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INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI:10.21474/IJAR01/12649
DOI URL: http://dx.doi.org/10.21474/IJAR01/12649

RESEARCH ARTICLE

ISCHEMIC STROKE IN A 3-YEAR-OLD CHILD REVEALING THROMBOSIS OF THE VERTEBROBASILAR TERRITORY, A CASE REPORT

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Manuscript Info

Manuscript History
Received: 05 February 2021
Final Accepted: 10 March 2021
Published: April 2021

Key words:-
Ischemic Stroke, Child, Thrombosis Of The Vertebrobasilar Territory, Coagulopathy

Abstract

Ischemic stroke in children is a rare occurrence. The causes are very varied and different from those of adults. Hematogenouscausesare an important source of ischemic stroke in children to look for. Imaging plays a major role in the positive and topographic diagnosis. It also makes it possible to identify the cause in certain cases and to guide treatment.

Introduction:-

The ischemic attack is due to a focal alteration of the arterial perfusion, causing cerebral damage and lasting sequelae. Ischemic stroke in children is a rare phenomenon; the incidence is estimated at 1-2 / 100,000 children per year [1]. The causes are very varied and different from those of adults. Coagulopathiesare an important source of ischemic stroke in children to look for. Imaging has a major role in positive and topographic diagnosis, it also makes it possible to identify the cause and guide treatment. We report the case of an ischemic stroke by thrombosis of the vertebrobasilar territory in a three-year-old child with coagulopathy.

Case Report:

We report the case of a 3-year-old boy, with no particular pathological history, who presented to the pediatric emergency ward for a fever of 38°C associated with hypotonia and vomiting, onset rapidly for 5 days, The child was initially put on antibiotic therapy. The evolution was marked by the appearance of disorders of consciousness without convulsions, neither motor, nor sensory deficit. On clinical examination: altered consciousness(GCS 9/15), presenting anisocoria (right areactive mydriasis), hemodynamically and respiratory stable (Fc: 120 bpm, BP: 95/60 mmgh, Fr: 22cpm, SpO2: 95% in ambient air), a temperature of 38.9 ° C and a capillary glycemia of 0.85 g / l, the neurological examination found a pyramidal syndrome without meningeal syndrome. The remainder of the clinical examination was unremarkable. After conditioning the child was put on oxygen therapy, antibiotics and antipyretics and underwent a biological assessment and imaging.

The lumbar puncture objectified a glycorachiaat 0.7 compared to the glycemia, albuminorachiaat 0.17g / l, Cytology: clear appearance, <03 elements, RBCs at 14 and no germs on direct examination. CBC: Hb at 11.3, Ht at 32.7%, GB at 14.320 g/l, PMNs at 9640 /mm3 and PLT: 372.000/mm3. PT at 100% and KCT at 27.Ionogram and renal function correct and CRP negative. ECBU: sterile culture. Blood culture: sterile.

The brain scan helpedto make the diagnosis of bilateral ischemic stroke of the superficial and deep territories of the posterior cerebral arteries and the encephalic MRI confirmed the recent ischemic stroke extended to the vertebra-basilar territory with arterial thrombus of the basilar trunk.

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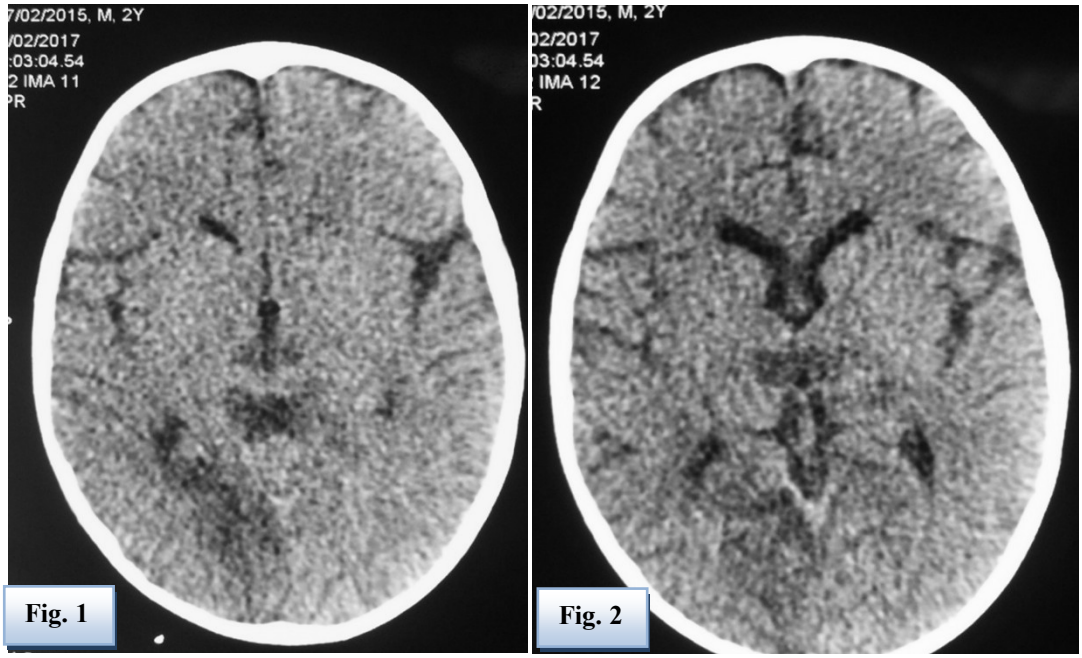


