectal exam was normal ganglion Troisier was absent. Biologically, PTH was normal, suggesting a tumor origin. The workup was completed by measuring PSA, ACE, CA 19-9, and AFP, which were normal. A Cerebral, thoracic, abdominal, and pelvic Computed tomography revealed stage D pancreatitis with generalized osteolytic lesions of the axial and peripheral (Figs. 1 and 2) squamous bones with thickening of the cavum (Fig. 3).

Malignant hypercalcemia secondary to nasopharyngeal neoplasia with bone metastasis was suspected. The workup was completed by serological profiling of the patient for CMV and EBV infection which came back positive. The evolution was marked by the persistence of high figures of the calcemia requiring the introduction of isphosphonates with a good clinical and biological improvement. After stabilization, the patient was referred to an oncology center for further diagnostic and therapeutic management.

Discussion

Malignant hypercalcemia is defined by a plasma calcium concentration >3.5 mmol/L (140mg/L) and/or the association of hypercalcemia (total calcemia >2.6 mmol/L) with severe manifestations. It is an uncommon electrolyte disturbance that constitutes a potentially life-threatening metabolic emergency [1].

Malignant hypercalcemia affects approximately 10%-20% of cancer patients [2]. It often complicates breast, lung and myeloma cancers, and rarely kidney, head and neck, and gynecological cancers [3].

Cavum cancer is a relatively rare cancer that mostly affects elderly subjects [4]. UCTN is the most frequent histological type. Several etiopathogenic factors have been described, including Epstein-bar virus infection [4].

Malignant hypercalcemia of tumor origin is due to excessive bone resorption responsible for the release of large amounts of calcium with an inability of the kidney to ensure phosphocalcic regulation [5]. Several mechanisms have been described, mainly tumor secretion of PTH RP (related protein), local secretion of cytokines by osteolytic metastases, excessive extra renal production of 1.25 dihydroxy vitamin D and ectopic secretion of PTH [1,6].

The clinical presentation of hypercalcemia is characterized by clinical polymorphism, and depends mainly on the extent and speed of onset [1,7]. Clinically, there are neuropsychiatric signs ranging from simple drowsiness to coma, cardiovascular signs represented by supraventricular or ventricular arrhythmias, arterial hypertension and QT segment abnormalities on electrocardiogram [7,8]. The renal system is not clinically unaffected, polyuro-polydipsic syndrome and renal failure are observed in the majority of patients with acute severe hypercalcemia, but in chronic forms, nephrocalcinosis is the main complication. The digestive system itself is affected, apart from nausea, pseudo-surgical abdominal pain, and vomiting, digestive involvement can be prognosticated by acute pancreatitis [7].

The paraclinical diagnosis of hypercalcemia requires the determination of total calcemia on 2 occasions coupled with the determination of albumin to calculate the corrected calcemia.

Malignant hypercalcemia is a very frequent complication of neoplastic pathology [9]. Multiple myeloma represents the first cause of malignant hypercalcemia of tumor origin followed by breast cancer, kidney cancer, and squamous cell carcinomas including lung cancer, esophageal cancer and tumors of the head and neck including cavum cancer [5].

The pathophysiology of acute pancreatitis in hypercalcemia remains poorly understood, but the most accepted hypothesis is obstruction of the pancreatic ducts by calcium deposits [10].

The management of malignant hypercalcemia is based on 4 main principles: correction of dehydration associated with hypercalcemia by isotonic saline according to the calcemia and the cardiac status of the patient [7]. Increasing urinary sodium excretion by loop diuretics reserved mainly for patients with acute renal failure or congestion [2]. The reduction of bone resorption is a very effective therapeutic approach and can be achieved by biphosphonates, the latter with the advantage of a long duration of action [7]. Cortico-steroids have been shown to be effective in the cases of hypercalcemia secondary to multiple myeloma or hematological diseases with hyperproduction of calcitriol [7]. Calcitonin should be used exceptionally in very severe hypercalcemia in combination with biphosphonates. Denosumab is a monoclonal antibody against RANKL. It should be used in cases

