A RARE PRESENTATION OF CEREBRAL VENOUS SINUS THROMBOSIS AS SUBARACHNOID HAEMORRHAGE

SURESHKHANNA CHELLIAH
Department of Neurology,
MADURAI MEDICAL COLLEGE AND HOSPITAL

Abstract:
Previously, Subarachnoid Haemorrhage (SAH) is most often due to aneurysmal rupture. Other vascular lesions are known to rarely cause SAH. Cerebral venous sinus thrombosis (CVT) can be difficult to diagnose because of its wide spectrum of clinical manifestations. Its diagnosis can be further complicated when patients initially present with acute SAH. We report a rare presentation of CVT as SAH. We report a case of 40 years old female patient presented with acute onset of severe headache with vomiting. Computerised Tomography (CT) contrast brain showed SAH in the basal cistern. Digital Subtraction Angiography (DSA) was done to detect the cause of SAH which was normal. Magnetic Resonance Imaging (MRI) of brain was done which showed thrombus in the superior, inferior sagittal sinuses, Transverse sinus and sigmoid sinus on both sides. Patient improved with anticoagulant therapy. This case highlights the fact that SAH may reveal a CVT, and emphasizes on the inclusion of Magnetic Resonance Venography (MRV) in the diagnostic workup of SAH, particularly in cases in which aneurysm is not detected.

Keyword: CEREBRAL VENOUS SINUS THROMBOSIS, DIGITAL SUBTRACTION ANGIOGRAPHY, MAGNETIC RESONANCE ANGIOGRAPHY, SUBARACHNOID HAEMORRHAGE

INTRODUCTION:
Cerebral Venous Thrombosis (CVT) can be difficult to diagnose and is further complicated when patients initially present with acute Subarachnoid Haemorrhage (SAH) [1]. SAH associated with CVT is rarely reported in the literature [2,3]. Acute SAH suggests the presence of a vascular lesion such as ruptured aneurysm and CVT is not generally considered in the diagnostic workup of SAH [4]. Surprisingly patients with CVT and radiologic signs of SAH are seldom reported [5,6]. SAH in these cases is probably due to raised venous pressure of draining venous tributaries [7]. We present the case of a female patient with dural sinus thrombosis whose initial
presentation was SAH. After therapy with subcutaneous Low Molecular Weight Heparin (LMWH), the sinus thrombosis resolved.

CASE REPORT:
40 years old non diabetic, normotensive female patient presented with acute onset of holocranial headache with vomiting and without photophobia or phonophobia since 2 days. She did not experience such a severe headache in the past and it was more severe in the early morning hours. She did not have any fever, seizures, loss of consciousness or altered sensorium or any ear discharge. Her neurological examination was unremarkable except for the neck stiffness and bilateral papiledema. Complete Hemogram and blood biochemistry were within normal limits. CT contrast brain [figure 1] showed SAH in the basal cisterns. She was treated with antiedema measures, and with Intravenous fluids. DSA was done to detect the the cause of SAH which was normal. Since her headache did not subside with treatment, MRI brain [figure 2, figure 3] with MRV [figure 4] was done which revealed thrombus in the superior, inferior sagittal sinuses, transverse and sigmoid sinuses on both sides. Patient was screened for hypercoagulable states — all were negative. Patient was treated with LMWH for 10 days followed by oral warfarin which was maintained on International Normalised Ratio (INR) of 2-3. Headache subsided over a period of 5-7 days. Patient was followed up at 2 weekly intervals for 3 months. Repeat MRI brain with MRV done which was normal.
DISCUSSION:
The spectrum of clinical presentation of CVT ranges from headache with papilledema to focal deficit, seizures and coma. Upto 75% of the cases are characterised by focal neurologic deficit and headache; 30-50% of affected patients present with seizures, often followed by Todd’s paresis. Rare, but classical, clinical pictures are those of superior sagital sinus thrombosis (4%) with bilateral or alternating deficits and/or seizures. SAH is related to ruptured aneurysm in 85% of cases [6] and nonaneurysmal perimesencephalic hemorrhage in 10%; the remaining 5% are related to a variety of rare conditions such as arterial dissection, Dural Arteriovenous (AV) fistula, Pituitary apoplexy and cocaine abuse. [6, 8, 9]

