Quest Journals
Journal of Medical and Dental Science Research
Volume 10~ Issue 2 (2023) pp: 82-84
ISSN(Online) : 2394-076X ISSN (Print):2394-0751
www.questjournals.org

Research Paper

Artery of Pacheron Stroke in a 7 year old Child

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Purpose: Artery of Pacheron (AOP) stroke is a rare condition, worse still occurring in a child. This is a case in an infant managed conservatively from presentation till complete recovery.
Case Report: A 7 year old female presented with headache, irritability, fever, vomiting, loss of consciousness and convulsions. CT scan showed significant calcifications in the basal ganglia bilaterally especially the lentiform nucleus. AOP infarcts are rare and clinical presentation are not like cerebrovascular accident.
Discussion: Artery of Pacheron (AOP) is an abnormal variant of the arterial supply of the thalamus. Its occlusion can lead to bilateral thalamic and rostral midbrain infarct presenting as memory loss, fluctuating levels of consciousness and altered mental status.

Received 05 Feb., 2023; Revised 13 Feb., 2023; Accepted 15 Feb., 2023 © The author(s) 2023.
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The patient was a 7 year old female, who presented with fall from height 10 days prior to presentation, fever of one day, vomiting of one day, convulsion of 9 hours. Was in apparent good health until 10 days prior to presentation when she came home from school with complaints of a fall. She had gone to write on the blackboard with a chalk and slipped. She hit her head on the ground. There was no loss of consciousness. There was headache and irritability.
Fever started 9 days later, was constant and high grade. Thermometer readings were 39 deg and paracetamol and tepid sponging all failed to relieve the fever.
Vomiting started same day. Was clear, projectile and contained recently ingested feeds. Was not blood stained and not foul smelling.
Convulsions was observed 9 hours prior to presentation. Was generalized tonic clonic with each episode lasting as long as 10 minutes. Patient had several episodes per day.
On account of these the child was taken to a peripheral hospital where she was given parenteral diazepam and paracetamol. However, she was noticed to be unconscious the next morning and was referred to our centre

Past medical history: She had been admitted at two for trauma to the head after which she started having seizures.

Birth History: Mother booked for care at 5 months. Attended ANC regularly. Not sure she received tetanus immunization. Delivery was vaginal and child cried at birth with history of yellowness of the eyes.

Immunization: completed immunization.
Developmental milestones: Sat at 5 months, walked at 2 years one month.
Examination showed a child, in respiratory distress, having tonic clonic seizures with arching of the back, febrile (39^0C), excessive oral secretions with carpopedal spasms

CNS: unconscious Glasgow coma scale GCS E-4 V-2 M-3 (9/15). Pupils equal, reacted slowly, hypertonia in all limbs.
CVS: pulse 180 bpm, BP 114/68mmHg
Respiratory: rate 40/minute, transmitted sounds.
Abdomen: full moved with respiration, no tenderness no organomegaly.
Diagnosis: Acute Bacterial Meningitis R/O Traumatic Brain injury, Raised intracranial pressure, ?Hypocalcemia

She was placed on 5% dextrose saline at 2/3 maintenance (966mls @ 10drops per minute). The following investigations were requested, malaria parasite, full blood count with bands and blood film, blood culture, serum electrolyte urea and creatinine, serum calcium, PO4, Retroviral screening, Brain CT Scan, lumbar puncture (for CSF microscopy, protein and glucose).
Antibiotics and antiseizures were commenced and seizure chart opened. She was managed as an unconscious patient. Intravenous Mannitol 1g/kg of 10% 8 hourly each dose given over 30 minutes 2 hourly. She was suctioned PRN, exposed and tepid sponged with SpO2 monitored..
All the requested hematological investigations were normal.
CT scan
Technique
Topogram followed by pre and post contrast axial slices of 3mm thickness and 5mm intervals taken from the base to the vertex. Coronal and saggital reformatted images were subsequently obtained.
Findings:
The topogram was unremarkabnle
Axial sections (brain window) demonstrate cerebral hemispheres with normal sulci and gyri. The grey white mater interface is maintained There are significant calcifications in the basal ganglia, bilaterally especially the lentiform nucleus. Note isolated punctate hyperdensities (HU:125) in the frontal lobes ?Significance. The interhemispheric fissure is in the midline. The components of the cerebral ventricles are normal in size, outline and position. No intra ventricular hemorrhage seen. The Thalami and Brain stem appear normal in outline and density. The orbits and their contents are normal. The demonstrated paranasal sinues and mastoid air-cells are well pneumatised. The cerebellopontine angles are free. The cerebellum is normal in outline and density. Evaluation of the bone window showed intact cranium.
Impression: Basal Ganglia Calcifications.
Differential Diagnosis:
1. Hypoparathyroidism
2. Normal variant.

