CASE REPORT

Septic arthritis of temporomandibular joint

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SUMMARY

Septic arthritis (SA) occurrence for temporomandibular joint (TMJ) is rare. Pain, fever, swelling or loss of TMJ function are the typical presentation. The more common diagnosis for these presentations is internal derangement, osteoarthritis and rheumatoid arthritis. Therefore, TMJ septic arthritis is a challenging diagnosis and at risk of delayed diagnosis. We present a case of TMJ septic arthritis in a 46 year old Malay female with underlying hypertension and hypercholestrolemia, which was diagnosed as internal derangement in the initial presentation. The initial radiograph was normal. Arthrocentesis procedure had temporarily relieved the symptoms before progressive facial swelling developed after a week. Contrast enhanced computed tomography (CECT) brain revealed left TMJ abscess formation with left condylar erosion. Patient subsequently improved after wound debridement, left condylectomy and antimicrobial therapy.

INTRODUCTION

Septic arthritis (SA) occurrence for temporomandibular joint (TMJ) is rare.[1] Patient usually presents with pain, fever, swelling or loss of TMJ function. The established aetiology is haematogenous dissemination or local spread. They are mostly caused by *Staphylococcus aureus, Streptococcus* and *Pseudomonas aeruginosa*.[1] Typically, patient comes with risk factors either trauma, systemic or autoimmune diseases, for example rheumatoid arthritis, diabetes, systemic lupus erythematosus or hypogammaglobulinemia.[2]

SA in the joints has a mortality rate of 12%. 75% of survivors have significantly reduced function of the affected joint. SA of the TMJ is known to result in significant morbidity if the diagnosis is delayed.[3] We want to highlight the importance of prompt TMJ septic arthritis diagnosis to avoid the complications.

CASE REPORT

A 46 year old Malay female with underlying hypertension and hypercholesterolemia presented with limited mouth opening for one month associated with pain and facial swelling. Patient had no fever or history of trauma. White blood count (WBC) was not elevated: 8.0x10^3/uL. Orthopantomogram showed subtle left TMJ joint widening only notice retrospectively (Figure 1). Symptoms persisted despite taking analgesia for two weeks. Arthrocentesis of left TMJ was done for closed lock of left TMJ (internal derangement) with the improvement of interincisal distance from 21mm (preoperative) to 36 mm (post-operative). The patient was well for 8 days before experiencing the same symptoms for one week. Progressive swelling noted from left TMJ region to left infraorbital region. It was warm, tender and firm. Yellowish discharge oozed from the inner part of the cheek. Mouth opening was 15mm. She was afebrile, WBC 10.1x10^3/uL and C-reactive protein (CRP):119 mg/L. Initial diagnosis was left parotid abscess. Contrast-enhanced computed tomography (CECT) brain noted collection around the left condylar process associated with erosion and left condylar process subluxation (Figure 2). Intraoperatively, there was pus discharge with inflamed capsule and left condylar head erosion. Wound debridement and condylectomy of left mandibular condyle were done (Figure 2). Histopathology examination (HPE) report was consistent with inflammation. Pus and tissue culture showed no growth. Patient was on IV ceftazidime 1g TDS for 16 days and IV metronidazole 500 mg TDS for ten days. Upon discharge, Tablet ciprofloxacin 500mg BD was given for 14 days. Patient was symptoms free after the procedure. No recurrence reported after six month follow up.

DISCUSSION

SA is a medical emergency due to its irreversible complication. TMJ septic arthritis leads to joint destruction and subsequent bony ankyloses and fibrosis.[3] In our patient, the most likely cause of delayed TMJ septic arthritis diagnosis is subclinical infection. Patient has no risk factor or fever, and serial white blood count was normal, despite having symptoms for one-month duration. On top of that, the initial radiograph showed a normal joint space, except for subtle widening at the left lateral aspect. The occult infection worsened after the arthrocentesis, which could be due to local spread to neighbouring soft tissue resulting from the capsular breach.

The established aetiology of TMJ septic arthritis is haematogenous or local spread. The haematogenous spread from the distant primary is more common.[3] The elderly and children less than three years old are most commonly involved.[2] The common risk factors in an adult include older age group, diabetes mellitus, rheumatoid arthritis, immunosuppression, skin infection, penetrating trauma and

This article was accepted: 10 January 2020
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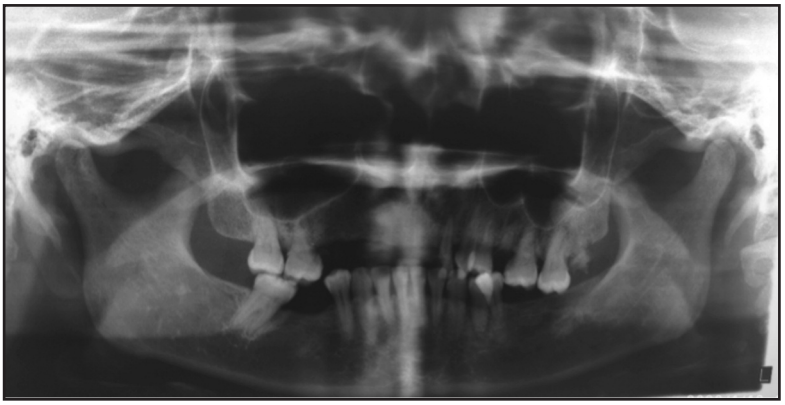

Fig. 1: Orthopantomogram done during admission shows no obvious findings except for subtle widening of left TMJ at lateral aspect (black arrow) notice retrospectively. No bony erosion.

