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Childhood Cerebellitis about a case in the paediatric department of Kamsar Hospital, Guinea.

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Abstract

Cerebellitis is a rare inflammation of cerebellar structures. We report the case of an 8-year-old girl, a student, presenting with acute post-infectious cerebellitis complicated by marked cerebellar atrophy.

The evolution under solumedrol and physiotherapy sessions was favorable; the child walks with a few falls.

Keywords: *cerebellitis, child, pediatrics, kamsar.*

Introduction

The cerebellum controls muscle tone, balance, and coordinates voluntary movements [1]. Cerebellitis is a rare inflammation of the cerebellar structures. It can be of infectious, post-infectious or post-vaccination origin. In the latter case, it can appear in the acute stage or develop after a silent period (parainfectious/post-infectious cerebellitis). It affects the individual in all age groups, although the incidence is higher in children where it poses a problem, diagnosis, therapy, but also prognosis [2]. Complications range from hydrocephalus to cerebellar atrophy and treatment includes corticosteroid therapy and associated physiotherapy [2; 4; 6; 9].

We report a clinical case of acute cerebellitis in an 8-year-old girl, a student, complicating a cerebellar atrophy of favorable evolution under corticosteroid therapy.

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The objective of this study was to describe the diagnostic and therapeutic approach in order to manage any post-infectious cerebellar syndrome early.

Conflict of interest: none

Observation

It is an 8-year-old girl admitted to the paediatric department of the Kamsar hospital for gait and balance disorder that has been evolving for 4 days.

History of the disease: According to his mother, the onset was brutal, marked by the onset of intermittent fever, accompanied by early postprandial vomiting, diarrhoea with semi-liquid stools three times a day and headaches.

A consultation carried out in a local clinic, where an assessment was carried out (Widal: TO=1/00, TH=1/400.; White blood cells: 16.56.109/l; Neutrophil: 79%, Lymphocytes 17.12%, RDT+/GE+: 180 T/µl, CRP+: 48 mg/l), Emmel test negative. She is treated with injectable ceftriaxone and injectable artesunate for 3 days. During this treatment, the parents found that the child had difficulty standing and walking, language disorders with retained appetite. The occurrence of these signs forces parents to consult the Paediatric Department of Kamsar Hospital.

Medical history:

The pregnancy is well monitored (5 ANCs performed, 2 doses of VAT received). Child born at term with a weight of 3000g, cries at birth, breastfed within hours of delivery. The child's up-to-date vaccination status according to the child's vaccination record. Several consultations for malaria and nasopharyngitis. The couple's only child, a healthy father and mother with no pathological history.

Clinical examination: Weight= 19 kg, Height= 112Cm, Heart rate= 96beat/min, Respiratory rate= 21 cycles/min, Temperature= 38.5°C, Blood pressure= 10/60mmHg, SpO = 99% in ambient air, BMI = 15.14kg/m2. Patient consciousness, normally coloured integuments and conjunctiva, no associated skin lesions, musculoskeletal system, no muscle atrophy, presence of muscular strength, there is a disturbance of coordination of movements and balance.

No signs of stroke, no history of sickle cell anemia or other hematopathies, the ORL(Oto-rhino-paharyngology) examination with otoscopy was normal, no notion of drug intoxication, no notion of gait disorder in the family

Digestive system: clean mouth, complete dentition, abdomen of normal volume, supple without palpable mass, audible intestinal peristalsis. Symmetrical chest, thoracoabdominal breathing, well-transmitted vocal vibration, audible vesicular murmur, no rattles. Palpable spike shock, normal heart sounds synchronous to peripheral pulses.

Para clinical examinations

ECB and CSF chemistry: proteins (1.16 g/l); blood glucose (3.5 mmol/l), 10-element lymphocyte.

Brain MRI at day 5: Demonstrated poorly limited diffuse edematous infiltration of the two cerebellar hemispheres as well as the median vermis without hyper signal diffusion or drop in the ADC, without also any underlying focal lesion or pathological contrast detected after intravenous injection of Gadolinium. There is also no mass effect on the V4, which remains of normal volume with a dilated upstream ventricular system. The deep durative sinuses are permeable. No intracranial bleeding. No noticeable meningeal contrast uptake. Conclusion, acute cerebellitis in children (Figure 1).

