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Intracranial Arteriovenous Malformations and Stroke in Neonate: A Case Report

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Summary: Stroke in infants and children is an important cause of morbidity and mortality and an emerging area for clinical and translational research. The clinical presentation of neonatal stroke is subtle and varied, which presents challenges in diagnosis as well as ascertainment of risk factors and outcome [1].

Hemorrhagic stroke is less common than ischemic stroke in children [2,3]. Several risk factors for hemorrhagic stroke have been identified in children, including vascular malformations, malignancy, and trauma. Hemorrhagic stroke in children is also associated with primary and secondary coagulation disorders including hemophilia, thrombocytopenia, liver failure, leukemia, and warfarin therapy [4,5]. Arteriovenous Malformations (AVM) are the most common cause of hemorrhagic stroke in children. The development of an AVM results from failure in the formation of the capillary bed between primitive arteries and veins in the brain during the first trimester of fetal life. The incidence of AVM in children is 1/100 000 and approximately 10% to 20% of all AVMs will become symptomatic during childhood [4-9].

Case Report

The male child was delivered with normal body weight after 37 weeks of gestation. The physical examination after birth did not reveal any abnormality. Gestation and delivery had been uneventful. There were no risk factors. After 24 hours, the child spontaneously developed clonic seizures of the right side of the body involving both arm and leg.

Trans fontanel ultrasound and lumbar puncture were normal, ruling out intracranial hemorrhage or meningitis. Laboratory finding showed a leukocyte count of 5500/mm3, with differentials of 41% neutrophils, 54% lymphocytes, 4% monocytes, and 1% eosinophils. The hemoglobin level and platelet count were normal. Serum electrolytes, creatinine, albumin, total bilirubin, protein, and osmolality levels were normal. metabolic study and serum homocysteine were normal.

Factor V Leiden G506 was normal, MTHFR C677, MTHFR A1298C, Prothrombin G20210A were normal. PT, PTT BT, CT, PLT were normal. Brain MRI revealed there was evidence of an area of hypo density of the right parietal region consisting of tubular structure representing vascular malformation (Figure 1).

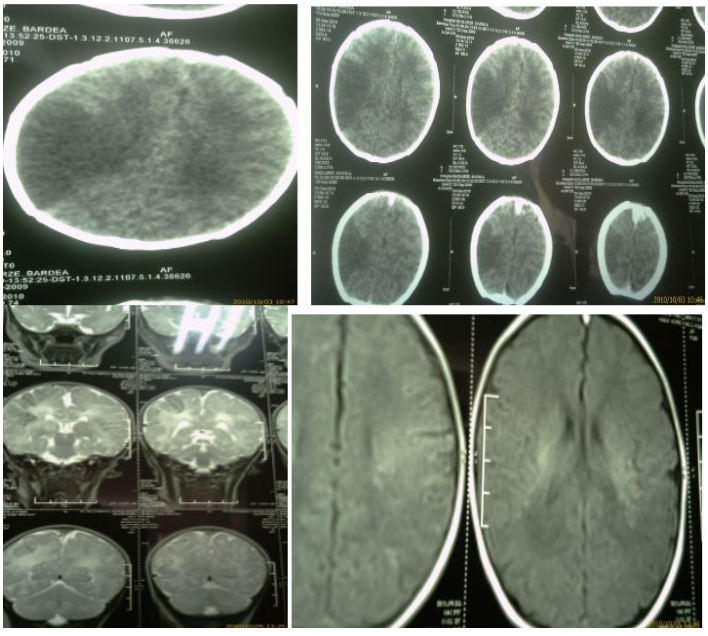
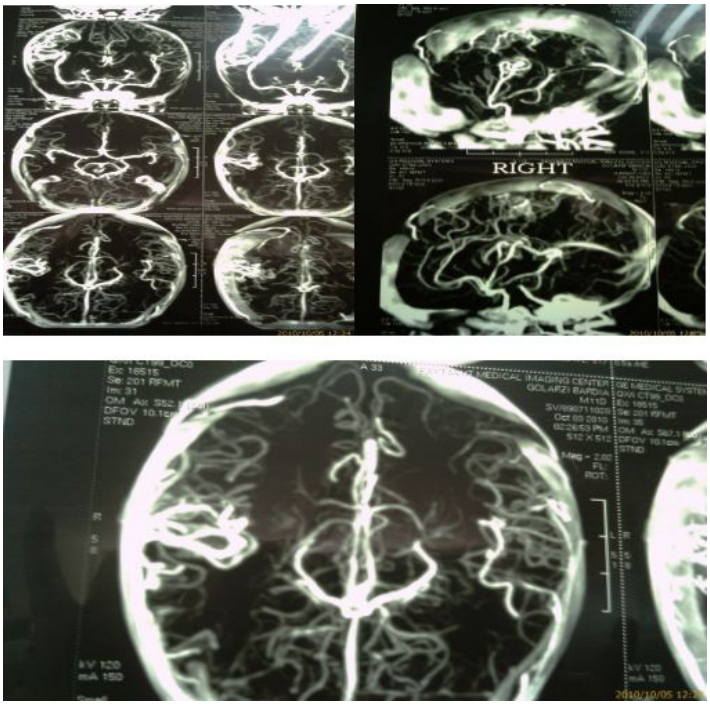


