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Case Report

A Case of Resolved Cerebral Venous Sinus Thrombosis after Prolong Ventilatory Support with No Neurological Deficit

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Abstract

Neonatal Cerebrovenous sinus thrombosis (CVST) is extremely rare, however it is a devastating condition and one needs to be aware of this condition to diagnose it. The risk factors for CVST are still not properly understood. The largest registry for stroke and for neonatal CVST is from the Canadian registry which quotes an incidence of 0.6 per 100,000 population per year. No data is present for the neonatal CVST in this region. To date there is no consensus on the role of anticoagulant therapy and therefore therapy is largely supportive, however individual cases have to be evaluated and treated on merit.

We use detailed retrospective review of chart as a methodology to ascertain our findings and all relevant laboratory investigations were fetched from the internal lab data for the case study.

The site of CSVT in newborns reflects distribution in adults and children, as this condition is more frequent in the superficial system than in the deep venous system (approximately one-third of the case of CSVT in newborns affects deep veins. Data on the correlation between the extent and location of clots and associated brain lesions have been reported [3]. Such correlations between venous anatomy and hemorrhagic injury is well known in premature children, where the persistence of the germinal matrix on the caudal scar is known. Germinal matrix causes intraventricular hemorrhage with possible later medieval venous infarction [4,5]. Thrombosis usually begins in the vicinity of the parietal lobe within the superior Sagittarius sinus, probably due to the peculiar course of the posterior frontal, parietal and occipital nerves. Management of neonatal CSVT is urgent, if necessary, with the treatment of any underlying condition and anticonvulsants. Anticoagulation therapy is controversial. The rationale for anticoagulation in CSVT is to prevent thrombus expansion and recurrence, favor spontaneous thrombus resolution, and prevent further brain damage due to hemorrhagic infarction. Anticoagulation with incomplete heparin and low molecular weight heparin (LMWH) is safe in pediatric patients with venous thrombosis of the brain [6]. Therefore, the risk of heparin-induced intracerebral bleeding is against the risk of bleeding due to progressive thrombosis.

Our case showed that appropriate and urgent management of a complicated case of cerebral venous sinus thrombosis (CVST), who was initially ventilated can become better and fully recovered despite controversial management of Anti-thrombolytic activity. The other important finding we observed was sudden improvement of our baby with removal of ventilatory support along with improvement of MRI findings.

Keywords: *Cerebral Venous Sinus Thrombosis (CVST); Ventilatory Support; Neurological Deficit*

Introduction

Cerebral Venous Sinus Thrombosis (CVST) may affect All pediatric age groups with an incidence of at least 0.67 per 100,000 per year in newborns [1]. However, its occurrence is underestimated as many physicians are unaware of the condition, radiological diagnosis is more difficult in newborns and, above all, clinical. The presentation is non-specific. Cases of early presentation may present with maternal comorbid (maternal pre-eclampsia/hypertension, fetal distress) and acute illness (asphyxia, respiratory problems), while late presentation is often accompanied by neurological symptoms such as seizures, lethargy, apnea, and poor feeding. Delayed presentation after an acute illness has also been observed, e.g. Dehydration [2]. The site of CSVT in newborns reflects distribution in adults and children, as this

condition is more frequent in the superficial system than in the deep venous system (approximately one-third of the case of CSVT in newborns affects deep veins. Data on the correlation between the extent and location of clots and associated brain lesions have been reported [3]. Such correlations between venous anatomy and hemorrhagic injury is well known in premature children, where the persistence of the germinal matrix on the caudal scar is known. Germinal matrix causes intraventricular hemorrhage with possible later medieval venous infarction [4,5]. Thrombosis usually begins in the vicinity of the parietal lobe within the superior Sagittarius sinus, probably due to the peculiar course of the posterior frontal, parietal and occipital nerves. Management of neonatal CSVT is urgent, if necessary, with the treatment of any underlying condition and anticonvulsants. Anticoagulation therapy is controversial. The rationale for anticoagulation in CSVT is to prevent thrombus expansion and recurrence, favor spontaneous thrombus resolution, and prevent further brain damage due to hemorrhagic infarction. Anticoagulation with incomplete heparin and low molecular weight heparin (LMWH) is safe in pediatric patients with venous thrombosis of the brain [6]. Therefore, the risk of heparin-induced intracerebral bleeding is against the risk of bleeding due to progressive thrombosis.

