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TEACHING FILES (GRAND ROUNDS)

DISSEMINATED CYTOMEGALOVIRUS INFECTION IN AN INFANT WITH CONGENITAL HEART DISEASE – MANAGEMENT ISSUES

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Clinical Problem

A 2-month-21-day old male infant presented with respiratory distress, jaundice for 4 days, intermittent clay-coloured stools for 12 days and one episode of convulsion. He was diagnosed with moderate ventricular septal defect via echocardiography on 25th day of life and was on furosemide and digoxin. He was born at term via vaginal delivery with birth weight of 2.4 kgs. An elder sibling died at 3 months of age due to congenital heart disease with neonatal hepatitis. On examination, the baby was 3.1 kgs, had jaundice with hepatomegaly and pansystolic murmur. Other systems were normal. Investigations are depicted in table-1.

Table 1: Serial Laboratory parameters of the patient

Laboratory parameters	Day 1	Day 5	Day 12	Day 13	Day 18	Day 21	Day 25	Day 28	Day 31
Haemoglobin (g/dL)	9.3	8.2	7.2	9.3	7.4	10.5	10.2	15.8	13.4
PCV (%)	29	26	22	28.7	23	31.9		47	40.7
TC (K/uL)	17240	13000	9710	8416	10500	12170	7060	13300	9980
Polymorphs (%)	39.1	45	40	50	57	63.3	76	86	75
Lymphocytes (%)	52.8	41	48	38	38	29.3	20	11	22
Monocytes (%)	6.8	10	11	10	5	5.3	2.7	1	2.9
Eosinophils (%)	0.2	2.5	0.7	16	1	2.1	0	0	0
Platelet count (K/uLl)	684	5.6 L	4.5 L	366	405	5.48 L	4.4 L	2.1 L	2.1 L
C-reactive protein (mg/L)	0.6	1.5	0.6	2.5		1.4	1.1	2.5	32.3
SGOT (U/L)	165	87	89		61	89	133	157	
SGPT (U/L)	157	89	84		82	115	136	88	
Albumin (g/dL)	4.2	3.5	3.4				3.7	3.3	
Bilirubin- T/D (mg/dL)	10.1/9.5	13.9/ 12.8	11.9/ 10.6	9.6/ 20		14.3/ 3.6	14.5/ 13.1	18.9/ 16	15.3/ 14
BUN (mg/dL)	18	11	13				20	39	33
Creatinine (mg/dL)	0.43	0.2	0.3				0.45	0.60	0.45
Lactate (mmol/L)	3	1.3				1.5	6.8	3.4	1.65

NOTE: PCV- Packed Cell Volume, TC- Total Count, BUN-Blood Urea Nitrogen.

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He was given intravenous (IV) levetiracetam and was on non-invasive ventilation for 3 days, shifted to non-invasive ventilation using pressure control for next 5 days followed by continuous positive airway pressure. He then had 1 episode of desaturation, worsening distress and seizure requiring re-intubation. High-resolution computed tomography of chest and pulmonary angiogram showed 1 cm wide patent ductus arteriosus inserting to left pulmonary artery origin, complete collapse of right upper lobe (RUL), mucoid impaction in RUL bronchus causing luminal occlusions and consolidations in lower lobes. Electroencephalogram was normal. Airway assessment suggested minimal posterior subglottic injury. Blood TORCH polymerase chain reaction (PCR) was positive for cytomegalovirus (CMV). Bronchoalveolar Lavage (BAL) culture was positive for Klebsiella Pneumoniae >105 CFU/ml. BAL CMV PCR showed viral load of 38225 IU/ml, with log of 4.5. Ophthalmological and hearing assessment were normal. Ultrasound abdomen showed increased echotexture of liver with mild hepatomegaly, right pelvi-ureteric junction obstruction with scattered internal echoes suggesting pyelonephritis. Urine routine showed 6-8 pus cells/hpf, bile salts and bile pigments. Liver biopsy showed fibrous expansion of portal areas with chronic inflammation, extensive intrahepatic cholestasis and giant cell transformation of hepatocytes with Metavir F2. Liver CMV viral load was 140425 IU/ml, log of 5.1. He was treated with IV gancyclovir, IV antibiotics, ursodeoxycholic acid and fat soluble vitamins. Thus, this child was detected to have disseminated CMV with CMV pneumonia, CMV viremia and CMV hepatitis.

Question – Is this congenital (cCMV) or acquired CMV (aCMV)? How to treat it?

Discussion:

CMV, a member of Herpesviridae family, is a double-stranded DNA virus known to stay dormant within the host and reactivate later.[1] CMV infection during early infancy typically occurs as a congenital infection, while postnatal acquisition is less common.[1] However, distinguishing between these two forms can often be challenging.[1] cCMV is diagnosed if the virus is identified in urine, saliva or dried blood spot samples within first 14 days of life, while aCMV is detected after this period.[2] cCMV is frequently observed in patients with acute respiratory distress syndrome and is linked to increased mortality including prolonged ICU stays and lung fibroproliferation.[3] In contrast, aCMV is not considered a serious health concern.[4] In our patient, CMV was detected at 3 months of age and after prolonged hospital stay, so it is not possible to comment whether it is cCMV or aCMV.

Common causes of acquiring CMV are contamination with blood and body fluids during vaginal birth, during transfusion and via breastfeeding.[5] CMV can affect multiple organs such as eyes, gastrointestinal (GI) tract and other tissues, especially in immunosuppressed individuals.[5] Among GI complications, CMV colitis and esophagitis are most frequently observed.[5] In certain situations (small for gestational age, congenital anomalies, etc.) there is a higher risk of developing CMV "sepsis syndrome" which includes apnoea, bradycardia, distended bowel and pallor with leukopenia, thrombocytopenia, elevated C-reactive protein etc.[4] Our patient never had a positive septic screen. In cCMV affecting major organs, antivirals should be given immediately, within first month of life and for 6 months.[6] In contrast, those with mild or no symptoms are not advised antiviral treatment.[6] aCMV is usually harmless in full-term infants and offers natural immunization.[4] Premature/very low birth weight infants may develop symptoms within 4–6 weeks after birth, often needing treatment.[4] For infants with life-threatening symptoms, IV ganciclovir for 2-6 weeks, followed by oral valganciclovir should be given.[6] In contrast, those without severe symptoms are treated with oral valganciclovir alone for the full course.[5] Antiviral drugs include IV Ganciclovir (6 mg/kg/day) every 12 hours or oral valganciclovir (16 mg/kg/dose) every 12 hours.[4]

Infants require periodic monitoring of absolute neutrophil count, platelet count, blood urea nitrogen, creatinine, liver function and developmental tests.[6] Follow-up involves close monitoring for long-term sequelae, developmental delays, audiological evaluations and multidisciplinary support.[6] Regular hearing examinations until 6 years of age and ophthalmic examinations until 5 years of age is necessary in cCMV.[6]

In our patient, it was not possible to assess whether the CMV was congenital or acquired. However, since he had CMV pneumonia, with CMV hepatitis and viremia, he was initially treated with IV ganciclovir and then shifted to oral valganciclovir for 6 weeks.

Compliance with ethical standards

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Conflict of Interest: None

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