Figure 1: Brain MRI.

CT Angiography revealed there was evidence of Dural based vascular malformation representing AVM of the right hemisphere (Figure 2).



Discussion

The incidence of neonatal stroke has been estimated to be in the range of 1 in 4000 deliveries [7]. The causes of neonatal stroke often remain obscure, although many reasons for neonatal infarctions have been reported. Stroke in neonates often presents clinically with seizures but not with focal neurological deficits such as hemiparesis [2-9].

Arteriovenous Malformations (AVM) are the most common cause of hemorrhagic stroke in children. The development of an AVM results from failure in the formation of the capillary bed between primitive arteries and veins in the brain during the first trimester of fetal life [10]. The incidence of AVM in children is 1/100 000 and approximately 10% to 20% of all AVMs will become symptomatic during childhood [3,6]. The average probability of a first hemorrhage is 2% to 4% per year, with a recurrence risk as high as 25% by 5 years.44 Magnetic Resonance (MR) imaging and MRA confirm the diagnosis of AVM [4,5,7,8]. Approximately 1% to 2% of aneurysms become symptomatic during childhood. Aneurysms in children are typically associated with other vascular lesions or chronic disorders. Cavernous malformations can also lead to hemorrhagic stroke in children. One third of cavernous malformations are familial. Cavernous malformations have recently been linked to abnormalities in the long arm of chromosome 7.4 [4,5,7-11].

The risk of recurrent intracranial hemorrhage is slightly elevated for a short period of time after the first hemorrhage. In 2 studies [12] the risk during the first year after initial hemorrhage was 6% and then dropped to the baseline rate, whereas in another study [13], risk of recurrence during the first year was 17.9%. The risk of recurrent hemorrhage may be even higher in the first year after the second hemorrhage and has been reported to be 25% during that year [10-17].

At present, there are 4 major treatment options available for patients with an AVM of the brain. The lesion can be monitored expectantly with the understanding that the patient would have some risk of hemorrhage or other neurological symptoms such as seizures or focal deficit. Alternatively, intervention can be undertaken with the goal of complete AVM obliteration, because subtotal therapy does not confer protection from hemorrhage. Management strategies include single or combined therapy applying microsurgery, endovascular techniques, or radiosurgery (Focused Radiation). Each treatment option has associated risks and benefits [13,15]. AVMs are often treated by more than one treatment modality. This occurs in one of two fashions. It is done as either a planned maneuver, typically with embolization followed by surgical resection or radiosurgery, or as an unplanned maneuver where one treatment modality fails and a second treatment modality is necessary to obliterate the AVM. This can occur in situations such as residual AVM after subtotalling surgical resection or resection of an AVM after incomplete radio surgical treatment [8,9,11,16].

Outcomes of treatment in subsequent sections generally include associated mortality and morbidity, although these are not reported consistently.

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