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Infant with Paediatric Astrocytoma and the "Rescuing Urgency" of Severe Acute Thrombocytopenia: Causally Related or Coincidental?

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ABSTRACT

Background:
Although immune thrombocytopenic purpura (ITP) has been associated with solid tumors among adults, this coexistence is exceedingly rare in children. We present an infant with a brain tumor associated with severe acute thrombocytopenia (SAT), which has been very rarely reported in association with pediatric brain tumors.

Case Presentation:
A 7-month-old male twin presented with a 2-day history of fever and a 1-day history of right subconjunctival haemorrhage that had progressed to involve the left eye. Initial investigations showed severe thrombocytopenia and a provisional diagnosis of ITP was made. He was managed conservatively but he developed a forehead swelling and subcutaneous bruising with minimal improvement in thrombocytopenia. Three days later he had multiple episodes of vomiting, worsening subconjunctival haemorrhage and somnolence. Transfontanelle ultrasound revealed intracranial haemorrhage and computed tomography brain imaging demonstrated left-sided mass effect. He was admitted to the Intensive Care Unit (ICU) and dexamethasone, ceftriaxone, omeprazole and apheresed platelet concentrate were given. Later he was transferred to a specialized neurosurgical facility where he underwent a craniotomy with mass excision of intra-operative astrocytoma (biopsy confirmed). He had a second craniotomy on account of residual tumor tissue revealed on brain magnetic resonance imaging 48 hours after the first surgery. He was subsequently discharged 9 days post-2nd craniotomy after other supportive management and optimisation. Investigations showed dramatic normalization and elevation of platelet count at post-discharge review. This was sustained throughout the follow-up clinic during the 12-week follow-up care with no recurring thrombocytopenia. Patient continued to record significant improvement in the motor function throughout the follow-up clinic.

Conclusions:
This case highlights severe acute thrombocytopenia as a potential early clinical clue to an underlying intracranial pathology in infants. While a paraneoplastic immune mechanism is hypothesized, the association may also represent a coincidental coexistence. Further studies are required to clarify this relationship

Keywords: Astrocytoma, Infant, Intracranial hemorrhage, Severe acute thrombocytopenia, Subconjunctival haemorrhage

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INTRODUCTION: -

USevere acute thrombocytopenia (SAT) in infancy is a significantly alarming clinical presentation with a broad differential diagnosis.[1] The causes of SAT may range from benign self-limiting infections to life-threatening hematologic or oncologic disorders. Among the leading causes in children, immune thrombocytopenic purpura (ITP) stands out as the most common etiology particularly following recent viral illness or immunization. ITP is an acquired autoimmune disorder characterized by the destruction of platelets and suppression of platelet production in the bone marrow. Its peak incidence is in between 2 to 5 years of age and a slight male predominance in infants under one year old.[2] While most pediatric ITP cases are transient and self-limiting its presentation with SAT in infants under 12 months is less common. ITP not responding to standard therapy should always prompt evaluation for underlying or coexisting pathology. Although the association of ITP with solid tumors has been well-documented in adults such a link remains rare in the pediatric population. Even more uncommon is the coexistence of SAT with primary central nervous system (CNS) tumors in infancy. The prevalence of pediatric brain tumors is approximately 5 per 100,000 children per year, making them the most common solid tumors in childhood.[3] Pilocytic astrocytoma, a World Health

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Organization (WHO) grade I glioma is the most frequent brain tumor in children which accounts for approximately 20–25% of all CNS neoplasms in the pediatric age group. These tumors usually present with signs of increased intracranial pressure such as vomiting, altered consciousness and focal neurological deficits. In infants the diagnosis is often delayed due to subtle and nonspecific manifestations. In infants open cranial sutures allow for some intracranial volume accommodation without early signs of raised intracranial pressure.[4] Thus, identifying early systemic clues such as unexplained thrombocytopenia or bleeding manifestations may be critical in early diagnosis of these intracranial tumors in infancy. In adults, paraneoplastic ITP has been hypothesized to occur due to antigenic mimicry or immune dysregulation induced by tumor antigens. This antigenic mimicry results in autoantibody-mediated platelet destruction.[5] First-line management of newly diagnosed pediatric ITP typically includes observation for mild cases and corticosteroids or intravenous immunoglobulin (IVIG) for moderate to severe cases. However, when a patient of ITP fails to respond to initial therapy and there is progressive bleeding manifestations and additional signs suggestive of central nervous system involvement (e.g., vomiting, drowsiness, or focal deficits) clinicians must maintain a high index of suspicion for alternative diagnoses. Brain imaging is not routinely indicated in standard ITP workup unless neurologic symptoms arise, however it must be kept in mind that early intracranial pathology may be masked by presumed ITP. This may lead to delayed intervention that are fraught with danger of catastrophic complications such as intracranial hemorrhage.[6] Despite the growing body of literature on pediatric ITP and brain tumors individually, the intersection between both conditions, particularly in infants, remains largely unexplored. This case report attempts to bridge this critical knowledge gap by proposing a plausible immunopathogenic link and calling attention to an overlooked diagnostic pathway.

