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Case Series

Challenges in the Management of Cerebral Venous Thrombosis in a Federal Medical Center: A Report of Three Cases

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Abstract

Cerebral Vein Sinus Thrombosis (CVST) is commonly underdiagnosed due to its myriad causes and varied clinical patterns of presentation. We report 3 cases of cerebral venous sinus thrombosis all presenting with headache in our hospital.

Two (2) males and one (1) female with CVST seen at the Neurology unit at the Federal Medical Centre Owo over a five (5) year period. Their ages ranged between 31 to 59 years. The presenting symptoms were severe throbbing headache, visual impairment, proptosis, seizures, multiple cranial nerve palsies and differential limb weakness. Risk factors identified were upper respiratory tract infection, urinary tract infection, protein S and antithrombin III deficiency.

Magnetic resonance venography confirmed the diagnosis in two of the cases. The diagnosis of the third case was made on clinical judgement and a normal cranial CT scan. They all had anticoagulant therapy with SC low molecular weight heparin, tab dabigatran, antibiotics, medical cerebral decompression alongside other treatment with favourable outcome (they all survived).

This case series highlights the importance of early diagnosis and risk factors identification in the management of cerebral venous sinus thrombosis.

Keywords: Sinus; Thrombosis; Venous

Introduction

Cerebral venous sinus thrombosis is referred to as presence of clot in the cerebral venous drainage system. The cerebral venous system receives tributaries from the valveless vein of the brain. It is divided into a superficial and a deep system. The superficial system comprises sagittal sinuses and cortical veins, which drain superficial surfaces of both cerebral hemispheres. The deep system consists of the lateral sinus, straight sinus and sigmoid sinus along with draining deeper cortical veins [1]. It was thought to be a relatively rare condition accounting for 0.5% of stroke cases [2]. Recent findings report an incidence of 13 per million per year [3]. Thirty (30) and seventy (70) cases were reported in Morocco and Senegal over a period of 5 and 7 years respectively [4,5]. It is common in young female [6,7]. Risk factors could be hereditary or acquired [8]. Hereditary risk factors include causes of hypercoagulability like protein C and S and anti-thrombin III deficiency, homocysteinemia, factor V Leiden homozygous mutation [9]. Puerperium, oral contraceptives and sepsis are acquired risk factors [7,10]. We report 3 cases of cerebral sinus thrombosis in Federal Medical Centre (FMC) Owo within 5 years.

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Case Presentation

Case 1

Mr B.S was seen 2017. A 42 years old man referred from a private hospital on account of headache of month duration, drooping of the left eyelid and double vision of 5 days duration. Headache was left sided, sudden in onset, throbbing in nature, graded 8 on a severity scale of 1 to 10. Drooping of the left eyelid was noticed suddenly with double vision. Visual disturbance was severe enough to prevent him from carrying out his daily activities. No history of vomiting, convulsion or differential limb weakness. No past history of TIA or stroke.

No history of recurrent rhinorrhoea, nasal blockade, excessive sneezing, boil in the nose or around the face prior to the onset of symptoms. He had occasional palpitation but no chest pain, cough, dyspnea or orthopnea or body swelling. He was a known hypertensive patient diagnosed 3 years ago on Tab Lisinopril 5 mg daily, aspirin 75 mg daily and moduretic 1 daily with good drug compliance. Not a known diabetic or asthmatic patient. Positive history of drooping of the eyelid the previous year and was given some medications (names not known) and symptoms resolved. He neither drinks alcohol nor smoke cigarette. No family history of similar illness. General physical examination revealed a young man, conscious and alert, not pale, anicteric, acyanosed, afebrile, not dehydrated any pedal oedema.

Pertinent findings on neurological system revealed that he was conscious and alert, with left eye proptosis and Cranial Nerve III, IV, VI Palsy. Motor system was essentially normal.

Cardiovascular system revealed a pulse rate of 100 bpm, normal volume, regular blood pressure was 110/80 mmHg, Apex beat was at the left 5th Intercoastal space, mid clavicular line,.1st and 2nd heart sounds heard. No added sounds.