In an exhaustive review of SAH, CVT was not included as a cause of SAH. [8] This omission is rather surprising because Erythrocytes are commonly present in the Cerebro spinal fluid (CSF) of patients with CVT. [3, 5, 10]

One can speculate that plain CT scan may cause small amounts of subarachnoid blood to be overlooked, especially when the blood is located in the sulci of cerebral convexity or when larger amounts of extravasated blood are present in subacute stages. [5]

The exact cause of SAH associated with CVT is unknown. Venous hemorrhagic infarct can be responsible for secondary rupture into subarachnoid space and cause SAH. [7]

None of the patients reported herein had intraparenchymal signs of bleeding on CT scan or Gradient Recalled Echo (GRE) MRI. Dural sinus thrombosis with secondary venous hypertension may lead to SAH into the subarachnoid space due to rupture of fragile, thin walled cortical veins. [7]

Sinus thrombosis may produce dilatation of cortical veins, which may rupture and bleed into the subarachnoid space and produce an SAH. [11]

A similar mechanism has been proposed to explain the presence of extravasated blood confined to the prepontine or interpeduncular cistern in nonaneurysmal perimesencephalic hemorrhage. Patients with CVT can present with headache of sudden onset, neck stiffness and imaging evidence of SAH, simulating a ruptured Intracranial aneurysm. [3]

The presence of acute SAH of the convexity, especially when basal cistern spared, should prompt dedicated vascular imaging of both Intracranial arteries and dural sinuses. [4, 5]

In most institutions, DSA which remains the criterion standard for detecting cerebral aneurysm, is still part of the systemic workup for patients with acute SAH. [3, 5]

Findings in the venous phase of DSA lead to the diagnosis of CVT, even if it is previously unsuspected, since noninvasive angiographic techniques are seen to be gradually replacing DSA [3] in the workup of patients with acute SAH.

The diagnosis of CVT is becoming more challenging. Indeed, in patients with SAH, a CT or MRA focused on the arteries of the circle of Willis does not provide adequate imaging of the distal arteries or the venous system during the same imaging session. [10]

Therefore, unless CVT is systematically considered in the diagnostic workup of SAH of the cerebral convexity [5, 7], it could remain undiagnosed when these noninvasive angiographic techniques are used. Dural AV fistulas, with cortical venous drainage, are at relatively high risk of SAH and have been associated with CVT, presumably because of impairment of cerebral venous drainage. [12].

CVT can be difficult to diagnose because of its wide spectrum of clinical manifestations. Diagnosis may be further complicated when patients initially present with acute SAH. [1]
The hemorrhage is probably induced by rupture of a dilated vein associated with superior sagittal sinus thrombosis. The location of SAH from Superior sagittal sinus thrombosis is usually different from arterial aneurysms. [2] Angiography remain the diagnostic gold standard. [3,5] Management of venous SAH secondary to CVT is also quite different from that of arterial SAH. Treatment of sinus thrombosis with heparin has long been controversial. The benefits of heparin have been demonstrated in a randomised and placebo-controlled trial of 20 patients. [13] In a further placebo-controlled trial, 60 patients were randomised to either LMWH followed by Warfarin or placebo. [14] The anticoagulated patients had better outcomes than the controls, but the difference was not statistically significant. The investigators suggested that anticoagulation was safe, even in patients with cerebral hemorrhage. [14] A subsequent study showed no clear benefit of anticoagulant treatment, but the investigators continued to suggest that there was a nonsignificant trend in favour of anticoagulant treatment. [11] In our patient, the effect of anticoagulation was good. Our patient probably had a hypercoagulable trait, even though the results of available coagulation tests were within normal limits.

REFERENCES:


