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Case Report

Paediatrics Section

Chronic Subdural Hygromas, Cerebral Atrophy, and Developmental Delay Following Paediatric Traumatic Brain Injury: A Case Report

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ABSTRACT

Traumatic Brain Injury (TBI) contributes significantly to the burden on healthcare systems. The variability in patient profiles and injury mechanisms complicates prevention, diagnosis and treatment, limiting advancements in the field. Although imaging modalities like Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) are essential in the diagnosis of TBI, they may miss subtle lesions. Children with TBI are at a high-risk of long-term neurological sequelae and these include seizures and developmental delays. This case report presents a six-year-old male child, fifth of birth order born out of non-consanguineous marriage, with developmental delays following history of fall at 2-3 months of age. Initial imaging studies reported bilateral Subarachnoid Haemorrhage (SAH) and inter-hemispheric bleeding. Subsequent scans revealed right-sided subdural hygroma and ischaemic infarcts. MRI brain findings demonstrated gliosis and cystic encephalomalacia. The patient developed Generalised Tonic-Clonic Seizures (GTCS), which were controlled with anticonvulsants. Later anticonvulsants were discontinued after a seizure-free period of two years. Upon recent admission, severe anaemia required blood transfusion and repeat MRI brain showed chronic subdural hygromas and cerebral atrophy. The patient was stabilised and discharged with ongoing developmental and neurological follow-up. This case underscores the significant long-term neurological and developmental consequences of paediatric TBI, highlighting the importance of early diagnosis, regular monitoring, and multidisciplinary management. Non-surgical approaches, combined with appropriate neuroimaging and clinical assessments, can effectively manage complications such as subdural hygromas and anaemia. Further research is needed to optimise care and improve outcomes for children affected by TBI.

Keywords: Long-term outcomes, Neurodevelopmental disorders, Paediatric neuroimaging, Subarachnoid haemorrhage

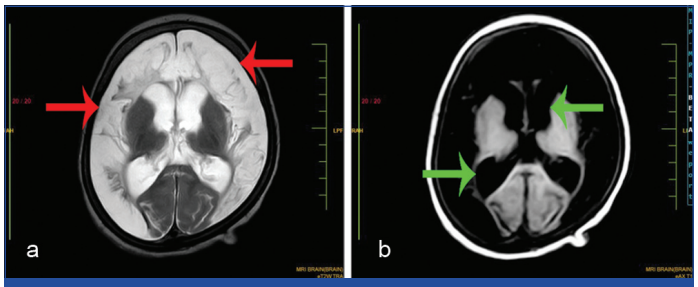
CASE REPORT

A six-year-old male child, born via a non-consanguineous marriage presented to the paediatric department with delay in achieving developmental milestones, initially noticed at six months of age, along with severe pallor and irritability for the past one month. The child was apparently alright until 2-3 months of age, when he experienced a fall from a swing at home from a height of approximately 1.5-2 feet. A Computed Tomography (CT) scan done at that time reported bilateral fronto parietal Subarachnoid Haemorrhage (SAH), inter-hemispheric bleeding and bilateral Sylvian fissure involvement. A follow-up CT scan done three months later reported a subdural hygroma in the right fronto temporal region, measuring up to 1.9 cm in the right frontal area along with an old ischaemic infarct in the left fronto temporal and right occipital regions. At 10 months of age, the child was evaluated by a paediatric neurologist due to developmental delay and seizure activity. Developmental delay was assessed based on the following parameters: in gross motor skills, the child was unable to roll over and sit with support; in fine motor skills, there was no reaching for objects and no pincer grasp. Socially, the child exhibited no stranger anxiety, and in terms of language milestones, the child did not laugh or produce monosyllables or bisyllables. An MRI of the brain revealed gliosis and cystic encephalomalacia in the bilateral fronto temporal regions, with ex vacuo dilatation of the adjacent lateral ventricles and thin linear calcifications. The child was also diagnosed with GTCS and started on syrup phenobarbitone at a dose of 6 mg/kg/day, administered in two divided doses, and this regimen was continued for three years. However, this medication was discontinued after two years of seizure-free status. There was no history of developmental delay or related conditions reported in any family members.

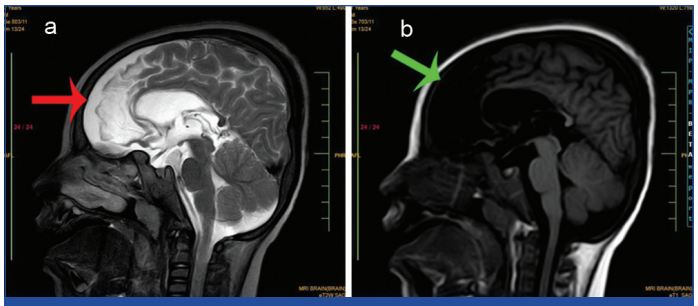
The child presented with delay in achieving developmental milestones, first noted at six months of age. While neonatal milestones were achieved, subsequent age-appropriate milestones were not met. The child was unable to roll over or sit with support, never reached for objects, and never demonstrated a pincer grasp. No evidence of stranger anxiety was noted to date, and the child did not demonstrate laughter, and did not speak monosyllables or bisyllables. Gross motor delay was evident as the child was unable to roll over or sit with support, with no improvement since six months of age. Fine motor delay was noted as the child failed to reach for objects or demonstrate a pincer grasp. Socially, the child exhibited no stranger anxiety, typically expected by eight months of age. Language delay was apparent, as the child did not laugh or produce monosyllables or bisyllables, as expected by six months. At six years of age, delays were observed in all four domains of developmental milestones, consistent with global developmental delay. Over the past one month, the parents reported severe pallor and irritability, which has progressively worsened, with irritability being most noticeable during feeding and handling.

On examination, the child was afebrile with a pulse rate of 120 bpm, respiratory rate 26 cpm, SpO_2 96% on room air, and blood pressure 96/54 mmHg. Palpebral conjunctival and palmar pallor were present, with no icterus, cyanosis, clubbing, or lymphadenopathy. The child was observed in the supine position with tightened, crossed legs. Higher mental functions, including intelligence, memory, and emotions, could not be assessed; no preferred handedness was noted. Pupils were equal and bilaterally reactive to light. Hypertonia was present in all four limbs, with power graded at 3/5 and deep tendon reflexes exaggerated (3+). Abdominal examination revealed hepatomegaly, with the liver

palpable 3 cm below the right costal margin in the midclavicular line. Cardiovascular and respiratory system examinations were normal. These significant delays across multiple domains formed the basis for the initial provisional diagnosis of global developmental delay with severe anaemia under evaluation. His initial complete blood count revealed severe anaemia, with haemoglobin of 3.1 g/dL. Other laboratory findings included a total leukocyte count of 6,900/µL and platelet counts of 1.32 lac/µL. MRI brain reported chronic subdural hygromas [Table/Fig-1] along both fronto-parieto-temporal convexities with bitemporal cranio-cortical thickening and cerebral atrophy [Table/Fig-2]. The MRI findings also indicated chronic small vessel ischaemia. Ear Nose and Throat (ENT) and ophthalmology evaluations were conducted and no acute findings were reported. Following a comprehensive assessment, including clinical evaluation, neuroimaging, and laboratory investigations, the final diagnosis was established as spastic cerebral palsy with severe anaemia.



[Table/Fig-1]: a) Axial T2-weighted MRI showing bilateral chronic subdural hygromas (red arrows) along the fronto-parieto-temporal convexities with significant bitemporal cranio-cortical thickening and cerebral atrophy; b) Axial T1-weighted MRI illustrating dark subdural collections (chronic hygromas) and marked cerebral atrophy, with enlarged lateral ventricles (green arrows) and widened sulci.



[Table/Fig-2]: a) Sagittal T2-weighted MRI showing chronic subdural hygromas with significant cerebral atrophy, particularly affecting the frontal (red arrow) and parietal lobes; b) Sagittal T1-weighted MRI illustrating cortical thinning and ventricular enlargement, consistent with chronic cerebral atrophy (green arrow) following Traumatic Brain Injury (TBI).

The child was admitted in the Paediatric Intensive Care Unit (PICU) and was given multiple blood transfusions. Initially, Packed Red Cells (PRC) was transfused at a dose of 5 mL/kg. After 24 hours, a second transfusion of 10 mL/kg PRC was administered, followed by a third transfusion of 15 mL/kg PRC, 24 hours later. A repeat CBC, conducted 24 hours after the last PRC transfusion, showed a haemoglobin level of 8.8 g/dL, Total Leukocyte Count (TLC) of 5,900/µL, and platelet count of 0.97 lac/µL. The child was started on syrup promethazine for irritability at a dose of 0.1 mg/kg per dose administered orally during the day and 0.5 mg/kg per dose at bedtime for five days. The medication was then continued only at bedtime for an additional three days before being discontinued. The child remained vitally and haemodynamically stable during the hospital stay. The patient was hospitalised for one week, during which stabilisation and necessary interventions were carried out. Stabilisation involved correcting severe anaemia through multiple PRC transfusions, managing irritability with syrup promethazine, initiating physiotherapy sessions, and starting baclofen 2.5 mg at bedtime to address spasticity. Following discharge, the patient was advised on the importance of developmental follow-up and outpatient care to monitor progress and adjust management as needed. The patient returned for follow-up one month after discharge. On follow-up, reduced irritability and minimal improvement in spasticity were noted. However, there was no significant improvement in developmental milestones at that time, underscoring the need for continued physiotherapy and long-term multidisciplinary management.