Case Report

A five day old male neonate, presented in Emergency Department due to dull activity, decrease oral intake and one fever spike at home, he was the 2nd child of the family. He was born in our hospital at 36 weeks of gestation through spontaneous vaginal delivery with the birth weight of 3360 grams. There was no maternal risk factor during pregnancy and all the antenatal scans were normal.

On arrival in emergency department his physical examination revealed that he was dull, lethargic with poor sucking. His capillary refill time was more than 3 seconds, respiratory rate of 35 beats per minute and heart rate of 170 beats per minute that showed tachycardia, his anterior fontanelle was full despite of clinical dehydration but rest of his CNS reflexes were normal. Due to his clinical condition investigations were performed and lab studies showed thrombocytopenia with deranged coagulation profile. The septic workup was sent, CSF profile showed partially treated meningitis, while the CSF culture showed no growth so, the baby was managed in line of meningitis.

At 6th day of life the baby suddenly showed signs of severe respiratory distress for which he was immediately intubated, lumber puncture was repeated that showed raised white cell count, an urgent MRI brain after neurological consultation was planned that showed filling defect in the superior sagittal sinus, torcula, straight sinus and bilateral transverse sinuses with gross distension suggesting, cerebral venous sinus thrombosis (CVST), diffuse gyral prominence with leptomeningeal enhancement representing meningitis and intraventricular hemorrhage. Due to the abnormal findings the hematology team was taken on board, and they mentioned that since the issue of thrombocytopenia is persistent, subcutaneous Enoxaparin should be given with cover of platelet transfusion. We started enoxaparin and levels of anti Xa factor were monitor and doses of enoxaparin adjusted accordingly as per international guidelines.

The baby remained intubated and improved slowly with weaning ventilator settings, he did receive 21 days of i/v antibiotics. We have repeated MRI brain after 33 days of treatment that showed resolved CVST and development of grade II intraventricular hemorrhage, so enoxaparin was stopped and baby was successfully extubated.

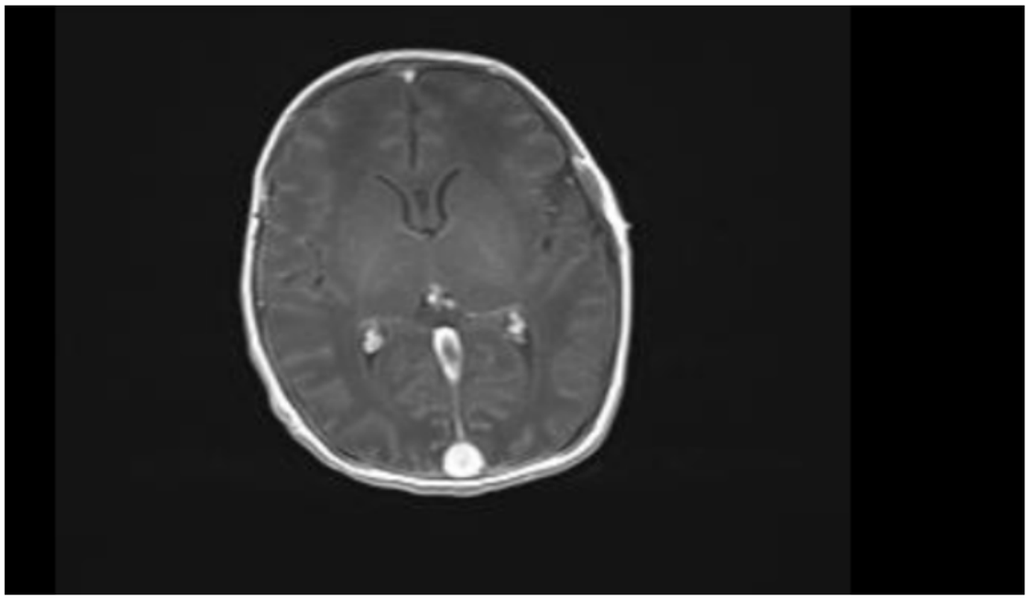

Figure 1: *Superior sagittal sinus, torcula, straight sinus and bilateral transverse sinuses are grossly distended and show continuous filling defect. Overall findings are suggestive of cerebral venous sinus thrombosis (CVST). There is diffuse gyral prominence with leptomeningeal enhancement representing meningitis.*

