Sweet Syndrome With Sterile Non - Bacterial Spinal Osteomyelitis

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INTRODUCTION:

Sweet's syndrome is a rare condition characterized by fever, leukocytosis, dense dermal infiltration of leukocytes, and painful indurated cutaneous plaques. Clinical data elaborating its relationship with sterile spinal osteomyelitis is relatively uncommon. We describe a case with underlying Sweet syndrome complicated with spinal osteomyelitis.

CASE

A 48 year old female underlying Sweet Syndrome presented with severe upper back pain with paraperesis. Imaging revealed T5 osteomyelitis with kyphosis. She underwent a T5 vertebrectomy and T3 to T7 posterior spinal fusion. Intra-operatively, there was pus and evidence of T5 osteomyelitis.

Histophatological examination of the T5 bone revealed acute on chronic suppurative osteomyelitis. Bone, tissue, blood and swab cultures revealed no growth after 2 weeks of incubation.

She regained full power postoperatively but she developed surgical site infection requiring surgical debridement. She was put on negative wound pressure therapy thereafter and her wound healed after 3 months.

DISCUSSION:

Chronic non bacterial osteomyelitis (CNO) or chronic recurrent multifocal oesteomyelitis (CRMO) have been described by Ferguson et al[1] and its rare association with systemic inflammatory syndromes such as Sweet Syndrome have been documented. Cultures of blood, bone biopsy are usually negative, even with polymerase chain reaction (PCR) analysis in CRMO[1]. There was an urgency to surgically decompress the abcess in this case as she presented with acute paraperesis. However, Girschick et al in a study conducted in children revealed the benefits of conservative management that outweighed the surgical treatment[2]. Conservative management included non-steroidal anti-inflammatory drugs, steroids and bisphosphonates.

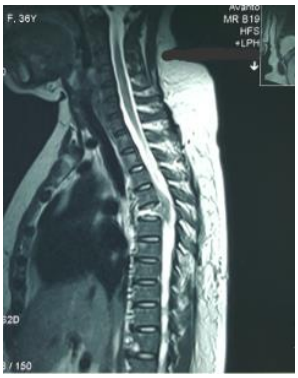

Fig 1 MRI spine : T5 osteomyeltis with collapse and cord compression

CONCLUSION:

Sweet syndrome with osteomyelitis are rarely related and erroneous suspicion of the microbial etiology of the lesion may result in unnecessary treatment not influencing the course of the disease. Conservative management should be commenced first rather than surgical treatment. However, in the presence of acute neurological deficit a more invasive treatment may be necessary to prevent the irreversible neurological complications.

REFERENCES:

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