DISCUSSION

TBI is the leading cause of death or severe disability in children [1]. TBI in children is a serious concern due to the high prevalence and its potential for long-term consequences [2]. TBI in paediatrics is different from adult TBI in terms of pathophysiology, clinical outcomes and long-term consequences. Children, particularly under two years of age are at increased risk for developing complications such as subdural hygromas and cerebral atrophy due to the immaturity of their brain structures and a higher propensity for diffuse injury patterns [3]. The collections of cerebrospinal fluid in the subdural space have been linked to both acute and chronic brain injuries in children and may be worsened by underlying conditions such as anaemia or coagulopathy [4]. Subdural hygromas in paediatric TBI are managed conservatively unless there are signs of mass effect or increased intracranial pressure [5]. In this particular case, chronic subdural hygroma was managed without surgical intervention and the patient was followed up with regular imaging and clinical assessments, which is consistent with the current best practice for non-symptomatic subdural collections. The role of repeated neuroimaging, particularly with MRI, has been emphasised in detecting chronic sequelae such as gliosis, cystic encephalomalacia, and atrophy, all of which were present in our patient and likely contributed to his developmental delays [6]. Blood transfusions given to the patient during recent hospitalisation were crucial in stabilising his condition and are often necessary when anaemia is severe to impair oxygen delivery to the brain.

Similar cases of paediatric TBI reported in the literature highlight the complexity and variability in presentation, management, and outcomes. Lee IS et al., reported a five-year-old male with severe TBI requiring a 298-day hospital stay, multiple neurosurgeries, and reconstructive procedures [7]. Challenges included hydrocephalus, recurrent haematomas, and scalp defects, with individualised nutrition improving outcomes. Our case shares features like chronic subdural hygromas and multidisciplinary care but differed in severity, requiring no surgeries or prolonged hospitalisation. These cases highlight the varied presentations of paediatric TBI and the importance of tailored management and follow-up. Tanikawa D et al., reported a 12-year-old female with severe TBI and cerebral infarction due to post-traumatic vasospasm [8]. Despite a Glasgow Coma Scale (GCS) score of 3 and subdural and SAHs on CT, no brain oedema was detected. Management included Intracranial Pressure (ICP) monitoring, sedation, seizure prophylaxis, and mannitol for ICP control. This case emphasises early vascular imaging in high-risk paediatric TBI to detect vasospasm. In contrast, our case lacked vasospasm but highlights the importance of vigilant neuroimaging and clinical monitoring to guide management in paediatric TBI.

Developmental delays, particularly in motor and cognitive domains are common in children with history of TBI, especially when injuries occur during critical periods of brain development [9]. Early intervention programmes focussing on physical, occupational and speech therapy can significantly improve the prognosis for children with TBI-related developmental delays. However results vary significantly depending on the degree of the initial injury and the promptness of treatment interventions [10]. This case emphasises how crucial a multidisciplinary approach is to the treatment of paediatric TBI. Routine monitoring, neuroimaging and developmental evaluations are crucial in managing the long-term consequences and to improve outcomes for impacted children. This case report also highlights the necessity for additional investigations into the pathogenesis of subdural hygromas and the role of anaemia in the healing process following TBI.

CONCLUSION(S)

This case highlights the complex and varied characteristics of TBI in the paediatric age group underscoring the importance of early diagnosis, continuous monitoring and multidisciplinary management. Chronic subdural hygromas, developmental delays and associated neurological sequelae observed in this six-year-old patient indicate the long-term consequences of early childhood TBI. While non-surgical management was effective in this particular case, need for regular neuroimaging and clinical follow-up is paramount to assess progression and to guide treatment. This case emphasises the critical role of individualised care in managing paediatric TBI, particularly in addressing complications such as anaemia, which can exacerbate brain injury. Further research is essential to enhance therapeutic strategies and improve the overall prognosis for children suffering from TBI.

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