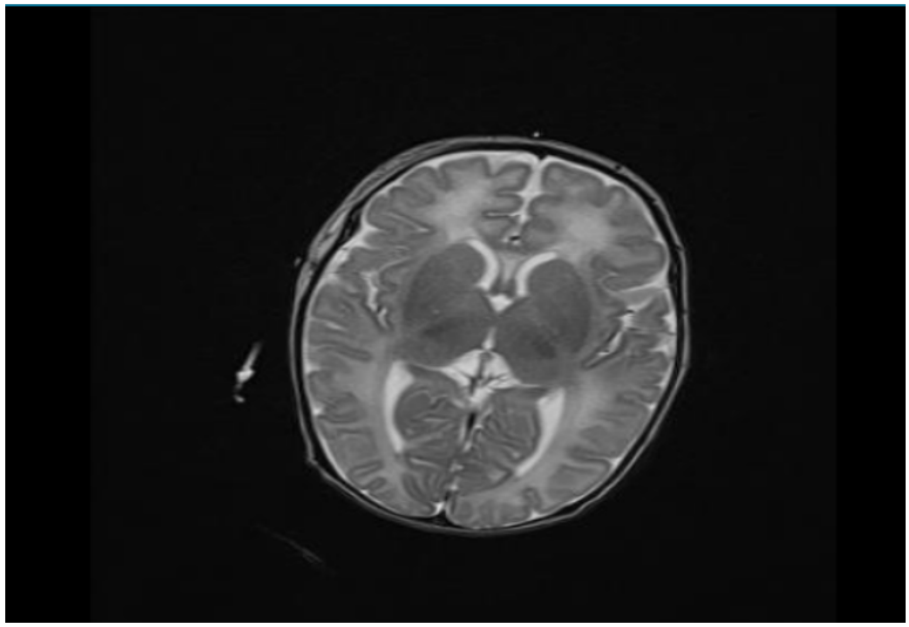

Figure 2: *Previously noted filling defect in the superior sagittal sinus, torcula, straight sinus and bilateral transverse sinuses is not visualised. The cerebral venous sinuses are nondistended on current examination. There is no cerebral oedema or leptomeningeal enhancement on current examination.*

Discussion

In our case, there was a delayed presentation and we suspect this could be due to dehydration, which matched clinical symptomatology since physical examination revealed that the child was dull, lethargic with poor sucking, which is coherent with the normally observed symptoms. Consistent with these findings was a full anterior fontanelle despite clinical dehydration, however no other association could not be established since no other anomalies were identified on physical examination. Findings of MRI brain were consistent with CVST as well as IVH, consistent with previous literature pointing towards association of CVST with other lesions e.g. IVH as in our case. The American College of Chest Physicians has recently published evidence-based clinical practice guidelines on anti-thrombotic therapy in newborns [7]. For neonates with CSVT without significant intracranial bleeding, they suggest articulation, initially with latent heparin and later with LMWH for at least 6 Weeks, and no longer than 3 months. In the case of significant intracranial bleeding associated with CSVT, radio-logical monitoring of thrombosis at 5 - 7 days is suggested and anti-coagulation if thrombus proliferation is noted. Likewise, we administered enoxaparin and anti XA factor levels were monitored according to dose adjustment. We followed the child and an MRI brain was performed that resolved the development of CVST and grade II atrioventricular bleeding.

Conclusion

Our case showed that appropriate and urgent management of a complicated case of cerebral venous sinus thrombosis (CVST), who was initially ventilated can become better and fully recovered despite controversial management of Anti-thrombolytic activity. The other important finding we observed was sudden improvement of our baby with removal of ventilatory support along with improvement of MRI findings.

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