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A Case Report of Stroke in Secondary Hyperoxaluria

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Abstract

Hyperoxaluria is most common in developed countries like the USA, but not sporadic in developing countries. Hyperoxaluria is a state, that occurs when a patient has an excess of oxalate formation in urine. hyperoxaluria is of two types- Primary hyperoxaluria is due to an inherited defect of alanine glyoxalate transaminase. Secondary hyperoxaluria is due to increased GIT absorption of oxalates. In hyperoxaluria, there are increased oxalate levels in the blood, which get accumulate in joint spaces, tissues, and bone marrow resulting in systemic oxalosis. The conjunction of hyperoxaluria and CNS ischemia stroke is unusual

Here we are discussing a case of 71 years old male patient with a known case of Crohn's disease for 20 years, with a history of small bowel resection 6 years back, with an intact colon, post-operatively not on regular medications. History of recurrent renal stones, history of insomnia, presented with CNS ischemic stroke

In this patient, there is an increased chance of secondary hyperoxaluria, due to raised absorbency of the large intestine to oxalates, which also increases blood oxalates levels, and there is a surge in accumulation of free oxalates in the large intestine lumen, due to fatty acids combined with luminal calcium. In patients with secondary hyper-oxalosis, that circulating oxalate precipitate could be a risk factor for stroke

1. Introduction

CASE HISTORY

71 years old male patient with K/C/O Crohn's disease with a history of small bowel resection(duodeno-jejunostomy), 6 years back, with H/O recurrent renal stones, H/O multiple times of extracorporeal shock wave lithotripsy, H/O insomnia, presented to casualty with C/O drowsiness, disoriented to time place and person, slurring of speech, hemiparesis in the right side of the body.

MRI showed infarctions in the left frontoparietal lobe, in the territory of the left middle cerebral artery. MRA showed the left distal M1 segment was abruptly terminated, indicating occlusion. Transcranial doppler showed circulating micro-emboli at the rate of 11 per hour. ABG showed metabolic acidosis

An evaluation revealed no signs of pathogenic, rheumatologic, or hypercoagulative conditions. Other investigations like hypercoagulative panel, transthoracic echocardiogram, and trans-esophageal echocardiogram were normal. Total count elevated 20,100cells/cumm, hemoglobin 6.5mg/dl, Serum calcium was low(42mg/dl) and serum oxalate levels were greater(138mg/dl) than normal, which is a possible microemboli source, 24 hours of urine calcium and oxalates were elevated, serum creatinine 1.9mg/dl, serum Urea 51mg/dl and kidney

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stone analysis showed calcium oxalate stones. CT KUB showed cortical nephrocalcinosis. The liver function test showed elevated alkaline phosphatase(ALP) levels, indicating osteoporosis. The fecal occult blood test was positive. ileoColonoscopy showed small erosions and loss of mucosal architecture on the terminal ileum

The patient was given initially a loading dose of clopidogrel 300mg, aspirin 300mg, atorvastatin 80mg, resuscitated with iv fluids and injection citicoline 1gm and injection piracetam 1gm and hemoglobin is corrected to 9.5mg/dl by 3 units blood transfusion, mucosal ulcerations are treated with antacids and started mesalamine 4 grams daily

The patient was discharged with Aspirin 75mg, Atorvastatin 20mg, Clopidogrel 75 mg, and Sodium citrate for urinary alkalization, Calcium supplementation, ibandronic acid(bisphosphonate) and Oxalate binding agents like sevelamer hydrochloride, mesalamine 4 grams daily for 8 weeks, the patient recovered well, with mild right side hemiparesis. The patient was advised to stop the dietary intake of food containing oxalates and to take plenty of oral fluids to prevent renal stone formations

2. Discussion

NORMAL OXALATE METABOLISM - The final product of the breakdown of amino acids is oxalate, which is created endogenously. Dietary sources of oxalate are also absorbed by the stomach, small intestine, and colon[10]. There is still some controversy around the precise process of oxalate absorption in the intestine[11]. Up to 50% of urinary oxalate in healthy people comes from dietary sources. Oxalate can be eliminated through excretion, urine dissolution, calcium precipitation in the stool, or gut microbial metabolism[10]. Under typical circumstances, all oxalate produced endogenously and ingested is eliminated in the urine. Although the amount of oxalate normally excreted by the urine varies, hyperoxaluria is defined as excretion exceeding 30–45 mg/day (0.40 mmol).

PATHOPHYSIOLOGY- Secondary hyperoxaluria is induced by an elevation of oxalate solubility in the gut lumen and a corresponding rise in intestinal permeability to oxalate brought on by bile salts and inflamed colonic mucus, as seen in inflammatory bowel disorder[9]. In healthy individuals, Dietary oxalate and calcium combine to form the insoluble compound Calcium oxalate, which is eliminated in the stool. Non-absorbed fatty acids instead bind calcium in the small intestine in secondary hyperoxaluria, preventing it from precipitating oxalate. As a result, soluble oxalate is available in the lumen at comparatively high levels and can passively diffuse out of the colon further into the blood, where it can then be eliminated by the kidneys. Additionally, the colon plays a part in the aetiology of hyperoxaluria. One series found that whereas 7 of 10 patients with steatorrhea and intact colons had hyperoxaluria, none of the five patients with steatorrhea and ileo-Jejunostomies reported. According to this research, the colon is the primary location of intestinal oxalate absorption.[9]

3. Conclusion

Secondary hyperoxaluria, in this case, is caused due to enhanced oxalate absorption in the intestine owing to increased oxalate permeability and the production of fatty acid and calcium complexes, resulting in greater levels of soluble oxalate. This process requires an intact colon to promote oxalate absorption. Partial bowel resection, bariatric procedures, jejuno-ileal bypass, Crohn's disease, and ulcerative colitis are associated with secondary hyperoxaluria[1]

Other causes of secondary hyperoxaluria like -

Increased dietary oxalates like spinach, legumes, potatoes, and rhubarb[2]. The bioavailability of oxalate from food and, consequently, urinary oxalate are influenced by the different forms of oxalate in food, food processing, cooking methods, and other elements in the meal. Oxalate intake in the diet is decreased by co-administration of calcium or magnesium, which bind with oxalate to form insoluble salts.[3,4]

Oxalobacter formigenes is an aerobic gram-negative bacteria that use oxalate as a source of energy and hence inhibits oxalate absorption in the intestine and consequently urinary oxalate excretion. After using antibiotics, this bacterium is lost, and its

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restoration could help with the treatment of hyperoxaluria.[5,6]

Increased consumption of oxalate precursors - Considering ascorbic acid is an oxalate precursor, consuming too much of it might cause calcium oxalate to precipitate. Ethylene glycol produces oxalate, which causes calcium oxalate deposits and renal failure. Owing to oxalate mobilization and deposition within the renal allograft, hyperoxaluria has also been described after renal transplantation. Patients with cystic fibrosis have been observed to have elevated oxalate absorption and tubular secretion, resulting in hyperoxaluria.[7,8]

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