Treatment

Ceftriaxone 1g in the morning, 900 mg in the evening, for 72 hours, Gentamicin injection 80mg/day taken once for 3 days, then replaced with Amoxi-clavulanic acid 500mg, Paracetamol 200mg IVL as needed. Hydrocortisone: 3 boluses (15mg/kg/day) then Solumedrol 5mg: 1 cp x2/ for 15 days; Alvityl cp: 1 x2/day, physiotherapy for 1 month. After two weeks of treatment with corticosteroid therapy, there is an improvement in walking and balance, speech becomes understandable. The child goes back to school and walks with a few falls.

A follow-up MRI performed at the 2nd month showed a widening of the cortical cerebello-vermian folds and the V4, related to atrophy. There is also no abnormality of the cerebral parenchyma. Ventricular filament supratentotrial undilated. Middle structure in place. Absence of pericerbral collection. Absence of sternal abnormality (Figure 2).

Discussion

We report a case of acute cerebritis in children in Kamsar where there is a problem of underdiagnosis because of its rarity. Cerebellar syndrome is characterized by the presence of gait and balance disorders, difficulty executing movement and hypotonia, with varying degrees and with varying degrees. It is now known that motor disorders in cerebellar ataxia are not due to paralysis [1]. From a pathophysiological point of view, two main mechanisms of occurrence have been described. A direct mechanism where the infectious agent directly colonizes the cerebellar cortex by hematogenous or neuronal pathway and an indirect or post-infectious mechanism related to perivascular inflammation or an autoimmune process occurring during a documented or undocumented infection [3]. At this level, the immune reaction is directed against the antigens expressed in the cerebellar tissues(2)It is generally post-infectious, especially varicella; in addition, EBV, enteroviruses and other neurotropic viruses can more rarely cause ataxia. The clinical context is often obvious [4]; by the onset of fever, headache, vomiting, gait disorder after a history oto- rhino-laryngology or upper respiratory tract infection [4; 5]. These prodromes were found in our observation. Some signs such as convulsive seizures, pyramidal syndrome, meningeal syndrome, and intracranial hypertension have been reported [7; 8]. However, salmonellosis and malaria can mask the viral infection in our tropical regions with limited resources. The analysis of the CSF was without particularities in our series. CSF analysis may be normal [6; 7] or show a cellular response [4; 8]. EBV-positive viral serology was documented in some studies [7]. In our observation, we were unable to highlight the causative agent due to the weakness of our technical platform. Differential diagnoses include posterior fossa tumors such as medulloblastomas and pilocytic astrocytomas, but also stroke, drug poisoning in children, and Friedreich's disease, an autosomal recessive spinocerebellar heredodegeneration. However, anamnesis, laboratory assessment and imaging can rule out these diagnoses [4]. On MRI, we observed a poorly limited diffuse edematous infiltration of the two cerebellar hemispheres as well as the median vermis. Cerebellar parenchymal involvement

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associated with significant cerebellar edema responsible for possible hydrocephalus and compression of the brainstem has been reported by some authors [6; 7]. Generally, hyper-intense FLAIR and T2 lesions on MRI are characteristic of cerebellitis [4]. The evolution was favorable in our series, with a gradual resumption of walking. In most cases, the course of cerebellitis is usually benign within a few days to a few weeks [7]. The follow-up MRI after 3 months of treatment had noted in our series, a marked cerebellar atrophy. Moderate or discrete cerebellar atrophy has been reported by some authors [7; 9]. Motor and or intellectual deficits are more frequent in etiologies other than chickenpox and especially in cases where the initial MRI was abnormal, and related to acquired cerebellar atrophy with or without gloss following inflammatory involvement [7]. The scheduled physiotherapy continues and has its effect. This functional rehabilitation is based on repetition methods that condition motor learning by the "trial and error" method [1].

