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Cerebral Venous Sinus Thrombosis (CVST) Complicating Middle Ear Infections, A Rare Complication in Post-Antibiotic Era: A Case Report with Review of Literature

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Mishra SC, Tyagi I, Gupta A, Tyagi L and these authors are contributed equally to this work.

1. Abstract

In the post-antibiotic era intracranial and extra-cranial complications of middle ear infections have become rare. Similarly, Cerebral Venous Sinus Thrombosis (CVST) which was a frequent complication of middle ear infections has become rare now. Here we present a case of a 27-year male who presented with a short history of severe headache and associated episodes of unretract able vomiting. The patient did not improve clinically even after prompt symptomatic management following which a Contrast Enhanced Magnetic Resonance Imaging (CEMRI) of head along with Contrast Enhanced Magnetic Resonance Venogram (CEMRV) was done which was showing right sided Otomastoiditis complicated with CVST and meningitis. Although the clinical signs of meningeal irritation and mastoid tenderness were not present on clinical examination. Patient was started on anticoagulant therapy and antibiotics for 2 weeks following which there was marked clinical improvement.

2. Clinical Presentation

A 27 years' male who is nursing staff by occupation presented to Emergency Department (ED) with complains of severe holocranial headache for 3 days with associated unretractable vomiting. Three days back when the patient woke up in the afternoon with severe headache following which he became unconscious. The patient regained consciousness after about 5 minutes when he developed the aforementioned episodes of vomiting. There was no history of trauma, fever, weakness over one side of body or visual disturbances. There was no such previous history. There is no history of addiction, Pulmonary Koch's or hypothyroidism. Patient was not on any antihypertensive medication.

However, there was history of episodes of right ear discharge one year back for which he took some medication and was symptom free for the past 1 year.

Clinical examination showed raised blood pressure of 140/90mm Hg with pulse rate of 86/minute. Patient was afebrile with no pallor, icterus, lymphadenopathy or peripheral edema. Respiratory system was within normal limits. No focal neurological deficits or cranial nerve palsies.

Patient was admitted for further evaluation and symptomatic treatment for headache was started in the form of anti-osmotic agents and antiemetics for vomiting. Even after prompt symptomatic treatment there was no significant clinical improvement following which CEMRI along with CEMRV was done which revealed right Otomastoiditis complicated by acute thrombosis of the right transverse and sigmoid sinuses along with meningitis. Patient was started on Low Molecular Weight Heparin (LMWH) and parenteral antibiotics. He was also investigated for inherited and acquired

hypercoagulable states.

There was marked clinical improvement and patient was discharged with the suggestion of follow up in Neurology OPD. He was asymptomatic at one month follow up and was advised mastoidectomy as per surgical opinion from ENT surgeon.

3. Investigations

Hemogram was normal with Hb13gm. TLC, DLC and platelets were within normal limits. ESR was raised (49), Prothrombin Time (PT) was normal (INR 1.14). S. creatinine was normal, C-reactive protein was normal, antinuclear antibody and anti-parietal cell antibodies were negative, Anti-phospholipid antibody profile was within with normal limits. Lupus anticoagulant was negative, Activated protein C resistance assay was negative, vitamin B12 level was normal, Homocysteine and Folate were within normal limits, PNH screen was negative, Anti-thrombin III activity level measured 129% and protein C functional assay were marginally abnormal with Anti-thrombin activity level measuring 129% (Normal range 80-120%) and protein C levels measuring 131%(Normal range 70% to 130%), Normal Protein S –Functional assay with Protein S activity level measuring 85% and mildly increased plasma fibrinogen level measuring 439mg/dl.

CEMRI with CEMRV showed fluid intensity in right mastoid air cells with distended hyper intense right transverse and sigmoid sinuses showing loss of normal flow void on T2 and T2 FLAIR (Figure 1a and Figure 1b). On axial T1 non-contrast scan (Figure 1c) there was evidence of distended right transverse sinus and on axial T1 post-contrast scan (Figure 1d) there was non-enhancing filling in the distended venous sinus suggestive of acute thrombosis. There was an associated focus of nodular leptomeningeal enhancement in the right temporal lobe (figure 1d) and left frontal lobe (Figure 1e) suggestive of meningitis. There was non-visualized of right transverse on 3D TOF MRV image (figure 1f).

