Bone Marrow Oxalosis : Case Report

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Abstract:
Hyperoxaluria is characterized by supersaturation of calcium oxalate in the urine, and is strongly associated with nephrolithiasis and nephrocalcinosis. Long-standing and unattended hyperoxaluria can progressively impair renal functions, and ultimately lead to renal failure. The devastating phenomenon is characterized by oxalosis, a condition in which calcium oxalate crystals are deposited in the extrarenal organs. [1] The common sites of oxalate deposition are the bones, bone marrow, blood vessels, central nervous system, peripheral nerves, retina, skin, and thyroid, etc. [2] Calcium oxalate has a very low solubility leading to nephrocalcinosis, urolithiasis and progressive renal insufficiency. [3] Here we present a case report of a 29-year-old female in whom a diagnosis of oxalosis was made on bone marrow biopsy.

Keywords: Bone Marrow, Oxalosis

Case Report: A 29-year-old female patient presented with history of vomiting since 15 days. Her relevant investigations were done. The complete blood count was done on fully automated haematology analyser showed haemoglobin level of 6.2gm/dl; white blood cell count of 2.5×10^9/l and platelet count of 90×10^9/l. Biochemical tests showed raised blood urea (250mg/dl) and serum creatinine (6.8mg%). X-Rays KUB showed presence of multiple stones in both the kidneys. Bone marrow studies were advised to evaluate pancytopenia. Bone marrow aspiration was a dry tap. Contact smears showed poor cellularity. The trephine biopsy showed replacement of the marrow by variable sized aggregates of grey-yellow crystals of oxalate. Conditions associated with calcium oxalate crystal deposition include primary and familial hyperoxaluria, a diet rich in oxalate, increased absorption or production of oxalate, and decreased excretion of oxalate as seen in renal failure. [4] Most patients with hyperoxaluria present with renal calculi at an early age. Our patient presented at 39 years age with bilateral nephrolithiasis, renal failure and oxalate deposition in the bone marrow. She had developed renal failure owing to obstructive uropathy. Her trephine biopsy showed replacement of the marrow by variable sized aggregates of grey-yellow crystals of oxalate.

Discussion:
Our patient was a 29-year-old female who presented with vomiting. She had pancytopenia, nephrolithiasis, renal failure and oxalate deposition in the bone marrow. She had developed renal failure owing to obstructive uropathy. Her trephine biopsy showed replacement of the marrow by variable sized aggregates of grey-yellow crystals of oxalate. Singh et al reported a case of primary hyperoxaluria in a 19-year-old female in which the diagnosis was not entertained because there was no past history of urinary calculi and no evidence of nephrocalcinosis on plain X-ray and ultrasonography. The disease was detected and
diagnosed only after kidney transplantation. [3] Kakkar et al reported bone marrow involvement in systemic oxalosis in a 30-year old woman. Variable degree of cytopenia or pancytopenia may accompany extensive oxalosis. Bone marrow aspirate may be unsuccessful due to extensive crystal deposition or accompanying fibrosis. Renal function tests are often deranged in patients with accompanying renal failure. [6] Hricik DE stated that mechanical obliteration of bone marrow by calcium oxalate crystals may lead to pancytopenia and hepatosplenomegaly as late extrarenal complications of oxalosis. [6]

Systemic oxalosis can be primary or secondary. Primary hyperoxaluria Type 1 (PH 1) is due to the deficiency of glyoxylate aminotransferase and Type II due to that of glyoxylate reductase/ D-glycerate dehydrogenase. PH 1 is an autosomal recessive disorder characterized by hyperoxaluria, calcium oxalate urinary lithiasis in childhood, nephrocalcinosis and renal failure which in turn leads to high blood oxalate levels and precipitation occurs throughout the body in the skin, blood vessels and joints. Secondary oxalosis occurs due to oxalate-rich diet, increased absorption or production of oxalate and reduced excretion as seen in renal failure. Early stage of oxalosis is asymptomatic and diagnosis is often delayed and sometimes made incidentally. [7] Although patients maintained on haemodialysis have previously been noted to have oxalate deposition in the bone marrow, it is rare for the diagnosis of oxalosis to be made by bone marrow biopsy. [8]

References:


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