CASE REPORT

Iatrogenic sinus thrombophlebitis – A case report

Lingamdenne Paul Emerson *,1, Anand Job, Sophia Amalanathan Rita Ruby Albert Anbuselvi

Department of ENT, Unit-I, Christian Medical College, Vellore 632004, Tamilnadu, India

Received 10 March 2012; accepted 4 April 2012
Available online 11 May 2012

KEYWORDS
Chronic venous sinus thrombosis; Chronic iron deficiency anemia; Septic thrombophlebitis

Abstract Mastoidectomy is one of the common surgical procedures done for exenterating infection of the mastoid bone. Intraoperative complications such as dural tear, lateral sinus bleed are known iatrogenic complications, however, infective sinus thrombophlebitis causing diffuse sinus thrombosis and meningitis following an iatrogenic sinus bleed is a rare complication and we discuss the etiology and the cause for this complication and management.

© 2012 Egyptian Society of Ear, Nose, Throat and Allied Sciences. Production and hosting by Elsevier B.V. All rights reserved.

1. Case report

Eighteen year old girl presented with complaints of right ear discharge and decreased hearing since childhood. The discharge was continuous, mucoid to mucopurulent and occasionally blood stained, relieved on taking oral antibiotics and aural medication. She did not report tinnitus, headache, nausea, vomiting or vertigo. There were no nose or throat complaints. She had chronic iron deficiency anemia which was treated.

On clinical examination the right external auditory canal was filled with red fleshy polyp. There was no evidence of seventh or other cranial nerve defects or vestibular function. Motor function was normal. Audiogram showed profound hearing loss on the right and normal hearing on the left. Radiographs of the mastoid showed sclerosis. A mastoid exploration was planned on the right side. Preoperatively there was a fleshy polyp filling the external auditory canal, which was arising from the middle ear which was filled with granulation tissue. The posterior canal wall was eroded. While performing the cortical mastoidectomy a forward lying sinus was encountered which was breached accidentally. Sinus bleed was controlled with gel foam, surgical bone and wax and the middle ear was packed with gelfoam and the surgery abandoned.

Postoperatively the patient was shifted to the ward for monitoring. On the 3rd postoperative day the patient developed focal seizures, fever, vomiting and her sensorium deteriorated. The patient was intubated and an emergency contrast CT scan of the brain performed which showed a rim enhancing focus in the lateral aspect of the right cerebellum, meningeal enhancement at the basal region and centrally enhancing filling defect in the right transverse sinus. Provisionally the patient was diagnosed to have lateral sinus thrombosis and meningitis. The patient was shifted to medical intensive care unit and was monitored for a week with invasive ventilation, iv antibiotics (Vancomycin, Piperacillin with clavulanic acid), anti-edema measures and low molecular weight heparin.
MRI scan of brain on the following day showed extensive filling defects in the right transverse and sigmoid sinuses suggestive of thrombosis, with thickening of the wall of the superior sagittal sinus suggestive of acute on chronic thrombosis (Fig. 1). Her acute episode was attributed to the compromised vascular supply and raised intracranial pressure. The patient was monitored in medical intensive care for 10 days. Histopathology of aural polyp was non specific inflammatory granulation. Her sensorium improved subsequently and she was weaned off the ventilator and shifted to the ward. Motor deficits that were noticed earlier in the right lower limb improved slowly and at discharge was able to walk without support. She was discharged on warfarin and is kept on follow up. After 1 year of follow up the external auditory canal and middle ear mucosa was normal and has no focal neurological deficits.

Discussion: Cerebral venous thrombosis (CVT) was first described in a post-mortem report in the French literature in 1825. It is an infrequent condition characterized by a wide spectrum of clinical presentations and modes of onset, which depend on the site, extent, and rate of progression of thrombosis. The two most frequent sites of thrombosis are the superior sagittal sinus (SSS), and the lateral sinus (LS).

The lateral sinus is the largest of the dural venous sinuses and it is connected anteriorly to the cavernous sinus by the superior petrosal sinus, it also receives the mastoid emissary vein. The lateral sinus joins the internal jugular vein at the base of skull. Due to rigid walls the lateral sinus has a constant caliber predisposing it to stagnation or thrombosis in case of local sepsis. This also facilitates propagation of thrombus within the sinus. Situated on both sides the lateral sinuses provide the main venous drainage of the intracranial cavity. The right lateral sinus is considerably wider than the left and so injury to the right sinus can compromise intracranial drainage until collaterals are established. Multiple pathophysioligic mechanisms and predisposing factors exist including hypercoagulable state due to iron deficiency anemia, extrinsic compression or local invasion of a venous sinus by tumor or an adjacent infective process (e.g. mastoiditis) or a low flow state within the venous sinus due to dehydration, pregnancy and postpartum state.

A well-known vascular complication of otitis media and/or mastoiditis is septic thrombosis of the sigmoid portion of the lateral venous sinus. Although it has become rare with the advent of antibiotics, it can still be fatal especially when associated with other intracranial complications. Clinical manifestations of cerebral sinus venous thrombosis (CSVT) are non-specific and may be subtle or present as seizures, coma, stroke, headache and raised intracranial pressure. Occasionally venous thrombosis may be mistaken for a psychiatric disorder, such as neurosis, hysteria or depression.

1.1. Pathophysiology

Cerebral venous thrombosis results from occlusion of a venous sinus and/or cortical vein and is usually caused by a partial thrombus or an extrinsic compression that subsequently progresses to complete occlusion, leading to venous hypertension, increased intracranial pressure, hemorrhagic infarction and otitic hydrocephalus. Common illnesses including ear infections, meningitis, anemia, diabetes and head injury, may be complicated by cerebral venous sinus thrombosis.

In our patient, an underlying chronic sinus thrombosis, possibly due to iron deficiency anemia, could have resulted in a diffuse cortical venous thrombosis because of sepsis. Injury to the lateral sinus during mastoidectomy is a potential pathway for introducing infection causing diffuse cortical venous thrombosis. The treatment of LST is both surgical and medical and in our patient the right sinus was breached and that probably compromised the intra cranial tension. Infective thrombophlebitis after iatrogenic injury should always be kept in mind and the patient monitored in case may develop sinus thrombophlebitis. A diagnosis using MRI with contrast and MRV is preferred over other modalities. The diagnosis may be made or suggested by CT brain scan, before and after intravenous contrast medium injection. Even though lateral sinus thrombosis is a well known intra-cranial complication, there are only a few case reports of cerebral venous thrombosis resulting from this condition.

This potential serious complication should be kept in mind when inadvertent injury to lateral sinus occurs and in a developing country where contributing factors for chronic sinus thrombosis such as malnutrition, dehydration and iron deficiency anemia are common.

Postoperatively patient has to be kept under observation so that any signs of raised intracranial tension can be treated immediately. Radiological contrast studies are considered very important in diagnosing the condition .The prompt recognition of the condition and the application of appropriate medical and surgical therapy will reduce the number of further complications as well as mortality rate.

References

3. Belman Anita Lesgold, Roque Qemente T, Ancona Richard, Anand Azad K, Davis Raphael P. Cerebral venous thrombosis in...