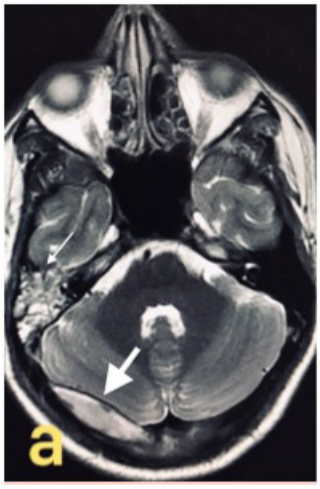

Figure 1a: Axial T2WI of brain showing fluid intensity in the right mastoid air cells (denoted by thin white arrow) and T2 hyper intense distended right transverse sinus (denoted by thick white arrow).

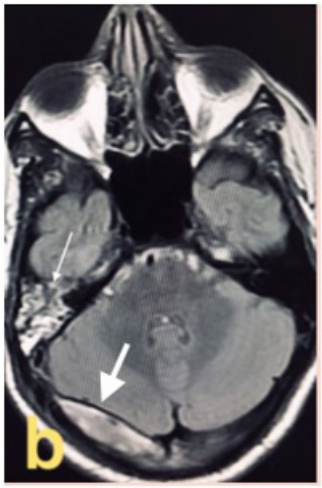

Figure 1b: Axial T2 FLAIR of the brain showing similar signal characteristics involving the right mastoid air cells (denoted by thin white arrow) and right transverse sinus (denoted by thick white arrow).

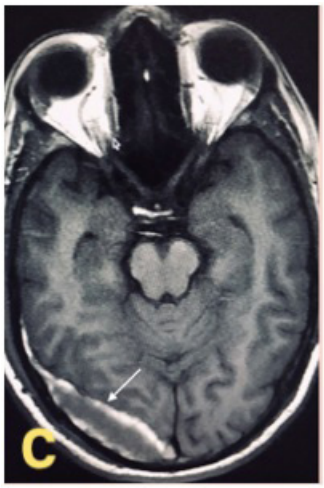

Figure 1c: Axial T1 non-contrast image showing distended right transverse sinus (denoted by white arrow).

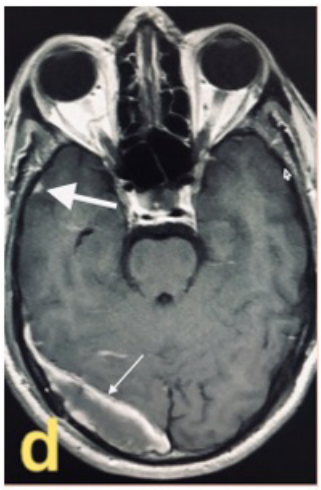

Figure 1d: Axial T1WI post-contrast image showing non-enhancing filling defect in right transverse sinus (denoted by thin white arrow) and focus of abnormal parenchymal enhancement in right temporal lobe (denoted by thick white arrow).

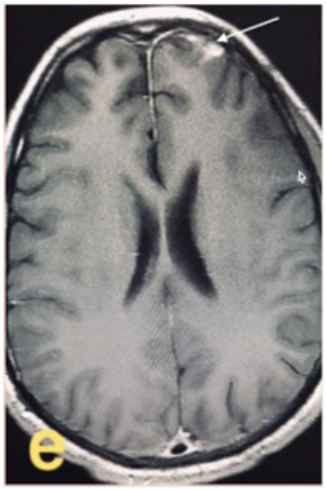

Figure 1e: Axial T1WI post-contrast images showing nodular focus of leptomeningeal enhancement in right frontal lobe (denoted by thin white arrow).

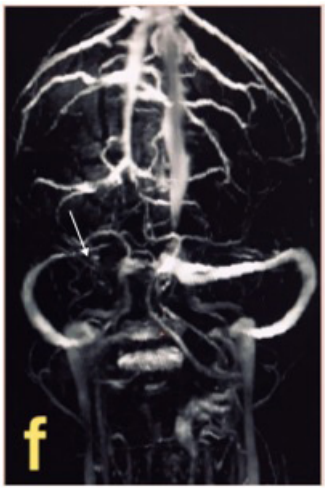

Figure 1f: 3D TOF MRV image showing the non-visualization of right transverse sinus and portion of right sigmoid sinus (denoted by white arrow).

4. Treatment, Outcome and Follow Up

1. Parenteral antibiotics were started for meningitis.

2. Injection low molecular weight heparin (LMWH) twice daily, subcutaneously followed by a course of oral anticoagulant Dabigatran Etexilate.

