# Case Report

# **Superior Sagital Sinus Thrombosis-A Case Report**

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#### ABSTRACT

A forty years old lady on 15<sup>th</sup> day of her puerperium presented in the emergency ward of Allied hospital Faisalabad with severe headache left sided hemiparesis and focal fits. On examination she had upper motor neuron signs on left side and bilateral papilloedema. CT scan brain showed multiple small areas of haemorrhages in a large infarct. CT venography showed superior sagital sinus thrombosis. She was anticoagulated. Cerebral

#### CASE REPORT

A 40 year old lady fifteen days after delivering a baby presented in the emergency ward of Allied Hospital Faisalabad with loss of consciousness. Four days prior to her admission she developed symptoms of severe headache and weakness of the left side of her body, which was sudden in onset. These symptoms were followed by focal fits on left side which were tonic clonic in nature.

She was taken to a nearby hospital where she was treated for three days and showed no improvement. On the last day of her stay in that hospital she became unconscious and was brought to Allied Hospital Faisalabad.

She had no past history of diabetes mellitus, hypertension, smoking, contraceptive use, cardiovascular disease, rheumatic fever or TIAs. There was no history of arterial or venous thrombosis. Family history was unremarkable.

On examination in the emergency ward this middle aged unconscious lady had a GCS score of eight. She looked rather pale and was breathing heavily and noisily. She was noted to have temperature of 100 degrees Fahrenheit, her pulse rate was 102 per minute, blood pressure 140/90 mmHg. In response to painful stimuli she could only move right side of her body. Further neurological examination revealed deep tendon reflexes on the left side to be of grade 3 venous thrombosis is a rare cause of CVA. It can be caused by number of congenital and acquired prothrombotic states and drugs. It usually presents with headache focal neurological deficit, fits, altered conscious level and papilloedema. Diagnosis is confirmed on CT venography and magnetic resonance venoghraphy. Patients suffering from this disease are treated with anticoagulants.

(exaggerated). Pupils were equal and normal in size with normal reaction to light. Fundus exam revealed Papilloedema on both sides. Examination of the chest revealed coarse crackles bilaterally in upper middle and lower zones both anteriorly and posteriorly. Cardiovascular and abdominal examination were unremarkable.

The investigations performed in the previous admission showed hematocrit of 31 percent; the white-cell count 15600 per cubic millimeter, with 87 percent neutrophils, 11 percent lymphocytes, and 2 percent of mixed cells. The platelet count was 569000 per cubic millimeter. Total Cholesterol was 305 mg/dl, Triglycerides 150 mg/dl, HDL 36 mg/dl and LDL 239 mg/dl. Calcium, Phosphorus, Liver function tests, Blood Urea, Serum Creatnine and Electrolytes were all normal. CT scan performed on the 1<sup>st</sup> day of admission also didn't show any abnormality.

On admission to Allied Hospital her CBC showed following picture. Hematocrit was 34 percent; the white-cell count 12500 per cubic millimeter, with 85 percent neutrophils, 14 percent lymphocytes, and 1 percent Eosinophils, platelets 133000 per cubic millimeter, ESR 105 mm after 1<sup>st</sup> hour. Total protein level was 7.3g per deciliter(albumin level, 3.2 g per deciliter; globulin level, 4.1 g per deciliter). Catheterized urine sample examination revealed

proteinurea and heamaturia.

The levels of blood urea, creatinine, glucose, total bilirubin, electrolytes, aspartate aminotransferase, and alkaline phosphates, were normal.

Computed tomographic (CT) scanning of the brain, performed without the administration of contrast Material on the seventh day of onset symptoms, revealed large hypodense areas with small and multiple hyperdense areas in different section. (Figure 1a and b). A computed tomographic venography was carriedout and result showed a large throumbus in the superior sagital sinus (Fig. 2a and b)

### DIAGNOSIS

Superior Sagital Sinus thrombosis.