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No abnormality was detected in the chest or abdomen, an assessment of Left Carvenous sinus thrombosis was made. Cranial CT scan was essentially normal, MR angiography was requested for how ever patient was unable to do it due to financial constraints, FBS 4.7 mmol/L, 2 HPP 7.0 mmol/L, PCV of 39%, WBC 8,500 mm, Neutrophil 66%, Lymphocyte 30%, Eosin 2%, platelet 174,000/mm, ESR 42 mm/hr. Electrolyte, Urea and creatinine result was essentially normal, PT: 13.8 sec (12 s-16 s), INR: 1:1.1 which is normal. Treatment was commenced with IV Rocephin, IV metronidazole, SC Clexane 80 mg daily, IV dexamethasone 4 mg for 1 week then to be tailed down gradually, IV 20% mannitol 250 mg 8 hourly to and IV lasix.

Ophthalmologist reviewed on the second day and they observed left eye proptosis, pupils round and slowly reactive to light and superior disc margin obliteration. A grossly enlarged inferior turbinate with bluish tinge was noticed during the Ear nose and throat Surgeon's review. Ear drums were observed to be clear with intact tympanic membrane. An assessment of Carvenous sinus thrombosis with allergic rhinitis was made. Airway nasal spray was prescribed.

On the 4th day of admission, headache had subsided and there was improvement in the action of the extraoccular muscles.

On 7th day of admission, patient was noticed to have developed hiccups which could have been due to the raised intracranial pressure, 20% manitol and IV lasix was continued and IV metoclopramide 10 mg 8 hourly plus Tab chlorpromazine 25 mg bd for 1 week. IV Omeprazole was added on the 9th day because patient complained of epigastric pain.

He was discharged on the 13th day of admission on SC clexane 80 mg daily for 17 days, Tab dexametasone which was gradually tailed down over the next 2 weeks. Omeprazole, warfarin and antihypertensives were continued on outpatient basis. He was seen at the clinic after 1 month of discharge, he was much better though yet to do brain MR angiography or venography. He was further requested to do PT, PTTK, INR, Protein C, S, antithrombin and to continue Tab warfarin and Amlodipine. He defaulted clinic visit afterwards and subsequently lost to follow-up.

Case 2

A 31-year-old female teacher referred on account of headache 1week, fever of 2 days and 3 episodes of convulsion. Headache was generalized, throbbing relieved with the use of analgesic. Fever was noticed 5 days later which was high grade associated with chills and rigor. There was no neck stiffness, cough, difficulty in breathing dysuria, loin pain.

Jerky movement of the left upper limb was noticed. About the same time which later became generalized lasting for 5 min and aborts spontaneously. No fecal or urinary incontinence. No altered sensorium. The first episode occurred 3 hr after meal. She had another episode at admission. No vomiting, yellowness of the eye, abdominal pain or distension.

No early morning facial puffiness, excessive frothiness of urine, hiccups or reduction in urine output. She is not a known hypertensive, diabetic or asthmatic patient. No past history of Transient ischemic Attack or Cerebrovascular Accident.

She had 3 confinements which were delivered *via* Caesarian section. Last confinement was 2 months prior to presentation. There was positive history of use of hormonal implants in the past. Married in a monogamous setting. She neither drinks alcohol nor smoke cigarette. No family history of similar illness.

Examination revealed a young woman actively convulsing, not pale, anicteric, acyanosed, afebrile with temperature of 38.2°C, no pedal oedema. Neurological system examination revealed no sign of meningeal irritation and no obvious cranial nerve palsy. Motor system revealed normal muscle bulk, hypertonia and increased deep tendon reflex globally, plantar response was extensor bilaterally. There was no impairment in sensation.

Cardiovascular system examination revealed a pulse rate of 74 bpm, normal volume, regular synchronous with the contralateral side. Blood pressure was 140/90 mmHg. Apex beat was in the 5th left intercoastal space, mid clavicular line. First and second heart sounds were heard. An initial assessment of Central nervous system infection most likely cerebral abscess was made to rule out viral encephalitis. Plan was to request for brain magnetic resonance imaging (plain, contrast) with other ancillary investigations. She was placed on intravenous Rocephin, 0.9% Normal saline, dexamethasone, Intravenous phenytoin 1000 mg in 250 ml 0.9% Normal saline stat then 100 mg 8 hourly.