CASE PRESENTATION:

A 7-month-old male twin presented with a 2-day history of fever and right subconjunctival haemorrhage that had developed one day prior to presentation. His physical examination was unremarkable except for the presence of conjunctival haemorrhage. The haemorrhage progressed from a single spot on the right eye to multiple spots, with eventual involvement of the left eye. There was no history of trauma, no recent vaccination, no eye discharge, or other developmental concerns. His vaccination record was up-to-date. Anthropometric measurements revealed weight, length, and head circumference all above the 97th percentile for the child's age. Initial laboratory investigations showed severe thrombocytopenia with a platelet count of 8,000/μL, haemoglobin of 9.9g/dL, and white blood cell count of 7,480/μL(neutrophils 24.9%, lymphocytes 64.5%).

Peripheral smear showed no blasts or schistocytes. There was no clinical or laboratory evidence of sepsis, disseminated intravascular coagulation or congenital thrombocytopenia. Bone marrow examination was deferred due to clinical instability. Based on these findings, a provisional diagnosis of immune thrombocytopenic purpura was made. Following a hematology consultation and given the absence of life-threatening bleeding and stable neurological status at presentation, conservative management with close follow-up 2 days later was recommended for the child. At the scheduled two-day follow-up, the patient's fever had resolved, but his mother reported finding a forehead bump and subcutaneous bruising. A repeat platelet count showed minimal improvement to 11,000/μL, and the patient was scheduled for a one-week follow-up appointment. Three days later, the patient's condition deteriorated with multiple episodes of vomiting, increased subconjunctival haemorrhage, marked sleepiness, and reduced activity. On examination, he was drowsy but arousable, with tachycardia (132 beats/minute) and tachypnoea (42 cycles/minute). Laboratory evaluation showed platelet count of 29,000/μL. The transfontanelle ultrasound revealed a left ventricular and cerebral hemisphere intracranial haemorrhage estimated at 50mL, while CT Brain imaging demonstrated left-sided mass effect.

The patient was admitted to the Paediatric Intensive Care Unit (PICU) with head elevation to 30 degrees, nil by mouth status, intravenous fluids at 75% maintenance, dexamethasone (0.15mg/kg every 6 hours), ceftriaxone (100mg/kg/day), and omeprazole (10mg daily). He received apheresed platelet concentrate at 10mL/kg, which improved his platelet count to 82,000/μL. He was subsequently transferred to a specialized neurosurgical facility with a modified Glasgow Coma Scale score of 13/15. The patient underwent a craniotomy with excision of a cystic and intracystic mass, seven days after the initial presentation. This excision was suspected to be a pilocytic astrocytoma. Intraoperative findings confirmed a left parietal lesion with elevated intracranial pressure. He was extubated 12 hours postoperatively but developed unilateral jerky movements of the left lower limb at 16 hours, requiring phenobarbital administration. 48 hours post-operation, the patient experienced severe abdominal breathing followed by a generalized tonic-clonic seizure with bradycardia and oxygen desaturation, which necessitated cardiopulmonary resuscitation and atropine administration. A subsequent brain MRI revealed residual tumor tissue, requiring a second craniotomy three days after the initial procedure.