#### DISCUSSION

Cerebral venous thrombosis is an uncommon cerebrovascular disease, with an estimated incidence of 3-7 cases per million per year.<sup>1, 2, 3</sup> Cerebral venous thrombosis is more common in women presumably because of pregnancy and oral contraceptive use <sup>4</sup> (about 75 percent of the adult cases are women).<sup>2</sup>

Several conditions can cause or predispose to cerebral venous thrombosis. These include following. Genetic prothrombotic states like antithrombin deficiency, protein C or S deficiency, factor V leiden mutation, prothrombin mutation. Acquired prothrombotic disorders like nephrotic syndrome, antiphospholipid antibodies, pregnancy and puerperium<sup>4</sup>, cancer, vasculitis, infections in the CNS and its neighboring structures.<sup>1, 2, 5</sup> Some drugs like oral contraceptives, hormonal replacement therapy, steroids, and oncology treatments.<sup>1,2,6</sup> The mechanical causes of sinus thrombosis are head injury, direct injury to the sinuses or the jugular veins, for instance, from jugular catheterization, neurosurgical procedures and lumbar puncture.<sup>1,2,7</sup> In our patient the risk factor was puerperium.

Presentation is highly variable. It may be a young lady having severe headache or an old man lying unconscious with dilated pupils. Most common symptoms and signs are headache (of severe intensity may mimic subarachnoid haemorrhage),<sup>2,8</sup> seizures, focal neurological deficits, altered consciousness and papilloedema which can present in isolation and in combination with others.<sup>1</sup>Our patient had most of the common symptoms.

The key to the diagnosis is the imaging of the venous system itself, which may show the occluded vessel or

A.P.M.C Vol: 3 No.2 July-December 2009

the intravascular thrombus. The current gold standard is the combination of MRI to visualise the thrombosed vessel and magnetic resonance venography to detect the non-visulisation of the same vessel.<sup>1,9</sup>

If MRI is not readily available, CT scanning is a useful technique for the initial examination, to rule our other acute cerebral disorder and to show venous infarcts or hemorrhages, but its results can also be entirely normal. CT venography is a promising new technique which has proved to be a reliable method to investigate the structure of the cerebral veins, with a reported sensitivity of 95% with Multiplanar Reformatted (MPR) images when compared with Digital Subtraction Angiography (DSA) as the standard of reference.<sup>2</sup>, Our patient's CT scan on the first day of signs and symptoms showed no abnormality and the one which was done on the 7<sup>th</sup> day of symptoms showed multiple hemorrhagic infarcts (figure 1a and b). Then we carried out CT venography which showed a thrombus in the superior sagital sinus.

Regarding treatment of the disease use of heparin has been an issue of debate for a long time. Recent guidelines have been published for the treatment of cerebral venous thrombosis, which combines treatment to specifically manage the various causes, antithrombotic treatment, and symptomatic treatment i.e. treatment of intracranial hypertension, seizures, headache. and visual failure. The aims of antithrombotic treatment are to recanalise the occluded sinus or vein, to prevent the propagation of the thrombus, and to treat the underlying prothrombotic state-in order to prevent venous thrombosis in other parts of the body, such as pulmonary embolism and to prevent the recurrence of cerebral venous thrombosis.<sup>1,2,</sup> In acute phase heparin must be give (even in the presence of hemorrhagic  $(infarcts)^2$  and patient should be orally anticoagulated. Duration of anticoagulation with warfarin should be 6-12 months aiming at an international normalized ratio of two or three.

In conclusion the diagnosis should be considered in young and middle-aged patient with recent unusual headache or with stroke-like symptoms in the absence of the usual vascular risk factors and in patients with CT evidence of hemorrhagic infarcts, especially if the infarcts are multiple and not confined to the arterial vascular territories.



Fig 1a

Fig 1b

Figure (1a and 1b) CT scan showing large Hypodense areas with small and multiple Hyperdense areas in different sections. Multiple Haemorrhagic infarcts.



Fig. 2a

Fig. 2b

Figure (2a and 2b) Images from CT Venography: arrows show thrombosis of the superior Sagital sinus.

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A.P.M.C Vol: 3 No.2 July-December 2009

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