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Case Report

Ischemic Stroke in Childhood; Case Report

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Abstract

Pediatric stroke is a severe pathology, with lasting devastating consequences on motor and cognitive abilities, school and social integration, and overall life projects.

Awareness of initial symptoms, implementation of pediatric stroke code protocols using MRI first and only and adapted management in the acute phase, personalized recanalization treatment strategies and multidisciplinary rehabilitation programs with specific goal-focused actions are key elements to improve pediatric AIS management and outcomes. We report here the case of a 1-year-old child with Avci with optimal management.

Keywords: Stroke; Children; Thrombolysis; Focal cerebral arteriopathy rehabilitation

Introduction

Ischemic strokes correspond to focal neurological suffering of vascular origin lasting at least 24 hours. Childhood ischemic strokes are 10 to 12 times rarer. Their incidence has been estimated at 3.3/100,000 children/year. There are many etiologies, and unlike in adults, atheroma is not the dominant cause of stroke. Despite this diversity of causes, the majority of arterial infarctions in children are grouped into a few nosological frameworks: embolic infarctions of cardiac, infectious, inflammatory, hematological, metabolic or genetic origin. The etiological diagnosis is guided by clinical elements and simple imaging, which can be supplemented by serial investigations in the absence of a known terrain. As identification of the cause can modify treatment and prognosis, an exhaustive etiological work-up must be systematically carried out, not only to find the cause, but above all to assess the risk of recurrence, establish the prognosis and propose an appropriate treatment. The aim of our work is to propose a practical strategy for evoking the positive and etiological diagnoses of ischemic stroke in children, in order to enable the prognosis to be formulated, and the appropriate therapeutic course to be determined, through observation in the intensive care unit of the CHU Souss Massa agadir Hassan II hospital.

Patient Information

The patient was a 1-year-old male with the following history

- No particular viral infection during childhood.
- No history of head trauma.
- No history of epilepsy in childhood.
- No history of bronchitis or hospitalization for respiratory pathology.
- No history of chickenpox.
- No notion of any other particular rash during childhood.
- Vaccination schedule up to date according to the family, with no notion of accidents or side effects.
- No recent vaccinations other than COVID-19.

Clinical Findings

The history of the disease dates back to 12/17/2021 at midnight with the onset of partial convulsive seizures in a febrile context (fever not quantified) lasting more than 30 min, with loss of consciousness postcritically then regained consciousness put on Gardenal maintenance and loading dose, the morning of 12/18/2022 he presented partial convulsive seizures lasting more than 10 min hence his admission to intensive care for further management, arriving from the pediatric ward.

Timeline

- Patient unconscious, not oriented in time and space,
- GCS = 8/15 ; V :1 Y :2 M :5
- Pupil with reactive right anisocoria (mydriasis right / left normal)
- Left hemiplegia + right facial paralysis.

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- Hemodynamically and respiratory stable.

BP = 119/76mmHg HR = 89bpm FR= 28 cpm SaO2= 98% T°= 37°

- Normal-coloured conjunctiva
- No IMO, no dehydration folds.

Standing impossible

- Walking impossible
- Global muscular strength: patient cannot hold the barré or the mingazzini
- Segmental muscle strength:

o 1/5 at MIG proximo-distally

o 4/5 at MID proximo-distally

o 1/5 at the 2 MS proximo-distally

- Muscle tone: presence of peripheral hypotonia
- ROT abolished in MIG, normal in MS and right MI
- RCP
- Presence of sensory level in all modes
- Examination of cranial pairs: presence of right facial paralysis.
- Convulsions stopped.
- Left-sided hemiplegia, left hemiparesis.
- Pupil with reactive right anisocoria (mydriasis right/left normal).

Thorax normal in appearance, no thoracic deformity or SDL

- V.V. well transmitted on palpation
- V.M. well perceived on auscultation, no rales or additional noises
- normal chest tone on percussion.

Thorax symmetrical, no deformity, no CVC, no jugular vein turgor.

- Peak shock in place, no hepato-jugular reflux.
- Harzer sign negative.
- B1B2 well-perceived, regular rhythm, no superadded noises.

Abdomen normal in appearance, no scars, no arches, no fire points.

- No CVC
- Abdomen supple to palpation, no tenderness, tenderness or contracture.
- No hepatomegaly or splenomegaly
- Free OH
- Presence of a bladder globe.
- TR: normal
- The rest of the examination was unremarkable.
- Patient underwent placement of a central venous jugular catheter and removal of a venous line on the dorsal aspect of the left foot following venitis Plus skin rash on presentation.
- The patient was put on Triaxone 50mg/kg/J i.e. 700 mg*2 /d for 14 days.
- Acyclovir 15 mg/kg/d or 150*3 /d for 14 days.
- Depakine 40mg*3/ d
- Feeding initially via nasogastric tube, then orally.
- Patient apyretic.