Conclusion

Childhood cerebellitis occurs after an infectious episode characterized by noisy symptoms of fever, headache, vomiting, and gait and speech disorders. Brain MRI is the key to diagnosis and allows the evolution of the disease to be monitored. Early corticosteroid therapy remains the appropriate treatment, its duration varies according to the authors and generally the course is favourable.

Keywords: cerebellitis, child, pediatrics, kamsar.

Iconography

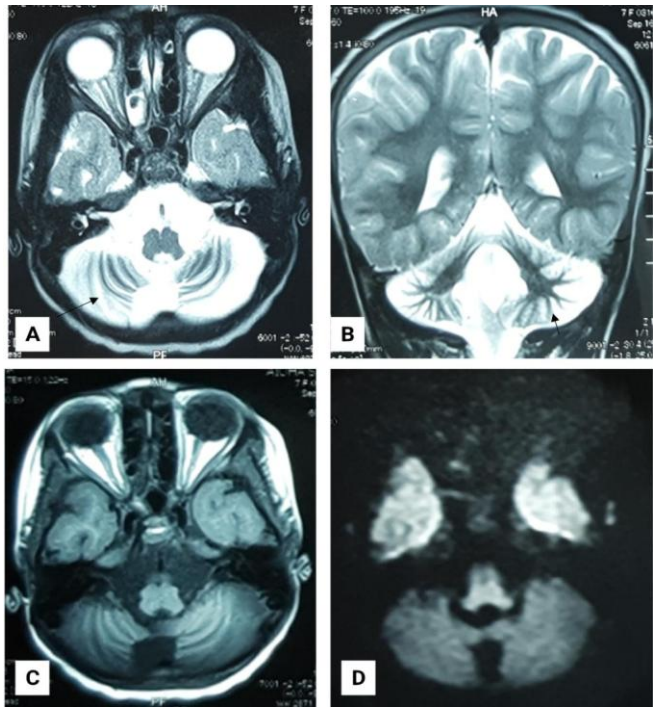

Figure 1: Brain MRI of an 8-year-old girl with gait and balance disorders evolving for 4 days.

Axial MRI T2 (A) and coronal MRI T2 (B) show a bilateral cerebellar diffuse hypersignal (arrow). No signal anomaly in axial T2 Flair (C) and Diffusion (D). No noticeable meningeal contrast consistent with acute cerebellitis.

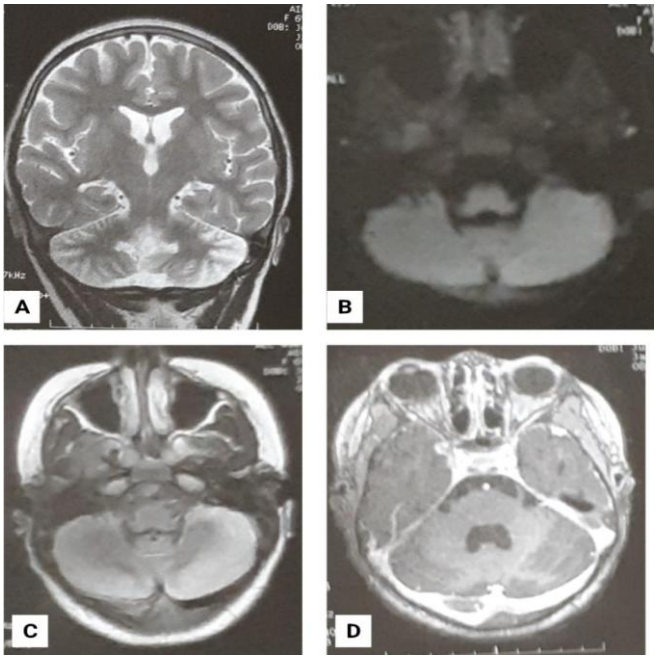

Figure 2: Brain MRI to control bilateral cerebellitis performed after 3 months in an 8-year-old girl with a clear improvement in symptomatology.

T2 (A) coronal MRIs show a more marked enlargement of the cerebellar cortical sulcus on the left. No signal abnormality in axial Diffusion (B), T2 Flair (C) or contrast in axial T1 FatSat, Gado (D), suggestive of bilateral cerebellar cortical atrophy, particularly on the left.

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