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Joint Deposit of Calcium Oxalate in Acute Oxalate Nephropathy: A Cause of Kidney Failure to Remember

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Abstract **Original Research Article**

Acute oxalate nephropathy, caused by either primary or secondary hyperoxaluria, is characterized by the presence of tubulointerstitial deposits of oxalate, accompanied by an interstitial inflammatory reaction. We report the case of a type 2 diabetic patient who developed terminal chronic renal failure on acute oxalate nephropathy. The patient was seen in the Emergencies Department with a loss of cutaneous substance at the level of the wrist with a whitish suppurative deposit. The radiological assessment showed various joint opacities at the level of the wrist, the metacarpophalangeal and interphalangeal joints. A non-regressive ARF (particularly in a diabetic patient) should lead to a search for exocrine pancreatic insufficiency with secondary hyperoxaluria. A kidney biopsy should be performed quickly to support this diagnostic suspicion.

Keywords: Wrist-Bone-Calcium-Nephropathy-Renal-Failure.

INTRODUCTION

Acute renal failure (ARI) is a major public health problem, the incidence of which has been increasing over the past ten years. In our country, it primarily affects the elderly, with multiple comorbidities and taking nephrotoxic medications. In addition to the usual etiologies (cardiac, drug and infectious), a new entity is described more and more often in the medical literature, acute oxalate nephropathy (AON), tubulo-interstitial damage linked to diffuse deposits of calcium oxalates. These are due to hyperoxaluria either primary (hereditary and linked to enzymatic defects of glycosylate metabolism), or secondary (increased intestinal absorption of oxalates), oxalates having mainly a renal elimination. An increase in the urinary elimination of oxalates (hyperoxaluria) will therefore lead to an oversaturation of the ultrafiltrate in oxalates and to the formation of crystals of calcium oxalates. This hyperoxaluria can be complicated by episodes of calcium oxalate urolithiasis or by diffuse calcium oxalate deposits with concomitant tubulointerstitial inflammation.

Calcium oxalate crystals cause severe pain and inflammation in and around joints. The crystals sometimes destroy the joints.

MATERIEL AND METHODS

Our work concerns the case of a 48-year-old patient, A.J, a chronic hemodialysis patient at the rate of 3 sessions per week, who consulted in the emergency department of the Ibn Sina Hospital for the appearance of a loss of cutaneous substance at the level of the wrists. The patient was then admitted and underwent a certain number of trimmings and bacteriological samples. The biochemical examination of the samples were in favor of calcium oxalate crystals. The patient also had cardiac symptoms and explorations revealed moderate pericarditis and mitral valve disease, which was subsequently treated by cardiovascular surgeons.

Figure 1: Clinical image showing the joint suppuration

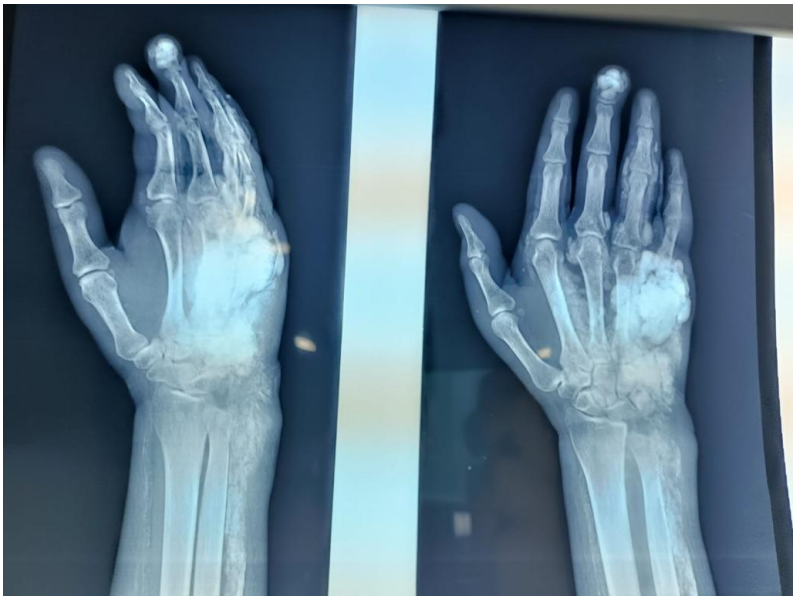


Figure 2: X-rays showing calcium deposits in the joints

RESULTS

Well-defined calcifications are discovered radiologically in our subject. They are most often located at the shoulder and at the level of the wrist. A number of these calcifications are perfectly tolerated clinically.

