Petrus Apicitis in a Previously Healthy 8-Year Old Female: A Case Presentation and Review of the Literature

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ABSTRACT

Petrus apicitis is a rare but serious sequela of suppurrative otitis media. Complications arising from petrus apicitis include cranial nerve palsies, meningitis, labyrinthitis, intracranial abscess formation, retropharyngeal abscess, venous sinus thrombosis, and death. The signs of petrus apicitis include otorrhea, retrobulbar pain, and abducens nerve paralysis. However, the classic Gradenigo’s triad is rare. Though traditionally treated with mastoidectomy and when necessary decompression of the petrous apex, the current trend favors more conservative management with myringotomy tube placement and high dose broad-spectrum antibiotics, reserving surgery for refractory cases. We present an 8-year-old female who presented with blurry vision and headache, who was diagnosed with petrus apicitis, and review the current literature.

INTRODUCTION

Intracranial complications of otitis media, including petrus apicitis, are rare but can lead to devastating complications when unrecognized or undertreated. Despite the decreased prevalence of the disease with routine use of antibiotic therapy, it continues to occur, though signs and symptoms are often indistinct and presentations vary enormously.

CASE PRESENTATION

An otherwise healthy eight-year-old female presented to the emergency department with two days of fever and blurry vision. For two weeks prior, she had been experiencing a left occipital headache. Her vital signs were within normal range. Physical examination was significant for left eye pain with upward gaze and a left abducens nerve palsy (Figure 1). Though her white blood cell (WBC) count was normal, differential showed a neutrophil predominance of 78%. Her inflammatory markers were elevated, with C-reactive protein (CRP) of 14 and erythrocyte sedimentation rate (ESR) of 43. A computed tomography (CT) of the head showed severe opacification of the left middle ear cavity and mastoid (Figure 2a). Magnetic resonance imaging (MRI) showed extensive abnormal enhancement of the apiical portion of the left petrous bone. Dural enhancement of the left medial temporal convexity and left tentorium was also seen (Figure 2b). Magnetic resonance venography (MRV) was negative for deep venous sinus thrombosis.

The patient underwent myringotomy with ventilation tube placement. Middle ear fluid culture was without growth. The child was discharged to home on hospital day six on one course of ceftriaxone, along with a short course of ciprofloxacin drops. Close outpatient follow up with Otolaryngology, Ophthalmology, and Infectious Disease was arranged.

On follow-up, the child continued to show gradual improvement. At her one-month follow-up, she was without facial pain or headache but remained with a left abducens nerve paralysis. At her two-month follow-up, she exhibited improved extracocular movement with only slight restriction of abduction on the left and her inflammatory markers normalized.

DISCUSSION

Prior to the widespread use of antibiotics, petrus apicitis was a common, often fatal, sequela of otitis media. 1–3 In 1904, Giuseppe Gradenigo first described the triad of symptoms related to petrous apicitis, including suppurative otitis media, deep facial pain resulting from trigeminal involvement, and abducens nerve palsy, now known as Gradenigo Syndrome.4 Cranial nerve involvement is the result of extradural inflammation. This inflammation can affect the trigeminal ganglion, and just medial to it the abducens nerve as they are separated from the petrous apex by only a thin layer of dura mater. Sixth nerve palsy associated with petrus apicitis is thought to be due to inflammation of the abducens nerve as it courses through Dorello’s canal, an inflexible channel through the petrous bone roofed by the petroclinoid ligament5. The time interval between the onset of the otitis and the manifestation of cranial nerve dysfunction varies widely, ranging from one week to three months6.

Petrus apicitis is differentiated into acute and chronic forms. Acute petrus is defined by abscess formation in a well localized infection with organisms most likely to cause otitis media, namely Streptococcus pneumonia, beta hemolytic streptococci, Staphylococcus species, Haemophilus influenza, Pseudomonas species, Moraxella catarrhalis, and various anaerobes1. With the advent of antibiotic therapy, complications of otitis media such as Gradenigo Syndrome have become rare.1 When petrus apicitis does occur, it is often recognized late due to the subtlety of its signs and symptoms2,3. Unrecognized and undertreated petrus apicitis can lead to intracranial complications including meningitis, intracranial abscess, Vernet’s syndrome, cavernous sinus thrombosis, hydrocephalus and even death.1,9 Diagnosis of petrus apicitis requires a high level of clinical suspicion. Retrobulbar and/or facial pain in the presence of parietal otorrhea should alert the physician to the possibility of petrus apicitis1. The diagnosis should also be considered whenever other cranial nerves are affected in a patient with chronic suppurative otomastoiditis3.

CT remains the first line imaging for possible lesions of the petrus apex1. CT will demonstrate petromastoid air cell opacification with destruction of superior transverse and a discozyte and giving it as the extent of inflammatory change6. MRI is useful to establish the extent of meningeal and cerebral involvement and to discern between the differing pathologies that feature in the petrus apex.

Traditionally, management was surgical, consisting of mastoidectomy with or without decompression of the petrus apex. More recently, a trend of conservative management, including myringotomy tube placement and high dose broad spectrum antibiotic administration, has gained favor. Given the difficulty isolating a causative organism, empiric antibiotic therapy is often necessary4. Empiric antibiotic choice is based on providing coverage for organisms most likely to cause otitis media, namely Streptococcus Pneumonia, beta hemolytic streptococci, Staphylococcus species, Haemophilus influenza, Pseudomonas species, Moraxella catarrhalis, and various anaerobes1.

Duration until resolution of the abducens nerve palsy varies from several days up to six weeks. This is usually the last symptom to resolve4. For those who do not respond to conservative measures or who present with established intracranial complications, surgical intervention in the form of apical petrosectomy remains mandatory1,9.

CONCLUSION

Using conservative management alone our patient experienced resolution of symptoms within eight weeks following myringotomy with tube placement and high-dose broad spectrum intravenous antibiotic therapy. Given the gravity of the potential complications of petrus apicitis, early recognition and appropriate therapy are crucial. Diagnosis should be considered in any patient presenting with facial pain and abducens nerve dysfunction. All patients should have a thorough clinical history focusing on otologic history and dedicated imaging of the temporal bone. It remains imperative to have a high level of clinical suspicion and to institute treatment with high-dose broad spectrum intravenous antibiotics while investigations are ongoing, as delay in treatment may prove fatal1.

REFERENCES
