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Case Report

Stroke in Sickle Cell Disease in Tanzania: Case Report

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Abstract: Stroke, is a significant cause of morbidity and mortality in sickle cell disease, occurring with an incidence of 10 to 25%. The risk of Stoke is highest during the first decade of life, Extensive research has established that cerebral stenosis, involving the circle of Willis, is the most common mechanism in children. A report of 11-year-old boy with Sickle Cell Anemia presented with history of left sided hemiplegia for 3 weeks that was of sudden onset. Computerized tomography of the brain revealed an increasing sulcal space more marked in frontal lobes associated with hypo attenuating area in the right frontal lobe. Features suggestive of Brain atrophy and right frontal lobe Ischemic.
Keywords: Sickle Cell Stroke Stroke In Sickle Cell Disease Pediatric Sickle Cell Disease.

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INTRODUCTION

Sickle cell disease (SCD) is an autosomal recessive disorder of the RBC in which individual who are homozygous acquiring traits from both parents, suffers sickled RBC who presents with a number of complications when subjected to conditions like hypoxia, dehydration and infections. Although the Sickle Cell gene mutation is most common in Equatorial Africa, it is also found in the Mediterranean regions of Europe and Turkey. The condition has now become prevalent worldwide due to the migration of people of various types of genetic makeup across the globe. However, the condition is much more observed in areas where malarial outbreaks are common [1].

Although stroke may be due to hemorrhage, embolism or infarction, heart conditions it has been found that cerebral infarction is the most common mechanism in children and young adults below 20 years and is thought to be the result of an occlusive vasculopathy predominantly affecting the large intracranial vessels in the circle of Willis [2, 3]. Strokes of either types i.e. ischemic and hemorrhagic strokes are not uncommon in pediatric patients having sickle cell disease which can also happen in adult patients. In addition to stroke, critical neurological signs and symptoms that are common in SCD include transient ischemic attack (TIA), seizures, headaches, and coma.

According to the World Health Organisation (WHO), a stroke is defined as a focused neurological impairment lasting more than 24 hours. On the other hand, a focal neurological impairments that last less than one day, even though acute neuroimaging may show abnormalities are termed as Transient Ischemic Attacks (TIA). Both symptomatic and asymptomatic cerebral infarctions (silent or overt) are possible.

CASE PRESENTATION

11year-old boy with a known history of sickle cell Anaemia on irregular clinic attendance at National Hospital, with poor adherence of Hydroxyurea tablets, he was on daily Folic Acid 5mg. Presented to Regional Referral Hospital with left sided body weakness (hemiplegia) for 3 weeks that was of sudden onset, involving the left upper and lower limbs, with mouth deviation to the right side, however no history of loss of consciousness or convulsion, no history of fever, headache or trauma prior to the onset of the presentations. For the last eight month patient presented with convulsions which was generalized, tonic clonic in nature, no history of loss of consciousness, frothing and

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jerky movements were not observed. The patient has history of two admissions no history of blood transfusions, no history of joints or bone pain.

On physical examination he was Alert, some pallor, not jaundice. He had a regular pulse rate of 88 beats per minute, S1 and S2 heard with no murmurs, SPO2 98% in room air, Chest was clear, the abdomen was soft no organomegally. Normal bowel sound, on neurological examination the patient has Facial asymmetry, difficulty word articulation. no tongue deviation, the power The power was 3/5 on the left lower limb, 0/5 on the upper left limb with decreased muscle tone, Deep tendon testing showed hyper reflexion with upcoming planter reflexes. Power of 5/5 on the right lower and upper limb.

The full blood count showed hemoglobin of 8.1g/dl, and a normal white cell count and differential distribution. CT scan of the brain revealed Ischemia of right lobe and Brain atrophy

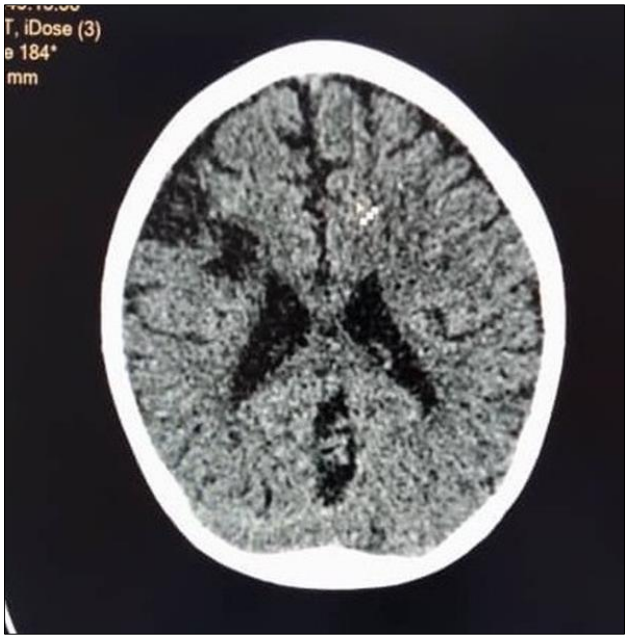

Figure 1: CT scan of the Brain showing increasing sulcal space, more marked in frontal lobes associated with hypo attenuating area in the right frontal lobe. Features suggestive of Brain atrophy and right frontal lobe ischemia