In the event of a single calcification, traumatic etiological elements are often found (professional and sports factors), so that even young subjects can be affected. One of the hypotheses favors an origin by a local hypoxia which would allow the transformation of the tendon into fibrocartilage on which would come to form deposits of apatite or crystals of calcium oxalate. Phagocytosis by macrophages of these microcrystals would be the basis of the inflammatory crisis.

The apatitic calcifications, if they are generalized, can be the expression of the metabolic disorders where one observes an increase in the calcic product, in particular during the end-stage chronic renal failure, in hemodialysis patients, during primary hyperparathyroidism, in case of hypervitaminosis D and in the milk-alkaline syndrome. They can also be the expression of the disease of multiple tendon calcifications. Genetic factors are sometimes highlighted.

In extensive calcinosis, qualified as tumoral, whether idiopathic or generalized, there is generally a slight hyperphosphatemia.

In diabetes mellitus, the frequency of shoulder calcifications rises to 22%. Some cases recognize an iatrogenic cause. This is how capsular or tendon calcifications can follow injections of long-acting corticosteroids. It has also been described in intervertebral discs after nucleortheses for herniated discs with triamcinolone hexacetonide; these calcifications were often the source of a recurrence of

painful symptoms and sometimes even of erosive disc disease.

Acute oxalate nephropathy should be sought in any acute renal failure of undetermined origin and rapidly progressive installation, particularly in a patient known to have diabetes. The urine sediment may show leukocyturia, epithelial cells (acute tubular necrosis) and/or oxalate crystals. These can be seen in two forms, the most common being calcium oxalate di-hydrate, the other form being calcium oxalate monohydrate. Urinary sediment may also be normal. Conversely, the presence of oxalate crystals does not necessarily mean that it is not oxalate nephropathy.

The purpose of collecting 24-hour urine by quantifying the flow of oxalates is to look for hyperoxaluria.

The diagnosis of acute oxalate nephropathy is obtained by renal puncture-biopsy, which reveals tubulointerstitial nephritis with the presence of crystals within the lumen of the tubules, in the tubular epithelial cells and/or in the interstice.

This diagnosis was highlighted in our case by the nephrologists after the patient was referred to them. This is how this calcium deposit at the articular level was for our patient the revealing element of the causal pathology and of the systemic attack.

Thereafter, after normalization of his cutaneous condition and once the suppuration was healed, the patient was able to benefit from a skin graft at the level of his loss of substance.