MRI angiography done two (2) days later showed thrombosis of the dural venous sinus, Packed Cell Volume 34%, WBC 12500/mm^3 neutrophil 62%, Lymphocytes 38%. Neutrophil showed left shift. Retroviral Screening is non-reactive. Na 146 mmol/L, K 3.9 mmol/l, HCO_3 18 mmol/L, Cl 94 mmol/L, Urea 3.9 mmol/L and creatinine 100 umol/L, PT of 13.8 secs, INR 1.2, Serum lipid profile revealed total cholesterol of 3.1 mmol/L, triglyceride 0.45 mmol/L, LDL-C 2.00 mmol/L, HDL-C 0.9 mmol/L, FBS 4.1 mmol/L, 2 HPP: 8.2 mmol/L.

Fever subsided by the 2nd day of admission, IV phenytoin was discontinued, and tab Levitiracetam 250 mg bd, SC clexane 80 mg daily were added. Levitiracetam was increased to 500 mg bd because patient had another episode of seizure. She took subcutaneous clexane 80 mg daily for 2 weeks and afterwards changed to Tab dabigatran110 mg bd. Intravenous dexametasone was tailed down gradually over a period of 2 weeks.

Urine and catheter tip microscopic culture and sensitivity yielded growth of Escherichia coli sensitive to nitrofurantoin. Urinalysis revealed nitrite and blood of 1plus. Tab nitrofurantoin 100 mg 6 hourly was added to the medication. She was discharged after 2 weeks on admission on tab Tab dabigatran 110 mg bd. Keppra was tailed down over the following week. She was seen at the clinic after 2 weeks. The results of some investigations done during follow up visit are as follows Protein S=16.4% (50%-140%), Antithrombin 70% (80%-120%), protein C: 78% (70-140). She was to continue dabigatran 110 mg bd to achieve a target INR of 2-3. At the next clinic, INR was 1.2 and Tab dabigatran was increased to 150 mg bd. At the last clinic visit she was still on tab dabigatran and she is presently being co-managed with the haematologist.

Case 3

A 59-year-old civil servant who presented with left sided body weakness and loss of consciousness that occurred 3 hr prior to presentation. Left sided weakness was noticed on waking up from bed in the morning. Sensorium was also noticed to be fluctuating. There was preceding history of headache of 3 days duration which was insiduos in onset, generalized and temporarily relieved with the use of paracetamol. There was positive history of slurred speech. No

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vomiting, neck pain or stiffness, no convulsion, no history of trauma to the head. No blurring of vision or double vision. No history of fever or nasal discharge. Not a previously known hypertensive, diabetes or seizure disorder patient. No past history of transient ischemic attack or cerebrovascular accident. He was married in a monogamous setting with 3 children. He neither drinks alcohol nor smoke cigarette. No family history of hypertension, diabetes or asthma.

General physical examination revealed a middle-aged man, drowsy not pale, anicteric, acyanosed, afebrile, not dehydrated, no pedal oedema. Neurological system revealed Glasgow Coma Score of 14/15, there was no sign of meningeal irritation, pupils were 4 mm in size bilaterally and reactive to both direct and consensual light reflex. There was no abnormality of cranial nerve 3, 4, 6, 9, 10. There were left cranial nerve 7, 12 palsy. No abnormality in speech and no sign of meningeal irritation.

Motor system revealed normal motor bulk globally, power of zero reduced tone and reflexes in the left upper limb and left lower limb absent pantar response and impaired sensation on the same side. Power was 5 on the right upper and lower limb, tone and reflexes were normal on the right upper and lower limbs.

Cardiovascular system revealed pulse rate was 76 beats per minutes, normal volume, regular, blood pressure of 130/80 mmhg, apex beat 5th left intercoastal space, mid clavicular line. First and second heart sounds were heard. Review of the respiratory system showed respiratory rate of 24 cycles per minutes with vesicular breath sounds. No abnormality detected on the abdominal examination.