Figures 1 and 2:- Cerebral CT in axial section without injection of PDC in the parenchymal window showing bilateral occipital hypodensity areas of ischemic vascular appearance involving the superficial and deep territories of the posterior cerebral arteries.

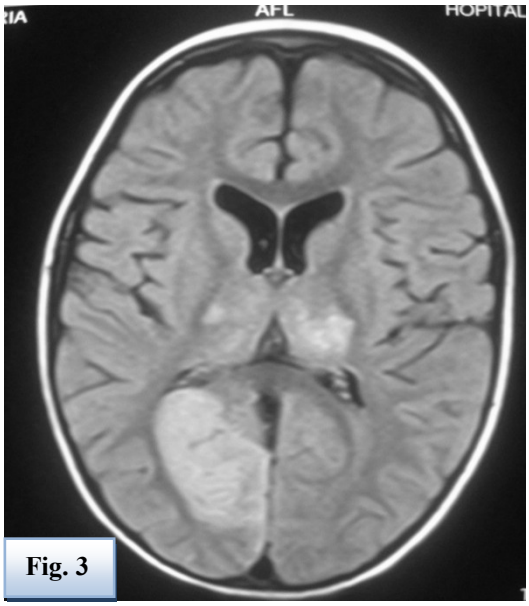
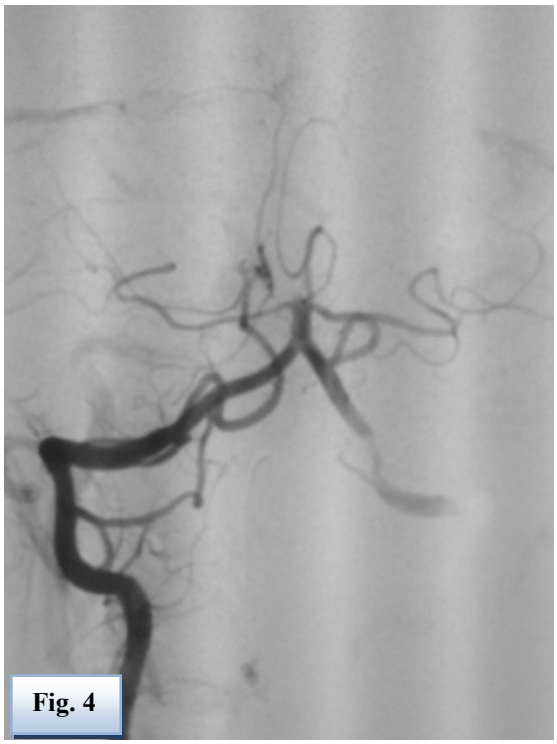
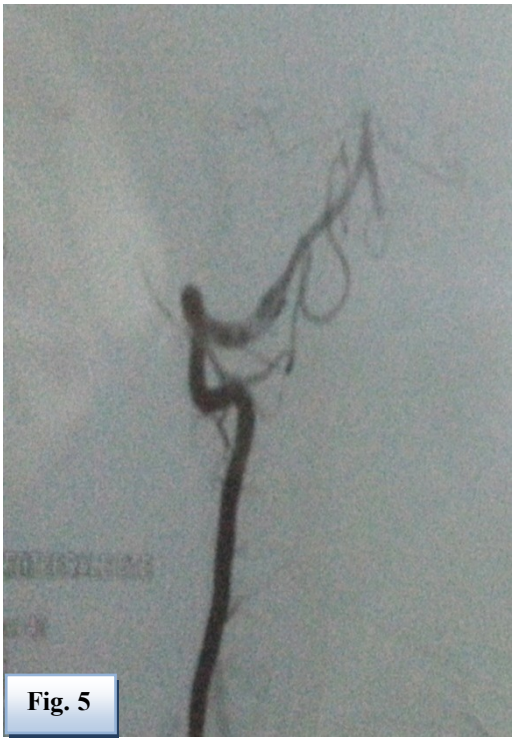


Figure 3:- Brain MRI in axial slices showing ischemic areas in hypersignal FLAIR.

As part of the aetiological assessment, a cerebral arteriography was performed which revealed a complete post-ostial occlusion of the left vertebral part partially extended to segment V2 and an occlusion of the basilar trunk just after the ostia of the two AICAs without visualization of the two superior cerebellar and two posterior cerebellar.