3. Oral anti-epileptics.

Patient was on follow up in neurology OPD and show marked improvement in the neurological symptoms related to CVST and is now dated to undergo radical mastoidectomy as per consultation with ENT department.

5. Discussion

The intracranial and extra-cranial complications of middle ear infections have decreased significantly in the post-antibiotic era. CVST, one of the complications frequenting middle ear and mastoid infections has also become rare. The mortality associated with CVST reached 100 percent in the early 20th century [1] and has now decreased significantly to less than 10 percent due to wide spread availability of highly potent antibiotics [2]. CVST can have associated morbidity due to septic cardiomyopathy, ARDS, anacusis and seizures [3].

However, due to indiscriminate use of antibiotics and antibiotic resistance the cases are still seen complicating the acute and chronic middle ear and mastoid infections [4].

In young adults hypercoagulable states both inherited (Protein C and protein S deficiency, factor V Leiden mutation etc.) and acquired (Trauma, Tumor, inflammation, pregnancy etc.) are a major risk factor [5, 6].

Infections involving the middle ear structures such as acute otitis and mastoiditis uncommonly cause thrombosis of the sigmoid and transverse sinuses due to structural contiguity [7, 8]. There is direct spread of the inflammatory process from the middle ear infections including acute Otomastoiditis via the small venues draining into the sigmoid sinus [9].

The thrombosis of the cerebral venous sinuses causes intracranial venous hypertension [8, 10]. The occlusion of the veins also leads to cerebral edema and venous infarction.

CSVT can have varied clinical manifestations, wherein 30% can present acutely within 2 days of blockage, half of the patients present in a sub-acute fashion within 2 days to 30 days, and 20% may present anytime from 30 days to six months [10]. Around 9 out of 10 adult patients diagnosed with CVST had ipsilateral headache [8, 10].

Other presentations include edema and tenderness over the mastoid process called as “Griesinger sign”, nausea, vomiting, altered mental status, seizures, focal motor deficit, double vision, and earache [8, 9, 10].

Intracranial hypertension leading to papilledema and resultant visual deficits has been reported in 13.2% of the patients [7, 9, 10]. Ophthalmoplegia can occur secondary to 3rd, 4th and 6th cranial nerve palsy with associated eye tenderness [8, 9, 10].

Uncontrolled intracranial hypertension can cause major complications such as permanent blindness, status epilepticus, coma, and death from cerebral herniation [8].

In cases of middle ear infection with suspected intracranial complications, imaging is imperative for confirming diagnosis & planning management.

CT head with temporal bone can show erosive changes in the bones, empyema, cerebral abscess, and also the thrombus in the cerebral venous sinuses which manifests radiologically as “delta sign” [3].

MRI head with contrast MRV is the most sensitive modality for confirmation of CVST [8, 11]. They thrombosed venous sinuses show low or absent flow and clot formation, which appears as in-

creased signal intensity in T1 and T2 images and the presence of inflammation in the brain and meninges [9, 11]. MRI can be normal in 3 out of 10 patients10. However, CT venography and MRI venography have 95% sensitivity [8, 10, 11].

Of late the treatment of CVST has become more conservative [12].

Once the diagnosis of CVST is established anticoagulation initiation with heparin is crucial [8, 10]. Heparin causes clearing of the thrombus from occluded cerebral veins/sinuses, reverses the thrombotic process as well as prevent further thrombus propagation and prevent pulmonary embolism [8, 11]. Unfractionated (UFH) and low molecular weight heparin (LMWH) are used with LMWH preferred over UFH due to practical advantages [10]. Routinely the duration of anticoagulation is 3 months, but 3 to 6 months for impermanent risk factors such as Trauma, pregnancy, infection etc. Longer duration of anticoagulation up to 12 months is required for pro-thrombotic states eg. Active malignancy [10, 11].

In patients not responding to medical management and showing progressive worsening, endovascular interventions (Thrombolysis and venoplasty) can be done and in cases with increased intracranial pressure causing pressure effects decompression craniotomy can be done [10].

With the advancements in neuroimaging, prompt diagnosis and early treatment has markedly decreased the mortality associated with CVST with good long term neurological outcome [13].

High index of suspicion and early diagnosis using modalities like CEMRV and CT can be lifesaving.

In young adults the hypercoagulable states are an important etiology of CVST. Thorough work up should be done in these patients to rule out such associated coagulation pathologies.

Undiagnosed CVST can have fulminant clinical course with increased intracranial hemorrhage and its complications like edema and mass effect.

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