DISCUSSION

Among the complications of sickle cell disease, stroke is one of the most serious, especially in children. Acute neurological impairments such as hemiplegia, facial asymmetry, speech problems, seizures, and less frequently, altered mentation or coma are characteristic of the clinical presentation [1-4]. In the current case, A 11-year-old male with a known history of SCD, presented with left-sided hemiplegia of sudden onset, associated with facial asymmetry and speech difficulties, which are consistent with a right hemispheric infarct.

According to DeBaun *et al*., (2014), Ischemic stroke is the most common type of stroke in children with SCD, accounting for more than 75% of cases. It results from large vessel vasculopathy, which specifically affects the circle of Willis, leading to cerebral infarction [5]. Though Brain MRI is preferred in detect cerebral infarct or hemorrhage [6]. In our setting CT scan was readily available therefore we used to differentiate between the two types of stroke.

The patient's history of generalized tonic-clonic convulsions during the previous months indicated some of the additional neurological symptoms that may be present in SCD, including seizures, headaches, transient ischemic attacks (TIAs), and cognitive impairment [7]. Moreover, seizures reported to be 10 times more prevalent in patient with SCD compared to general populatin [8].

A low haemoglobin level causes sickling and hinders the delivery of oxygen to tissues, particularly in the cerebral vasculature. Our patient's haemoglobin was 8.1g/dl, which is in line with literature showing anaemia contributes to cerebral hypoxia in SCD patients and increases the risk of stroke [5-9]. Prompt blood transfusion initiation to lower hemoglobin S (HbS) proportion and enhance oxygen delivery is the cornerstone of acute stroke management in sickle cell disease (SCD). There was no possibility of Exchange transfusion therefore, we perform a simple transfusion aiming not to exceed Hb of 10g/dl. Following transfusion, the child in this instance displayed clinical improvement, which is in line with earlier research demonstrating the advantages of transfusion in acute stroke and its function in secondary prevention [2-10].

Although seizures related to stroke carry a lower risk of subsequent epilepsy, based on our patient's history, there was a strong suspicion of recurrent seizures, which justified prescribing valproic acid [11].

Reduction of cerebrovascular complications and improvement in quality of life have been reported in SCD patients using hydroxyurea, which increases fetal haemoglobin (HbF) levels and decreases hemolysis and vaso-occlusive crises [12, 13], however, poor adherence to hydroxyurea and irregular clinic visits were likely contributing factors in the child's stroke event.

Exchange transfusion shown to be superior over simple transfusion in preventing secondary stroke [9]. However up to date there is no clinical evidence showing differences in outcome between exchange transfusion and simple transfusion in managing acute stroke.

Transcranial Doppler ultrasonography is an essential non-invasive screening method that is recommended for all children with sickle cell disease (SCD) between the ages of 2 and 16 in order to identify those who are at high risk of stroke (>200 cm/s velocities) warrant a chronic transfusion therapy [2-13]. The lack of such screening in this case, likely due to limited resources, points to a gap in stroke prevention strategies in low-resource settings like Tanzania [3].

Clinical improvement following stoke depends on extent of injury, timing in intervention and accessibility of rehabilitation services. Our patient shows clinical improvement few days post transfusion, however in the long run, the prognosis is uncertain due to possibilities of permanent neurological damage and the risk of recurrent stroke. Moreover, early physiotherapy and continuous follow-up are crucial in maximizing the recovery and prevention of further neurological complications.

CONCLUSION

In this case we have observed the classical presentation of overt stroke in pediatrics with sickle cell disease for instance; focal neurological deficit and supportive evidence of cerebral infarct show in Brain CT scan. Moreover, we have learnt that; lack of preventive measures, poor adherence to clinic and hydroxyurea are the among challenges in managing sickle cell disease in resource limited setting.

Enhancing comprehensive care including patient education, and increasing accessibility of hydroxyurea and Trans Doppler ultrasound will improve patient outcomes and substantially reduce morbidity and mortality attributed to sickle cell stroke.

Stroke in SCD is multifactorial, but high-risk individuals can be identified by simple well-established strategies such as transcranial Doppler ultrasonography. There are approaches for both primary and secondary interventions, which have been shown to be effective and need to be incorporated into management guidelines for SCD patients. Before schemes are recommended into health care policies, research in the appropriate setting is required.

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