Figures 4 and 5:- Cerebral arteriography demonstrating a complete post-ostial occlusion of the left vertebral column partially extended to segment V2 and an occlusion of the basilar trunk just after the ostia of the two AICAs without visualization of the two uppercerebellar and the two posterior cerebrals.

A thrombophilia workup revealed resistance to activated protein c 162 sec (Leiden factor V mutation) with: Antithrombin III 105%; 73% Prot C and 48% Prot S.

The child received partial aspiration of the thrombus and was then put on heparin therapy and antiplatelet therapy. The evolution was marked by improvement on the neurological level (GCS at 12/15) with transfer to the pediatric department.

Discussion:-

Ischemic stroke is extremely rare in the pediatric population, with an incidence of less than 2 to 3 per 100,000. [2]

We report the case of a 3-year-old child, in whom the initial symptomatology was not very suggestive of an ischemic stroke. In general, the clinical symptomatology in children is very variable. Acute hemiplegia is the usual mode of disclosure. It is rather flaccid and predominates in the upper limb. Other symptoms may occur such as: speech disorders, headache, vomiting, disturbances in consciousness, impaired alertness and epileptic seizures. When the ischemic phenomenon involves the vertebrobasilar territories, the warning signs can be visual, ranging from a simple blur to blindness if the attack is bilateral; Cerebellar involvement responsible for acute ataxia may be associated.[3,4]

The aetiologies are multiple but rare; most often the cause is not found and the stroke does not recur. [5]

Three main causes are often intertwined: Arterial disease (focal cerebral, post varicella or dissection of the cerebral arteries), heart disease (congenital and cardiac arrhythmias) and hematogenous causes [6].hematogenouscauses are an important cause of stroke in children; There are four categories: [7,8,9]Hemoglobinopathies with sickle cell anemia in the first place; Hypercoagulopathies; Myeloproliferative and myeloblastic syndromes and dysimmunity causes.

Hypercoagulopathies result from a congenital deficit of physiological antithrombotic proteins. It is, in particular, the deficit in proteins C and S, in antithrombin III, in heparin cofactor II, in factor V Leiden, in plasminogen, in

activator of tissue plasminogen or of an excess of inhibitor of plasminogen activator and activated protein C resistance. The cause of ischemic stroke in our patient was an activated protein C resistance-type coagulopathy.

Activated protein C splits factor Va at three sites: Arg306, Arg506, Arg679 with a consequent deceleration of the coagulation cascade. Dahlbäck et al [10] identified a plasma, in 1993,a resistance to the anticoagulant activity of activated protein C. In 1994, Bertina et al. [11] linked this resistance to the presence of a mutation on factor V, or V Leiden, which corresponds to the substitution of Arg 506 by a glutamine. This mutated factor V becomes an obstacle to effective inhibition of the coagulation cascade by activated protein C.

It should be noted that a transitory deficit of these factors is possible at the time of the stroke without it being directly at the origin. Thus, the interpretation of these results is often delicate and requires the help of a hematologist expert in hemostasis.

The positive diagnosis of ischemic stroke is based on imaging. Thus, a brain scan should be performed urgently. It help to confirm the existence of a stroke and sometimes to specify the topographical diagnosis.However, when the examination is performed within the first few hours, the extent of ischemia may be underestimated [5].However MRIis the reference examination, it detects the affected areas early, provides topographical data complementary to the positive diagnosis and allows to largely unravel the etiological investigation [5].

In our patient, CT and MRI made it possible to easily make the positive diagnosis of stroke and to specify the topography.

The etiological explorations help for a better and accurate treatment of the cause. These include MRI angiography, cerebral arteriography, cerebral ultrasound (before the fontanel close), cardiac ultrasound and blood tests.[6].

In our case, the cerebral arteriography helpedto specify the site and the extent of the arterial occlusion; it also helped to perform partial aspiration of the thrombus. As for the blood test, it allowed the etiological diagnosis.

The diagnosis of factor V resistance to activated protein C (APC) is based on: A functional assay of plasma coagulation and analysis of the factor V gene.

The treatment of these strokes is mainly based on the treatment of the cause and on anticoagulants. Sometimes surgery is necessary to remove the clot.

Conclusion:-

Early diagnosis of childhood stroke is difficult because of the variability of onset symptoms mimicking other pediatric illnesses. The clinical diagnosis is the key moment; It is used to trigger additional examinations urgently. MRI or brain scan confirm the diagnosis, look for the cause and direct treatment. Any child suffering from a stroke, even if the initial clinical recovery appears rapid and complete, must benefit from an exhaustive etiological assessment.Theaetiologies are much more numerous and varied than in adults and coagulopathiesare an important cause of stroke in children to look for. The etiological investigation should ideally be carried out in a specialized pediatric setting.

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