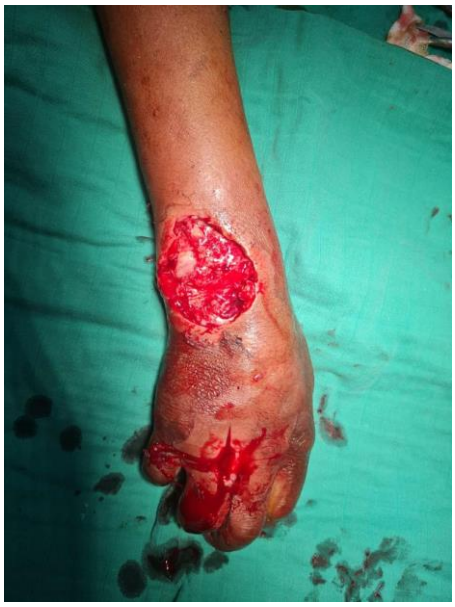


Figure 3: Clinical picture after cleaning the suppuration

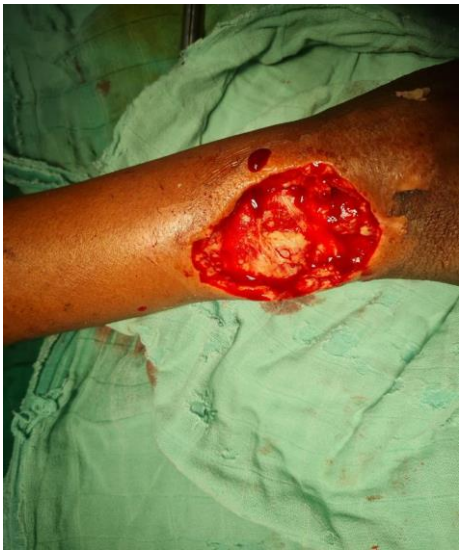


Figure 4: Clinical picture after cleaning the suppuration