Cranial computerized tomography scan done revealed area of hyperdensity mixed with surrounding hypodensity in keeping with right parietal haemorrhage due to intracranial space occupying lesion with intra tumoral bleed or haemorrhagic transformation of a large parietal infarction. Brain Magnetic resonance imaging/ Angiography, Full blood count, PT, PTTK, INR, Chest radiograph,12 leads Electrocardiography, Echocardiography, vertebral and carotid doppler ultrasound, Electrolyte, urea and creatinie, serum calcium, phosphate and uric acid were requested for.

Cerebral decompression was done with Intravenous 20% mannitol, Lasix, 0.9% N/saline, ceftriazone, 50% Dextrose water was given for caloric support. Consult was written to the speech therapist, physiotherapy and neurosurgeon were invited.

Neurosurgeons review of the cranial computerized tomography scan is that of a right temporooccipital intracerebral haemorrhage with perilesional edema, right hemispheric brain swelling and partial effacement of the right lateral ventricle and they made an impression of right spontaneous intracerebral haemorrhage? aetiology and based on this some investigations were requested for and the results are as follows serum d dimer 10,000 ng/ml (1 ng/ml-500 ng/ml), PT, PTTK, INR, Sodium 135, potassium, 3.1, chlorine: 110, bicarbonate: 15, Urea 6.7, creatinine: 142 umol/l, Calcium 2.52 mmol/L, (normal), Phosphate 8.5 mmol/L(N) Albumin 34 g/L(N), uric acid 0.25 PCV 48%, WBC 21,000/cmm^3, neutrophils 48%, lymph 46%. Platelets 258,000/mm^3 PT was 16.2 sec (12s-16s). Brain magnetic resonance angiography and venography were requested because of the elevated serum d- dimer raised a suspicion of a prothrombotic state. Plain Brain Magnetic resonance imaging done revealed right parietal hemorrhagic CVA with significant perilesional edema Repeat D dimer was still >10,000 ng/ml one week later.

Brain magnetic resonance venogram done after two weeks on admission showed reduced caliber of the left transverse sinus with non-outlining of the left sigmoid sinus and internal jugular vein. An assessment of? cormobid cerebral venous sinus thrombosis with? Haemorrhagic transformation of cerebral infarct was made. SC clexane 40 mg was added. Haematologist reviewed and another clotting profile was requested for. Tab dabigatran was to be added to the drug regimen after subcutaneous low molecular weight heparin has been used for 5 days.

Intravenous dexamethasone was subsequently tailed down over the following 2 weeks. Repeat INR was 1.8. Power on the Left upper and lower limb improved gradually to MRC grade 3 and patient was encouraged to sit out of bed. He was discharged on the 29th day of admission on Tab dabigatran 150 mg bd. He has been seen twice at the clinic since discharge and power on the left upper limb has improved to MRC grade 4 and 5 on the left lower limb (Figures 1 and 2).

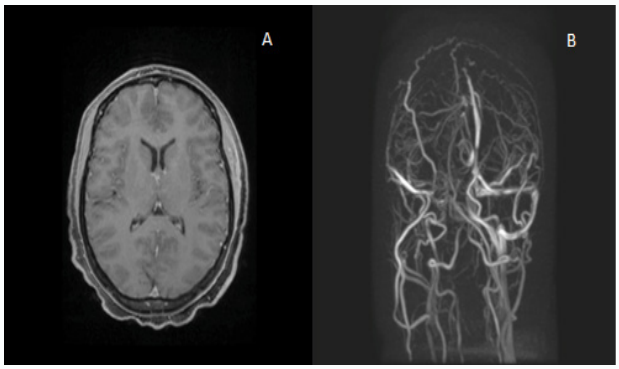

Figure 1: (A) Axial T1 brain MRI with contrast of the second patient. (B) MRV of the same patient showing thrombosis of dural venous sinus.