Diagnostic Assessment

Imaging:

- CT scan performed on 18/12/2021: AVCI of the territory of the right Sylvian artery and the right posterior cerebral artery.
- Angioscanner on 12/19/2021: No abnormalities
- Echo-cardium: LV not dilated for age; not hypertrophied; correct segmental and global kinetics.

. LVEF: 60%.

. Valvular structures healthy.

. Dry pericardium.

. Thin and complaining ICV.

. Non-dilated auricles, intact inter-atrial septum, no visualizable shunts .

. no obstructions on chase tracts.

. Situs solitus normocardia.

- ECG: rhythm disturbances + non-specific T-wave abnormality.
- MRI performed on 04/1/2022: in favour of AVCI in the territory of the right Sylvian artery and the right posterior cerebral artery.
- Chest X-ray on 18/12/2021: normal.

Biological work-up:

- On admission:

Hb =8.6 Plq = 120000 WBC =12000 PCT < 0.5 CRP = 3

- Renal: normal
- Liver workup: normal
- ECBU: 10,000
- Proteinuria = 2 g/l - urinary creatinine = 12mg/l
- CSF cytobacteriological examination: (//)
- Glycorrhea = 0.81 / blood glucose = 1.27
- Proteinorrachia = 0.86
- Cytology: hyperleukorrhagia at 120 elements/mm3 predominantly lymphocytic + presence of 50 RBCs

- Sterile after 48 h
- COVID19 serology:
- IgG positive at 3.80
- IgM negative
- HIV, HAV, HBV, HCV serology: negative
- Quantiferon: positive
- Thrombocytopenia work-up ; Positive (protein S deficiency with normal prothrombin)
- Immunological work-up:
- NAA = negative
- Anti-DNA = negative
- Bacteriological examination of skin lesion swab performed on 15/10/2021: sterile after 48h incubation.
- Histological examination of skin lesion performed on 18/10/2021: in favor of necrotizing bullous dermatosis probably drug-induced.

Followup and Outcomes

- Neurological improvement was marked by the disappearance of sensory, sphincter and swallowing disorders, with complete recovery of motor function in the 2 MS, with regression of motor disorders in the 2 MI (2/5 proximal and 5/5 distal).
- Proctologically, anal hollowness regressed.
- The skin lesion is healing.
- Cytobacteriological examination of CSF performed on 21/10/21:
- Cytology: 58 elements predominantly + 55 RBCs
- Proteinorrachia = 0.3
- Glycorrachia = 0.96 / glycemia = 1.26
- Sterile culture after 48 h incubation.
- Biological workup on 10/25/2021:
- WBC = 13240
- Hb = 13.3
- Plq = 400000
- Renal: normal
- Liver workup: normal
- BBB = normal
- Therapeutic InterventionThe decision was to start the patient on:
- Triaxon 2.5g x2/d
- Aciclovir 450mg x3/d for 15 days
- A bolus of solumedrol 750mg/d for 6 days, followed by prednisolone 20mg per os: 2.5cp/d.
- Immunoglobulin (Tegeline): 6 vials/d => 50cc/h SAP for 04 days.
- Soluskin cream: 1 app x2/d
- Tulle gras

Discussion

According to the American Heart Association- American Stroke Association definition, ischemic stroke is an episode of neurological dysfunction caused by focal cerebral, spinal or retinal infarction involving a specific vascular district, and presenting with symptoms lasting more than 24 hours or until death, as well as neuroimaging, pathological and/or other objective evidence of focal ischemic lesions [1].

The occurrence of ischemic stroke in pediatric age, despite its rarity, implies age-related particularities in terms of risk factors, etiopathogenesis, clinical presentations and therapeutic approaches [2-6]. The lack of validated, evidence-based data on thrombolytic and endovascular treatment in children represents the main limitation to the prevention of disabling or life-threatening sequelae [2].

The aim of this review is to provide an up-to-date overview of current evidence and an insight into future perspectives on the management of acute ischemic stroke in children. A detailed analysis of the epidemiology, clinical presentation and management of neonatal stroke was beyond the scope of the authors, although readers may find useful updates in the references provided [3,4-5].

Conclusion

Physicians approaching to ischemic pediatric stroke should consider several differences versus adults that represent remarkable clues for the diagnosis and the treatment: [1]. the more significant etiopathogenic role of intracranial non-atherosclerotic arteriopathies, thromboembolic com- plications of congenital cardiopathies, and haematological disorders such as sickle cell's disease or coagulopathies; [2]. the high frequency of atypical presentations in children (e.g., headache only or no evident clinical symptoms); and ([3]. the lack of validated guidelines for thrombolytic and endovascular treatments in paediatric age with obvious negative consequences on the prevention of permanent neurological sequelae.

Author Statements

Patient Perspective

The patients agree to share their perspective on the treatment(s) they received.

Informed Consent

The patient give informed consent.

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