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A Rare Cause of Iron Deficiency Anemia in Patient with Ankylosing Spondylosis

Abstract

Metal on metal implants due to the wear-tear phenomenon tend to release the metals causing its toxicity. Most commonly seen with metals like cobalt, chromium, and so on. The metallic deposition in the surrounding soft tissue then enters the systemic circulation causing the effects. Cobalt Metallosis usually has clinical features of cardiomyopathy, polyneuropathy, bilateral optic neuropathy, bilateral sensorineural hearing loss, thyroid disorders, and polycythemia. Here is a case of a young male present with Iron Deficiency Anemia.

Keywords: Metallosis; Toxicity; Cobalt; Implants

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Introduction

Hematological manifestations of Cobalt toxicity were Polycythemia, Leucopenia, and Thrombocytosis. Polycythemia is usually seen in patients who receive cobalt chloride tablets orally which induces HIF-1 alpha leading to increased erythropoietin levels. With chronic exposure can cobalt rarely leads to decreased expression of Divalent Metal Transporter 1 receptors in the duodenum. This causes decreased absorption of iron from the GI tract and the patient presents with Iron Deficiency Anemia.

Case Summary

A 34year old gentleman with no previous co-morbidities presented with complaints of difficulty in walking for 1 year wherein the patient was unable to walk in a straight line without support with his body swaying to the left side and pain in the left hip and associated with numbness and tingling sensation in both lower limbs. The patient had dyspnoea on exertion for 4 months of NYHA grade 3 and developed shortness of breath walking on approx. 100 meters and associated with swelling of lower limbs. The patient also complained of decreased sleep duration and decreased interaction with family members including his wife and child.

The patient was diagnosed with HLA B-27 positive Ankylosing spondylitis 20yrs back and stopped all the medication for 3 years. The patient had undergone both hip replacement surgery right side (2009) and left side (2011).

On examination, he looked severely pale with bilateral pitting

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edema. Vitals were stable with slight tachycardia of 108/min and the rest of the systemic examination was normal.

Hemogram showed HB-4.4g/dl, leukocytes count-4600 platelet count-2.5 lakh mcv-70 and absolute reticulocyte count of 0.2%. With peripheral smear showed microcytic hypochromic anemia. Kidney and liver function tests were normal. Total protein was 4.3g/dl with albumin 2.6g/dl. Iron profile was iron-13, UIBC-294 TIBC-497, and saturation-4.7%. Serum LDH being 183. Iron deficiency anemia was diagnosed.

2D Echo was with normal chamber dimensions and ejection fraction of 55%. USG and CECT Abdomen were within normal limits.

On further evaluation for iron deficiency anemia CECT abdomen and upper GI endoscopy were done which was normal. Anti TTG was 0.6 which was in the normal reference range (**Figures 1 and 2**).

A nerve conduction study showed reduced nerve conduction in the left nerve with lower amplitude values in the left peroneal and tibial nerves. No response was recorded in the bilateral sural nerve suggestive of sensorimotor involvement of lower limbs and upper limbs.

With Iron Deficiency Anemia, polyneuropathy, and psychiatry manifestation, the patient was planned for toxic metal screening



Figure 1 Xray pelvis showing B/L hip implants with displaced left implant.



where serum cobalt and cadmium levels were 6.34microg/L and <2.5microg/L respectively. Urine Cobalt levels were 29.92microg/L.

X-Ray of the Pelvis showed a misaligned left hip implant and an orthopedic surgeon posted the patient for hip implant replacement and reconstruction.

Intra Operative we could see extensive wear and tear of left hip implant and deposition of metals in the soft tissue around the joint.

Postoperative day 28 serum and urinary cobalt levels were 3.99microg/L and 18.27microg/L and hemogram showed HB-10.5 TLC-5600 and platelets-2.5lakh iron profile was Iron-102, TIBC-263 UIBC-161 sat-39.

Discussion

Prosthetic hip-associated cobalt toxicity has been seen with various systemic manifestations commonly being neurological symptoms such as polyneuropathy, optic nerve atrophy, and sensorineural hearing loss. Mechanism which has proposed is disruption of mitochondrial oxidative phosphorylation, neurotransmitter modulation, and direct neuron cytotoxicity [1].

Other systemic manifestations include hypothyroidism, cardiomyopathy leading to complications like Atrial Fibrillation and Flutter, psychiatric manifestations like depression, constitutional symptoms of fever, irritability, anorexia, fatigue, and weight loss. There was no clear correlation seen between serum cobalt levels and their clinical presentation [2].

In the natural course of the disease, thyroid dysfunction, sensorineural deafness, vertigo, and cardiomyopathy improved with improvement in ejection fraction once cobalt levels became <5microg/L after removal of the default implant. But the polyneuropathy and optic nerve atrophy didn't show signs of recovering [3].

In Haematological manifestation, patient with cobalt toxicity by oral intake of cobalt chloride tablets is transiently seen with polycythemia caused by increased levels of hypoxic inducible factor 1-alpha. Once iron stores are exhausted then it leads to Iron deficiency anemia. Patients with Iron deficiency anemia showed increased mean urinary cobalt levels [4].

The effect of cobalt on bone was shown to be at the level of osteoclast. Osteoclasts were easily stimulated even at lower toxic serum levels of cobalt leading to brittle bone and easy fractures [5]. Divalent metal ion receptors present in the duodenum helping in the absorption of various divalent metals like iron, zinc, cobalt, lead and chromium are downregulated in presence of high cobalt levels leading decreased absorption of various divalent metal ions [6].

The average failure rate of Metal-on-Metal prosthesis at seven years is 11.8% for resurfacing and for total hip replacement was seen at 13.6% which is higher than the acceptable minimum [7]. Failure rate of 49% was seen with Depuy ASR hip implants at six years interval [8].

Conclusion

Prothesis - associated metallosis mostly commonly seen due to wear and tear of the implant. It has varied presentations one of which is iron deficiency anemia. Cobalt toxicity has clinical features of cardiomyopathy, polyneuropathy, bilateral optic neuropathy, bilateral sensorineural hearing loss, and thyroid disorders. Cobalt metallosis can be suspected in patients with implants refractory to oral iron medications and an iron-rich diet. If a patient has high levels of cobalt in serum and urine this means patient implants have wear and tear with soft tissue deposits. The only cure is to replace this implant with other safer implants.

Conflict of Interest

There was no conflict of interest during this case study4.

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