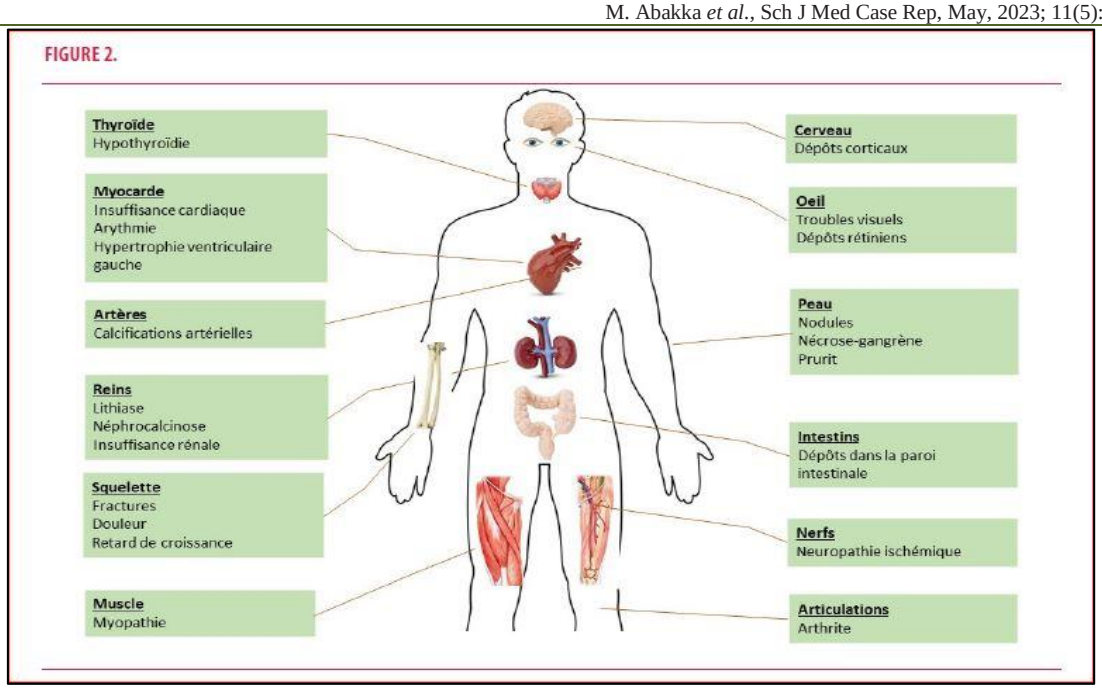


Figure 5: Symptoms of the primary hyperoxaluria

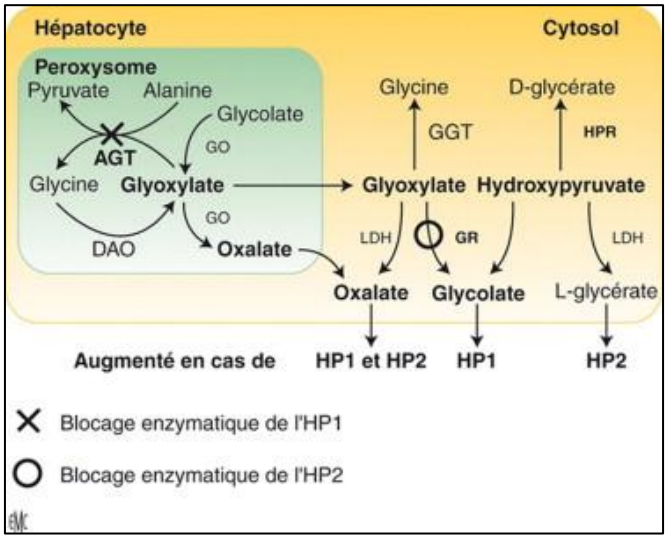
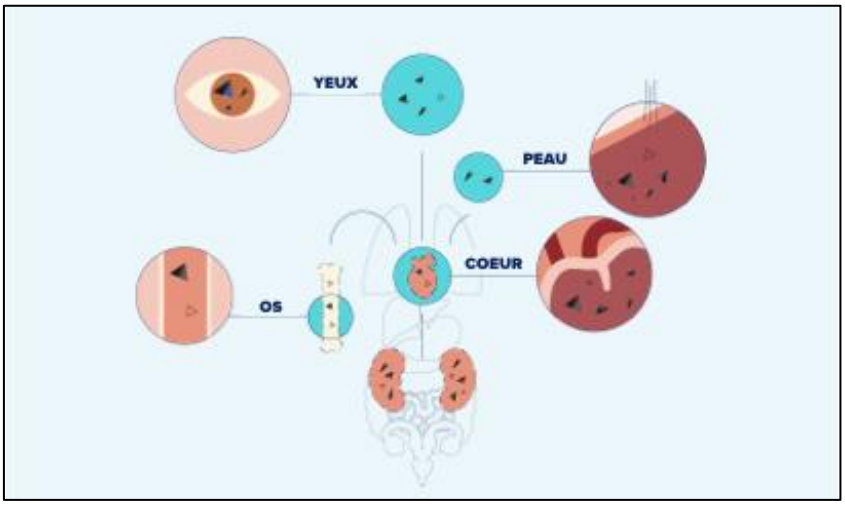