Two days after the second craniotomy, the patient returned to the PICU of our facility. Neurological examination showed right sixth cranial nerve palsy, right-sided hemiparesis, and regression of developmental milestones. Laboratory investigations showed dramatic normalization and elevation of platelet count to 540,000/μL, his complete blood count showed a PCV of 27%, Hb: 8.9 g/dl WBC: 10,400 (N-36.6%, L-53.5%). Serum renal function test revealed hyponatremia, (sodium was 133.8 mmol/l), other parameters were within range.

Management included intravenous dexamethasone, packed red blood cell transfusion, transition from phenobarbital to oral levetiracetam, and continued antimicrobial therapy. Ophthalmological assessment confirmed intact optic nerves bilaterally. Regular physiotherapy was initiated to address right-sided weakness. The patient also received intensive nursing care with careful monitoring of fluid input and output. We discontinued IV dexamethasone six days after the second craniotomy and commenced oral dexamethasone, which was then discontinued on day eight. After completing ten days of intravenous antibiotics, he was transferred to the ward at 8 days post-2nd craniotomy and discharged the next day. The patient thereafter continued to recover at home with ongoing physiotherapy, gradually regaining developmental milestones, though still behind his twin in development.

DISCUSSION:-

ITP is the most common cause of acute thrombocytopenia in children and may be triggered by viral infections or vaccinations. This is in keeping with our decision to consider ITP as a provisional diagnosis based on the SAT and relative lymphocytosis in this well-vaccinated infant on his first visit. Our choice of watchful waiting in this patient is in keeping with first-line recommendations of current guidelines in the initial management of newly diagnosed ITP.[7] The minimal improvement in the patient's platelet count on follow-up visit

also left us hopeful, in agreement with previous reports of spontaneous resolution of acute ITP within twelve months in most children. However, our patient soon developed an intracranial hemorrhage (ICH), leading us to intervene with steroids and platelet transfusion in line with established guidelines. ICH is an exceedingly rare complication of ITP, occurring in less than 1% of ITP cases. Prophylactic transfusion would also not significantly alter the course of this disease due to the potential for the antiplatelet antibodies to attack transfused platelets, just as cases of ICH occurring during and after steroid therapy have been documented.[8]

Ordinarily, astrocytomas account for up to half of pediatric brain tumors and usually present with clinical features related to increased intracranial pressure and focal neurologic deficits.[9] However, since our patient did not present with any of these clinical features initially, we missed the rescuing urgency of the presenting SAT till the patient developed ICH much later in the course of the disease.

Moreover, we noted a sharp transition from preoperative thrombocytopenia to postoperative thrombocytosis. It must be emphasised that postoperative thrombocytosis may also be explained by inflammatory response to surgery, or recovery from consumptive thrombocytopenia, and therefore the occurrence of SAT in our patient with Paediatric astrocytoma cannot alone confirm a paraneoplastic mechanism. Steroid therapy alone cannot also explain the postoperative thrombocytosis because patient had commenced steroid therapy days prior to surgery with no dramatic improvement in platelets count.

Indeed, this theory of a solid tumor precipitating ITP or ITP resolving following tumor resection has been reported in a few adult cases. Our case reports this in infantile age group. There is a similar case report of an older child with chronic ITP, where the platelet count did not improve immediately after tumor excision for few months.[10] Nevertheless, our patient's platelets count returned to normal consistently postoperatively throughout the subsequent months of follow up period. In addition, unlike the older child with chronic ITP who underwent a splenectomy before the resolution of her thrombocytopenia, our patient did not have to undergo any splenectomy after his craniotomy. This perhaps, may suggest an immunological association between astrocytoma in our patient and the SAT he initially presented with. More cases however need to be studied to establish this association.

CONCLUSION:-

This case underscores the importance of reconsidering the diagnosis in infants with severe acute thrombocytopenia who develop neurological symptoms or fail to follow the expected clinical course of ITP. While an immunologically mediated association with astrocytoma is hypothesized, causality cannot be established from a single case.

DECLARATIONS

Ethics Approval and Consent to Participate: Ethics Approval was waived, but confidentiality was ensured, and informed consent was obtained from the parents of our case

Consent for Publication: Consent for publication was obtained from the parents of our case.

Competing Interests:

The authors declare that they have no competing interests

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Author Contribution:-

OK, AU, SO : contributed to patient management, data collection, and manuscript drafting. ZE,CD,FA, : participated in data interpretation, literature review, and manuscript editing.TM : supervised the study and approved the final manuscript.

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