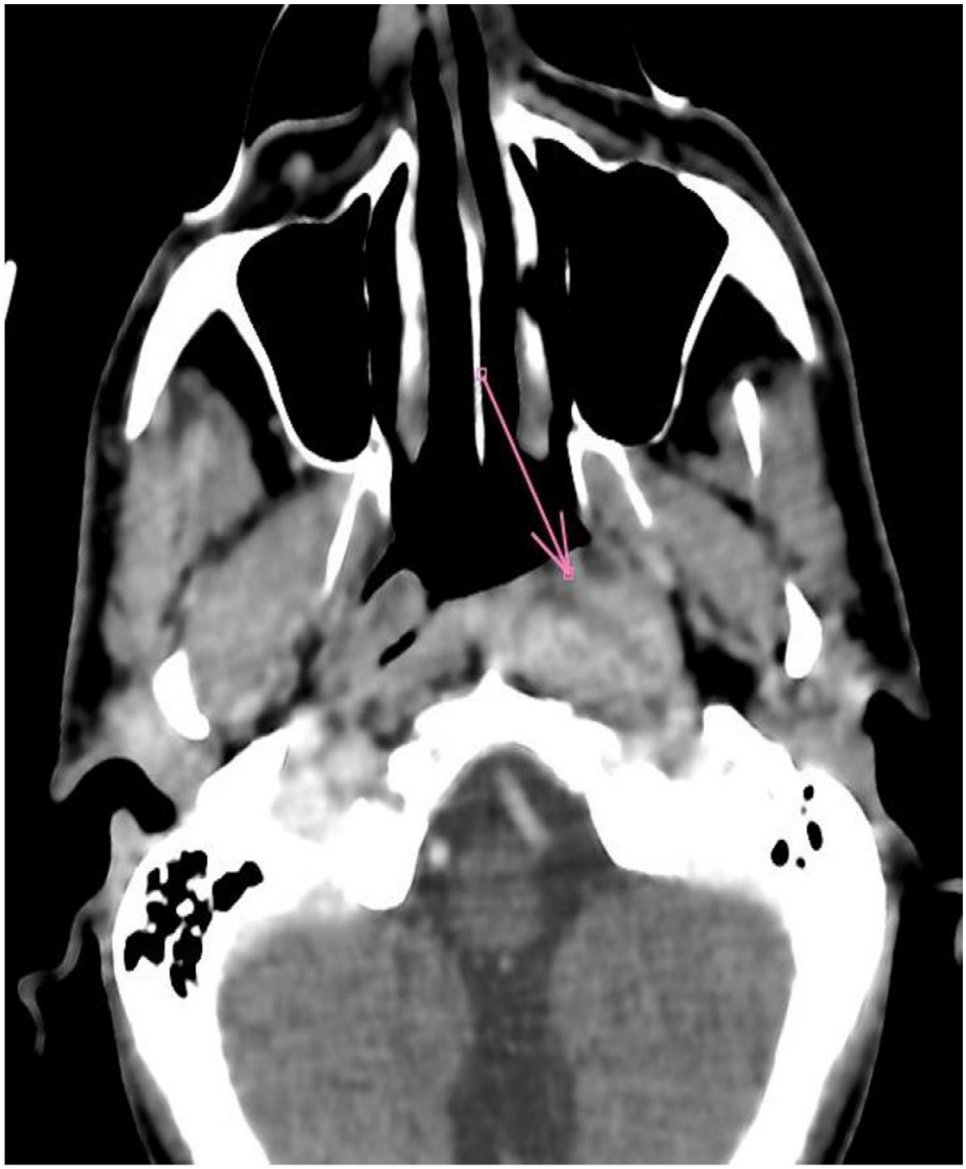

Fig. 3 – CT scan of the cavum with injection of contrast medium showing tissue thickening of the left posterolateral wall of the cavum erasing its mucosal reliefs of tumor appearance.

of recurrent tumor-induced hypercalcemia or hypercalcemia resistant to biphosphonates [2].

Finally, extra-renal purification remains the only therapeutic choice in cases of severe hypercalcemia that may be life-threatening, in cases of severe congestive heart failure, in chronic hemodialysis patients and in subjects with a GFR <10-20 mL/min [7].

The prognosis of malignant hypercalcemia remains poor. Of 75%-80% of patients with malignant hypercalcemia already have metastatic disease. Bone metastases are present in almost all patients with malignant hypercalcemia associated with breast cancer, thyroid cancer, and multiple myeloma. The median survival of patients with malignant hypercalcemia is on average 1-3 months, and is shorter with very high calcemia figures, which shows the severity of this condition [2].

Our patient already had acute pancreatitis on admission secondary to his hypercalcemia, and imaging showed generalized bone lysis more in favor of metastatic neoplastic disease, which unfortunately suggests a very poor prognosis.

Conclusion

Malignant hypercalcemia represents a real challenge for the practitioner because of its multiple complications ranging

from simple renal failure to severe acute pancreatitis and the neoplastic origin remains the main cause to be sought.

Patient consent

Obtained.

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