Figure 6: Primary hyperoxaluria physiopathology



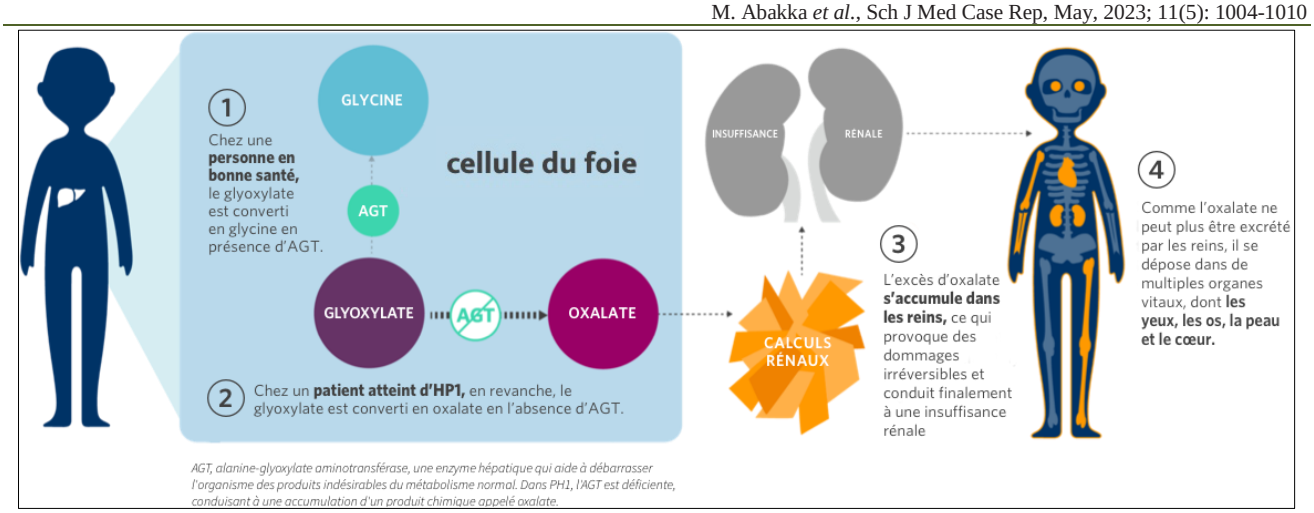
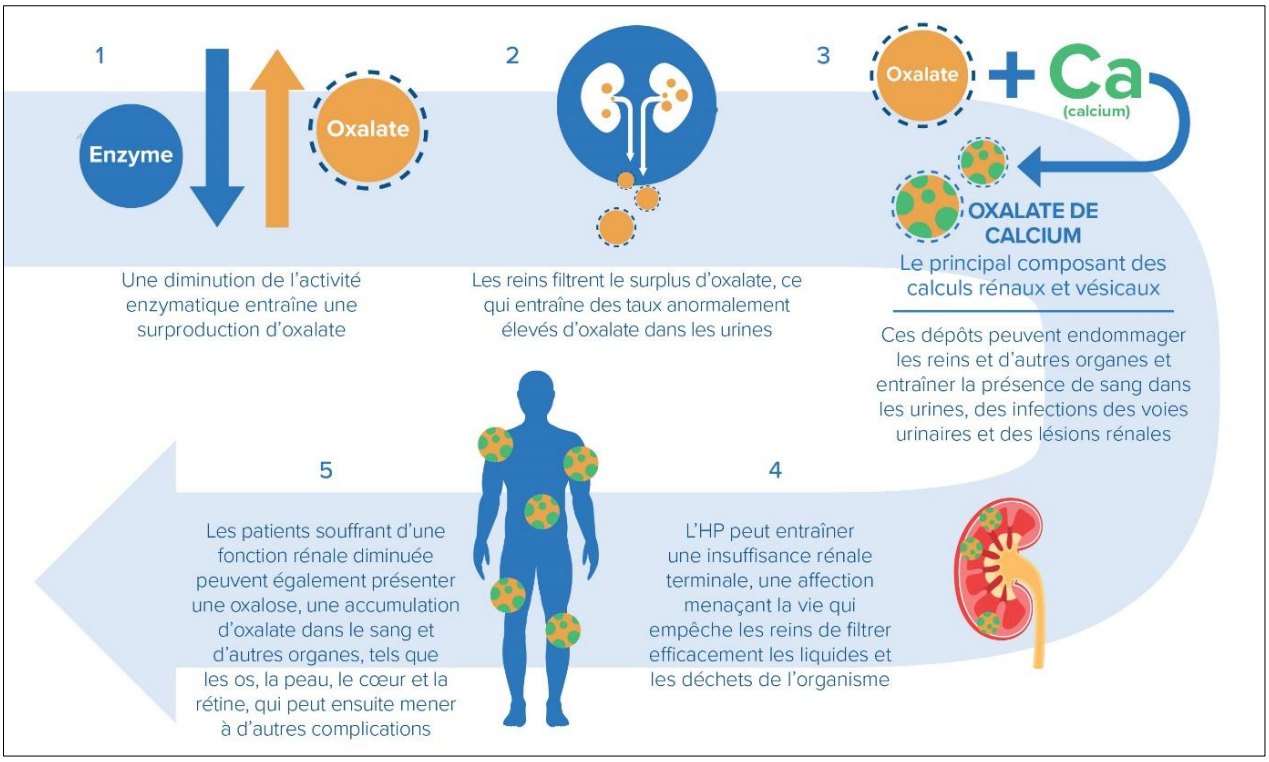


Figure 7: Other diagram explaining the physiopathology of Primary hyperoxaluria



DISCUSSION

Oxalate nephropathy, which is an acute renal failure, with a rapidly unfavorable evolution, originates from hyperoxaluria. Primary hyperoxaluria, which is related to an inherited deficiency of liver enzymes responsible for glycosylate metabolism leading to endogenous overproduction of oxalic acids and hyperoxaluria, has been the subject of an excellent review article published recently.

Secondary hyperoxaluria, which results in turn from an increase in the intestinal absorption of oxalates, can be observed in the following situations: diet rich in oxalates and malabsorption of fatty acids occurring in chronic pancreatic exocrine insufficiency, in inflammatory bowel disease or in short bowel syndrome after bowel surgery.

