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Case Report

A rare otorhinolaryngologic cause of Collet-Sicard syndrome

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ABSTRACT

The Collet-Sicard syndrome (CSS) is a rare clinical condition with unilateral palsy of lower cranial nerves IX, X, XI and XII without sympathetic involvement. This was first described by Frederic Collet. Primary or metastatic neoplasms or vascular diseases are the usual causes of CSS. Infection is a very rare cause of CSS. Skull Base Osteomyelitis (SBO) is a rare, complex, and fatal infection of the skull bones that commonly involves parts of the temporal, sphenoid, and occipital bones. The disease was first described in 1959 by Meltzer and Keleman. Malignant otitis externa (MOE) is the most common cause of SBO, particularly in temporal regions. Here we report a case of collet Sicard syndrome due to skull base osteomyelitis resulting from an otogenic infection, malignant otitis externa. This case report aims to create awareness of such a neurologic condition and also the importance of early suspicion, diagnosis and treatment of skull base osteomyelitis to prevent such complications.

Keywords: Collet siccard syndrome, Skullbase osteomyelitis, Cranial nerve palsy

INTRODUCTION

Collet-Sicard syndrome (CSS) is the unilateral palsy of lower cranial nerves IX, X, XI and XII.[1] This causes paralysis of the vocal cords, the palate and the trapezius muscle, sternocleidomastoid muscle; secondary loss of the sense of taste in the back of the tongue, and anesthesia of the larynx, pharynx, and soft palate.[2] CSS is a very rare condition resulting from lesions at the base of the skull that affect both the jugular foramen and hypoglossal canal. This can occur due to different causes, including tumours (primary or metastatic), trauma, vascular lesions, inflammatory processes, and iatrogenic complications.[1] Here we report a case of collet Sicard syndrome due to skull base osteomyelitis resulting from an otogenic infection.

CASE REPORT

A 72-year-old lady known diabetic presented to the hospital with complaints of pain left ear for 2 months and left ear discharge of one month duration. She also gave history of loss of taste, difficulty to swallow food and voice change.

Her pain started initially as a nocturnal earache, progressive, throbbing in nature and temporarily relieved on medications. The otalgia progressed to a diffuse pain extending to occipital area and left side of face. The patient noticed ear discharge mucopurulent, continuous and often foul smelling. She complained of hard of hearing, left more than right. She complained of dysphagia for one month with loss of taste. Her voice change was gradually progressive with weak and breathy voice. She is diabetic for last 25 years and she was on insulin since the past 12 years. She is also a known case of coronary artery disease on medication. She also gave history of pulmonary oedema 2 years back following an episode of acute kidney injury for which she had to undergo dialysis. On examination, external auditory canal showed purulent discharge. Tympanic membrane of left showed a small perforation with pulsatile discharge.

Right tympanic membrane was intact. Nasal endoscopy showed mild deviation of nasal septum. No active nasal discharge or crusts in the cavity. Her tongue was deviated to left. Palatal palsy noted on left side with absence of gag reflex. She was unable to shrug her shoulder on left side.

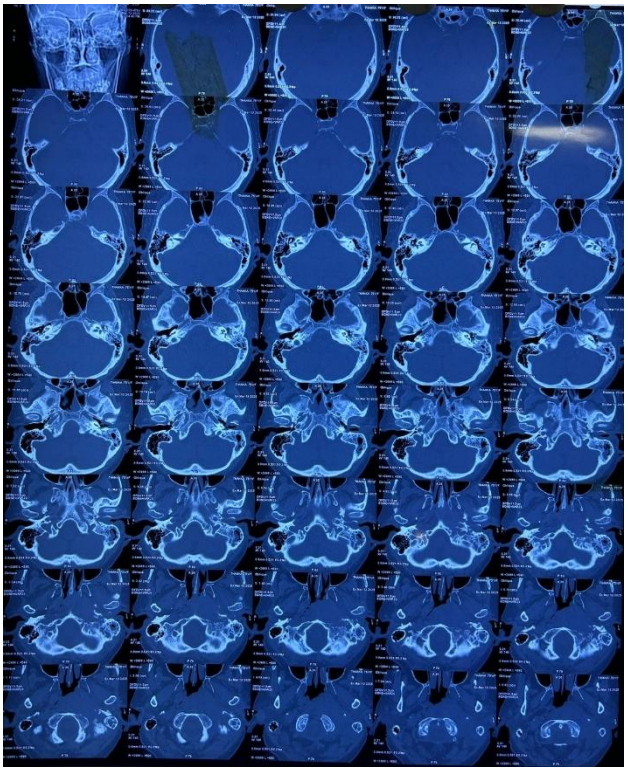

Figure 1: HRCT temporal bone showing the lesion.