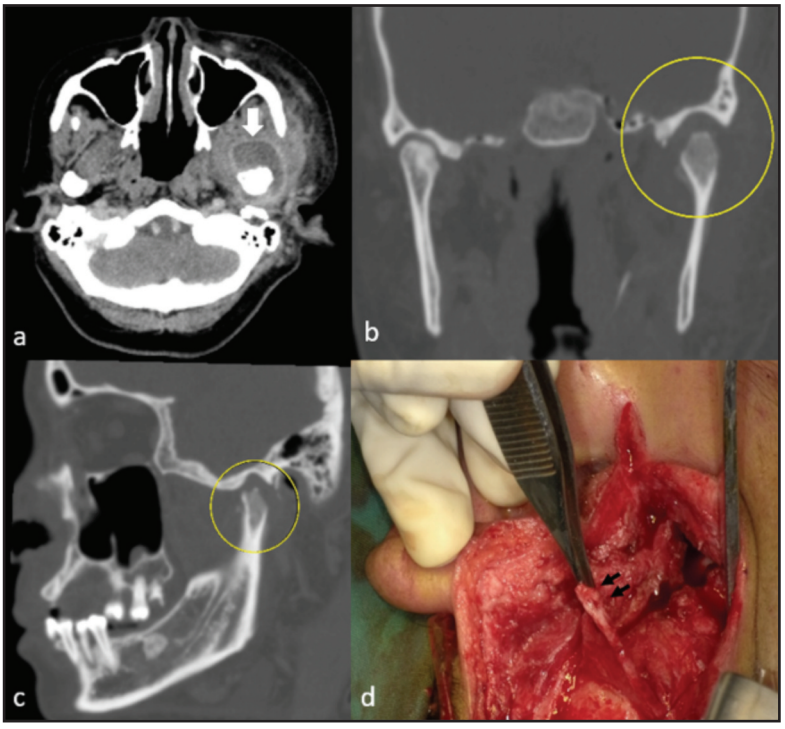

Fig. 2: Contrast enhanced CT brain: a. axial soft tissue setting showed rim enhancing collection at the left condylar process (white arrow); b. coronal reconstruction and c. sagittal reconstruction in bone setting showed left condylar erosion, joint widening and subluxation (yellow circle); d. Intraoperatively, left condylar erosion identified (black arrow).

arthroscopy procedure.[2] Few publications have reported local spread to TMJ after the molar tooth extraction, intra-articular injection, facial burns and otitis externa. The TMJ capsular breach due to blunt trauma has facilitated haematogenous spread of infection.[4]

The common presenting complains of SA TMJ are warm, erythematous pre-auricular swelling associated with pain, limited mouth opening and malocclusion. Differentials include infection; dental abscess, pharyngitis, retropharyngeal abscess, peritonsillar abscess, mastoiditis, parotitis, sialadenitis and lymphadenitis.[4] However, in the absence of inflammation, neoplastic process arising from mandibular condyle, parotid tumour, as well as other systemic diseases, namely rheumatoid arthritis, gout and pseudogout are diagnoses of choice.[3,4] Early diagnosis of TMJ septic arthritis is not easy as not all cases exhibit signs of inflammation. Therefore imaging plays an important role in suspected SA. The earliest sign of septic arthritis is joint effusion as a result of pus and inflammatory exudates accumulation in a joint space. Both MRI and CT imaging can detect joint effusion in the early stage.[5] However CT imaging is an excellent tool in demonstrating bony anatomy of TMJ, temporal bone and skull base. It can detect bony erosion and evaluation of potential extension to the contiguous soft tissue. MRI is superior in demonstrating the soft tissue anatomy surrounding the TMJ.[5] MRI was not done in this patient due to non-availability in the district hospital.

Elevated white blood count above the 12,000/mm^3 is one of four parameters used for clinical prediction of SA.[2] The serum erythrocyte sedimentation rate (ESR) and C-reactive protein are systemic acute phase reactants that can be elevated in SA. These three markers are non-specific and can be elevated in a non-infectious inflammatory condition. The procalcitonin level is a serum marker that can rise rapidly due to bacterial infection. Unlike ESR and CRP, serum procalcitonin remain low in systemic inflammatory disorders.[2] Nevertheless, these four markers are not predictive for SA. Therefore, correlation with the clinical history, physical examination and culture findings are mandatory.

There is no clear consensus on the management of TMJ septic arthritis and a few methods have been proposed including needle aspiration, arthroscopy and arthrotomy.[4] However, if the abscess formation is established, exploration and surgical drainage must be performed immediately. If bony erosion and cartilage erosion are identified intraoperatively, the condylectomy is required.[4] A paediatric patient diagnosed with TMJ septic arthritis is treated by aspiration alone and washout in acute setting. Immediate empirical antibiotic therapy is mandatory when diagnosed and later should be tailored to culture and sensitivity. Duration of antibiotic treatment should be at least 30 days.[4]

CONCLUSION

SA requires rapid and aggressive treatment as infection can lead to quick, permanent destruction. The pitfall in diagnosis is due to the rarity of the case and overlapping clinical findings with other inflammatory diseases coupled with lack of specific laboratory findings. Thus, high clinical suspicion and timely diagnosis are crucial in minimising the risk of ankylosis, impaired joint mobility and function, bone deformity and fibrosis.

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