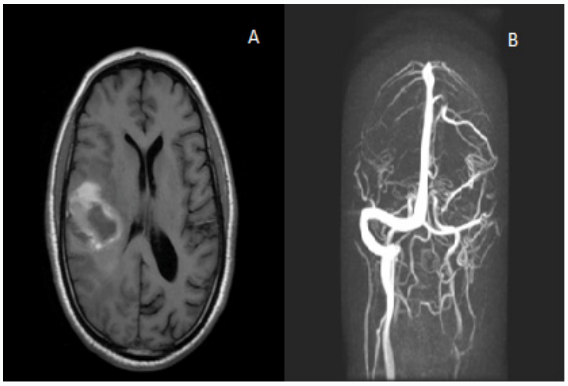

Figure 2: (A) Axial T1 brain MRI of the third patient showing right parietal hemorrhagic CVA with significant perilesional edema (B) MRV of the same patient showing showed reduced caliber of the left transverse sinus with non-outlining of the left sigmoid sinus and internal jugular vein.

Discussion

Cerebral venous thrombosis is most likely to occur in adults younger than 45 years old [10]. The commonest clinical feature in our study was headache similar to the reports in other studies [5,6]. Others include visual impairment, proptosis, seizures, left hemiplegia/ hemiparesis, cranial nerve palsy which could have resulted from of intracranial hypertension and focal neurological syndrome [10,11]. Risk factors in our report include rhinitis and urinary tract infection. Infection has been identified as the commonest risk factor in sub-

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Saharan Africa [5,12]. Protein S and antithrombin III deficiencies were identified in the second patient.

Imaging is key in making the diagnosis of this condition because it has so many differentials. The preferred and most sensitive diagnostic investigation is Magnetic Resonance Venography [13]. It helps to identify areas of venous occlusion, infarction and consequent cerebral oedema [14]. The first patient couldn't do this due to financial constraints while others did. Dural venous sinus, transverse sinus and sigmoid sinus were affected in our study. Superior sagittal sinus and the transverse sinuses are the commonly affected [7,11]. One of our patients had a serum d-dimer of 10,000 ng/ml which increased our suspicion of thrombosis. Sidhom et al. [10] reported a positive D dimer result in 46% of his study population. The evidence-based value of using a low d dimer in conjunction with a low clinical suspicion to rule out DVT of the lower extremity and for PE is less established in cerebral venous sinus thrombosis [15]. More studies should be done in this regard so as to develop an algorithm for investigating patients with CVST especially in resource poor settings like ours. This same patient had haemorrhagic transformation of a cerebral infarct co-existing with CVST Other studies reported ICH as a presentation in CVST [7,16,17]. Rupture of small vessel as a result of increased capillary and venous pressure from blocked venous sinuses could explain this [18].

Anticoagulation forms the mainstay of treatment [14], Low molecular weight heparin was given to our patients and they showed clinical improvement afterwards. They were discharged on tab dabigatran with the INR target of 2-3 for 6 months. The patient with protein S and antithrombin deficiency will be on anticoagulant for life and she is being comanaged with the Haematologist. Intravenous dexamethasone or mannitol was given to reduce the intracranial pressure. Seizure was managed in the second patient with Intravenous phenytoin as she presented in status epilepticus and later continued with tab Levitiracetam which was tapered gradually till it was discontinued. Intravenous antibiotics were administered in all the patients. Prognosis of CVST is good as evidenced in our study and International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT) [19]. Making the diagnosis was quite challenging as we depended on our clinical acumen in the first patient due to lack of funds for MRV after a normal cranial CT scan. The other patients had to travel to the neighbouring state for MRV because it is not available in our Centre leading to a delay in making the diagnosis and commencement of anticoagulant. Hereditary risk factors for CVST could not be exhausted as only one of our patients was able to screen for thrombophilia disorders during follow up which revealed Protein S and antithrombin deficiency, others could not. Studies have identified the above listed factors as the reasons for underreporting of CVST in sub-Saharan Africa giving a false impression that it is a rare disease [5,12].

Conclusion

We concluded that CVST is not as rare in sub-Saharan Africa as it was initially thought to be. The diagnosis could be challenging because it has many differentials and facilities for making the diagnosis are not readily available. We recommend that more studies be done along this line that could lead to development of algorithm for management of CVST. Tertiary centres in the country should be well equipped with neuroimaging techniques to aid in the proper management of patients.

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