On investigations, her total count, ESR, CRP were raised. Blood sugar level elevated and renal parameters deranged. HRCT Temporal bone showed a soft tissue density in the left middle ear, aditus, antrum and mastoid air cells and extending to left external auditory canal. Ill defined soft tissue lesion in left half of nasopharynx extending to parapharyngeal space. There is erosion of left lateral aspect of clivus and left temporal bone.

MRI skull base reported T2/FLAIR hyperintense fluid areas seen in left mastoid air cells and middle ear cavity. Diffuse stir hyperintense areas also seen extending up to posterior nasopharynx medially. Fluid areas also seen extending into external ear and external auditory canal laterally with erosion of mastoid air cells.

Diffuse marrow oedema noted in skull base including clivus and left side of atlas. Pus culture and sensitivity yielded coagulase negative staphylococcus aureus. Fungal stain and culture were negative. It was proved to be a case of skull base osteomyelitis She was admitted at a tertiary care centre and treated with intravenous antibiotics. Diabetic control attained by insulin. She had developed seizure during the hospital stay and was started on phenytoin. Renal parameters, ESR, CRP improved following 4 weeks of antibiotics and supportive care.

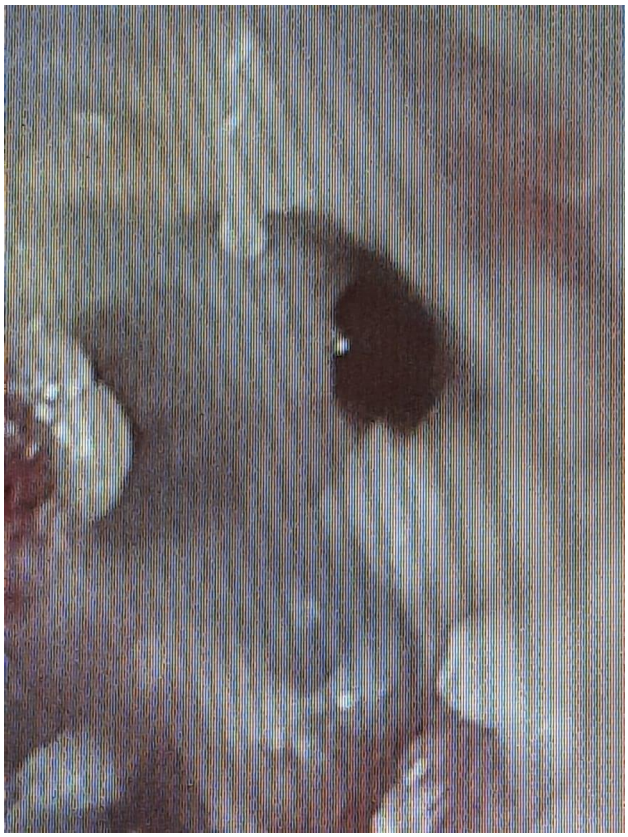

Figure 2: Tympanic membrane perforation as seen on oto endoscopy.

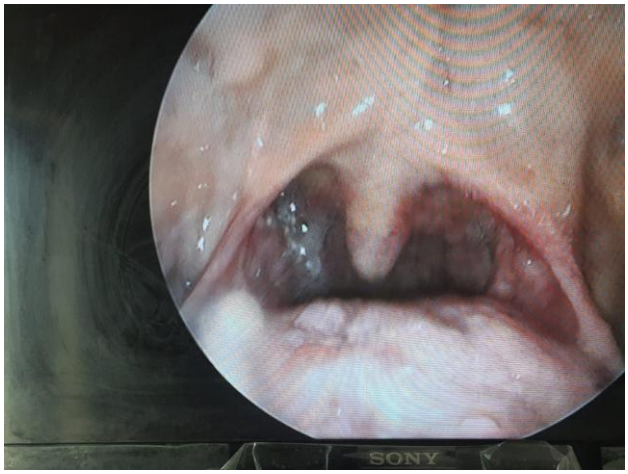

Figure 3: Palatal palsy.

On follow up, it was noted that she has lost her voice completely. Examination revealed that her ear discharge had subsided. Neurologic condition persisted. Considering her advanced age and comorbidities surgery was not considered as a viable option. She was advised admission and intravenous antibiotics at our hospital. Patient refused and hence the patient continues to be on oral antibiotics as per culture report.