Oxalate is an organic salt present in many foods (cocoa, tea, spinach, rhubarb). Once ingested, the oxalate is either absorbed in the form of a free ion at the apical membrane of the enterocytes of the colon then filtered, secreted and eliminated via the kidneys, or it complexes with the calcium present in the intestinal lumen, forming then insoluble and poorly absorbable complexes. The intestinal absorption of oxalates is therefore influenced by the concentration of calcium and magnesium in the intestinal lumen, by the innate absorption capacities of the colonic mucosa, by gastric emptying and transit. The Western population ingests between 80 and 120 mg/24 h of oxalates, of which

approximately 10% is absorbed. Absorption greater than 15% is considered hyperactive absorption. Thus, a high intake of oxalates can occasionally be complicated by hyperoxaluria and oxalate nephropathy.

In pathologies inducing fatty acid malabsorption, two factors contribute to an increase in the enteric absorption of oxalates and therefore to hyperoxaluria. Fatty acids form complexes with calcium in the intestinal lumen, the increase in these leads to a decrease in calcium available to complex with oxalate and therefore to an increase in oxalate in the form of absorbable free ion . In addition, fatty acids and bile salts increase the permeability of the intestinal mucosa to oxalate.

Thus, since the beginnings of the treatment of morbid obesity by bariatric surgery (jejuno-ileal bypass introduced in the 1950s), enteric hyperoxaluria, and secondary to the reduction in the intestinal absorption surface of fatty acids, has been described. The change in operative technique with the replacement of the jejuno-ileal bypass by the Roux Y gastric bypass has only slightly modified the prevalence of oxalate nephrolithiasis and OAN in these patients.

In addition, exocrine pancreatic insufficiency, characterized by a lack of pancreatic enzyme production, induces poor digestion of food and is clinically manifested by steatorrhea (accumulation of fatty acids in the intestinal lumen). The diagnosis of chronic pancreatitis is clinical with weight loss and steatorrhea; biological with a decrease in pancreatic elastase in the stool; radiological with the demonstration of pancreatic calcifications and histological with the appearance of progressive fibrosis of the pancreatic glandular parenchyma.

An oxalate nephropathy can thus occur in patients known for chronic pancreatitis or can reveal this pathology, as demonstrated by a retrospective study, published in 2011, analyzing twelve patients with oxalate nephropathy. Eight patients were already known to have pancreatic insufficiency but in the other four, pancreatic insufficiency was diagnosed after the onset of OAN. Pancreatic elastase was lowered in five patients. Imaging showed pancreatic calcifications in ten patients and pancreatic atrophy in four patients. The existence of type 2 diabetes therefore seems to be a factor favoring the appearance of ON, in a context of associated exocrine pancreatic insufficiency.

The treatment of this disease is primarily based on the treatment of hyperoxaluria and its cause. It consists first in reducing the quantity of absorbable oxalates through a diet low in oxalates and fatty acids and rich in calcium. Phosphate binders, commonly used in patients with CKD, such as lanthanum carbonate, have been shown to significantly decrease hyperoxaluria in animal models. Decreasing calcium oxalate stone precipitation can be achieved by rehydration and normalization of magnesium levels. In case of exocrine pancreatic insufficiency, enzyme substitution should be started. Pyridoxine, used with modest success in primary hyperoxaluria type 1, has no place in the treatment of secondary hyperoxaluria. For patients who have had bariatric surgery and suffer from hyperoxaluria complicated by ACF, the possibility of restoring digestive continuity can be considered in certain cases. Clinical trials to determine the efficacy of probiotics containing Oxalobacter formigenes in patients with primary hyperoxaluria have so far not yielded conclusive results.

The prognosis for ON is poor if hyperoxaluria is not effectively treated, with frequent progression to end-stage renal disease requiring chronic dialysis, as illustrated by our findings presented here and the largest case series reported in the literature.

CONCLUSION

PHO is a rare disease, but probably under-diagnosed and whose renal prognosis is reserved. In secondary hyperoxaluria and in the absence of an obvious short bowel syndrome, exocrine pancreatic insufficiency should be sought. In the event of non-reversible ARI and in particular in type 2 diabetic patients, a PBR must be quickly carried out. The treatment of hyperoxaluria must be done as soon as possible in order to slow the progression to end-stage renal failure.

In our case, the articular deposit of calcium oxalate was the main revealing sign, unfortunately late diagnosis because the patient was already at the stage of terminal chronic renal failure and dialysis.

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