Clinico-pathological correlation advised.

A definitive diagnosis of bilateral thalamic stroke in keeping with Artery of Percheron Stroke was made.
Patient was admitted in HDU. Should avoid light and sound, to continue NG feeding.
She was placed on Tabs Epanitin and parenteral Omeprazole, and was transferred to the ICU. For strict input and output chart, intranasal oxygen intermittent at 2-3 L/m.
She was placed on syrup qinine. Blood culture results shows Enterococcus fecalis sensitive to Augmentin. She was given Augmentin 90mg/kg in 2 divided doses

By 28 day of admission she was fever free and seizure free, other parameters remained the same, GCS 8/15, hypertonia still present. After a month and 6 days on admission GCS was 10/15. Other parameters were the same. By 2 months on admission physiotherapy was commenced. 8 days later she could sit without support, moved all the limbs normally, could not talk but recognized her mother.
The vital signs were normal. An assessment of good clinical progress was made. The Nasogastric tube was removed. She was discharged 2 days later.
On follow up she presented with persistent scratching of the ear. Ear nose and throat reviews showed wax impaction. It was treated with ear drops and analgesics. She had intermittent seizures lasting less than a minute, conscious, expressive aphasia, power grade 4 in all limbs, hypertonia and ataxic gait. She continued epitin and physiotherapy

Discussion

Anatomy

Artery of Percheron (AOP) is a rare anatomical variant of posterior circulation arteries of the brain.
Gerald Percheron studied thalamic blood supply and described its anatomical variants depending on the arteries it arises from.
A single thalamic artery arises from the proximal posterior cerebral artery between the basilar and the posterior communicating arteries and supplies both paramedian territories of the thalami.[1]
This is due to hypoplasia or agenesis of the other P1 segment.
This common trunk is known as AOP.
A compromise of the blood flow of AOP leads to bilateral paramedian thalamic infarction with or without rostral midbrain involvement.
The result is the clinical picture of impaired arousal and memory, language impairment and ocular movement disorders.[2]

Apart from ocular movement disorders the above clinical features were present in the index patient

Epidemiology
Prevalence remains unknown but was found to be present in 7-11.7% of autopsy studies. Although it seems to be a common anatomical variant, strokes due to AOP territory infarction are rare. No studies in our environment, but was 0.4% in an analysis done in Mexico involving 3750 patients with first ischemic stroke. It was 0.5% in another analysis on 3712 patients with ischemic stroke. In thalamic strokes AOP territory strokes are in the range of 4-18%. Its rarity and unusual clinical presentation for a stroke, causes a delay in instituting advanced brain imaging and diagnosis

Clinical Features
The clinical picture is impaired arousal and memory loss, language impairment and sometimes ocular movement disorders.

Radiology
Non-Contrast CT of the brain is often normal misleading the physician to consider other pathologies other than a stroke.

Prognosis
Depends on the extent of infarction but has been found to be good with regards to morbidity and mortality. AOP stroke is a rare clinical finding.[3]

Conclusion
The above case presentation shows it can present in any age group. A good knowledge of the clinical presentation, non-contrast CT scan and a high index of suspicion would be helpful in preventing undue delay in diagnosis and treatment.

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