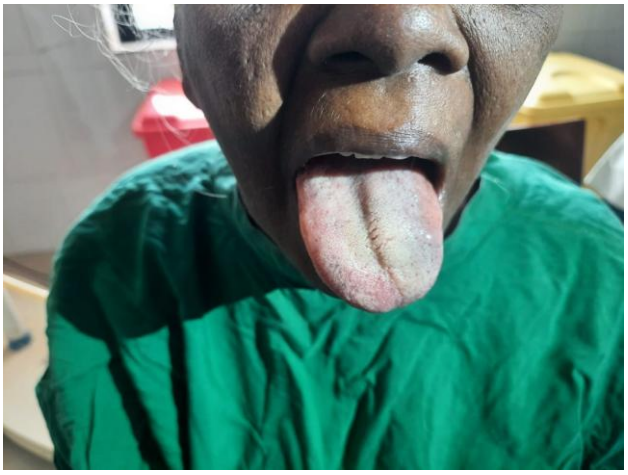

Figure 4: Tongue deviation due to hypoglossal nerve palsy.

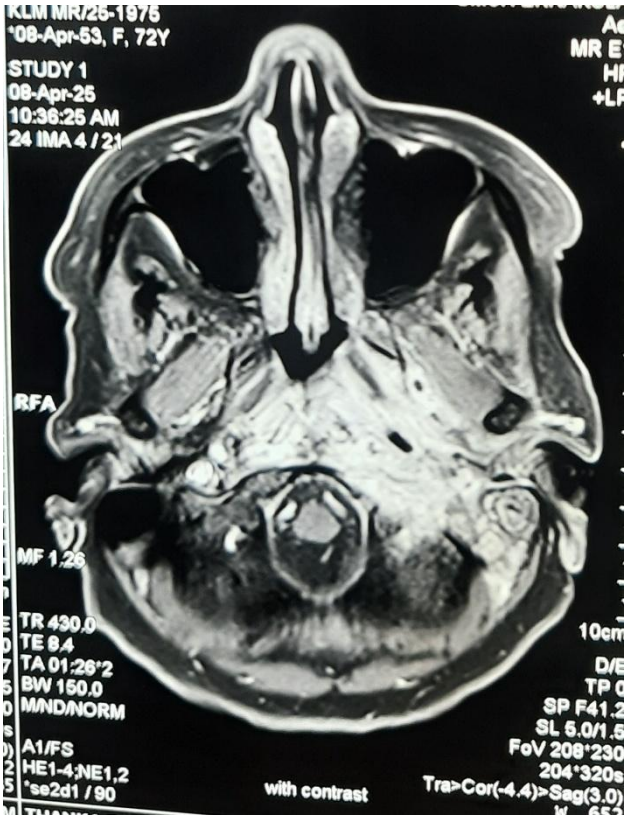

Figure 5: MRI showing the lesion.

DISCUSSION

Skull base osteomyelitis (SBO) is a complex and fatal clinical entity. SBO can be a direct complication of otogenic, sinogenic, odontogenic, and rhinogenic infections. SBO remains a great challenge probably due to its increasing prevalence and high mortality rate.[3] Skull base osteomyelitis can involve the jugular foramen and its associated cranial nerves resulting in specific clinical syndromes. CSS describes the clinical manifestations of palsies involving cranial nerves IX, X, XI, and XII due to the involvement of cranial nerves through jugular foramen and hypoglossal canal.[4]

Our case is an elderly lady with uncontrolled diabetes and multiple comorbidities. She developed an otogenic infection, malignant otitis externa, progressed to middle ear causing tympanic membrane perforation. The infection also involved the skull base resulting in skull base osteomyelitis with bone erosion. SBO involving jugular foramen and hypoglossal canal resulted in paralysis of IX, X, XI, XII cranial nerves termed as collet siccard syndrome.

Metastasis is found to be the most common etiology of CSS.[1] Infectious causes of CSS are considered rare.[2] Low WK et al has reported a case of skull base osteomyelitis from otitis media due to a retained foreign body presenting as the CSS.[4] Infectious CSS with skull-based osteomyelitis was reported by Sibai et al due to incomplete treatment of otitis media.[2] CSS as a complication of chronic middle ear infection was reported by Blazina et al.[5] A Case of CSS Caused by malignant otitis externa was found to be reported only by Bonda et al.[6]

CONCLUSION

Though the prevalence of skull base osteomyelitis is increasing, cases of CSS due to skull base osteomyelitis are still rare. This case report creates awareness of potential neurologic complications that can result from skull base osteomyelitis. It also emphasises the importance of early suspicion, early diagnosis and treatment of skull base osteomyelitis in patients with multiple comorbidities to prevent neurologic conditions like CSS.

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