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CONGENITAL CYTOMEGALOVIRUS INFECTION IN AN INFANT - AN UNUSUAL PRESENTATION –A CASE REPORT

Pediatrics

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KEYWORDS

INTRODUCTION

Congenital cytomegalovirus(CMV) infection is the leading non-genetic cause of sensorineural hearing loss(SNHL) and neurodevelopmental sequelae.CMV is the largest of human herpes virus[1]. Although CMV rarely causes symptoms in normal individuals ,it is an important cause of infection in immunodeficient patients. Congenital CMV infection can present with symptomatic infection in approximately 10% of infected newborns whereas 90% of infected infants asymptomatic in the newborn period. It presents with severe multiorgan disease in less than 5% of infants.[2]

CASE REPORT

Newborn born to a 25 year old G_2P_2 mother at POG 35 weeks by preterm vaginal delivery at home , said to have cried immediately after birth and breast fed with in first hour of birth. Neonate remained symptomatic duing first month. Antenatal history was uneventful.

Baby was admitted on day 36 of life with erythematous rash all over the body for 6 days and purulent discharge from both eyes for 4 days. Rash initially present in the perianal and gluteal region , gradually involving whole of the body associated with peeling of skin over the dorsum of hands and feet. There was no h/o fever .

On physical examination his weight – 2.2 kg (0 to -2 Z score),length – 50 cm (0 to -2 Z score) ,OFC -31.5 cm (< -3 SD MICROCEPHALY), Temperature -99.8 degree F , HR-128bpm, RR-28. Ponderal index was 2.6(SYMMETRICAL IUGR).Pallor was present and on per abdomen examination ,liver was firm with round margins on palpation and was felt 6 cm below right costal margin with total span of 10 cm and spleen was palpable 5 cm below the left costal margin, firm in consistency with round margins .Rest of the systematic examination was normal.

Fig 1

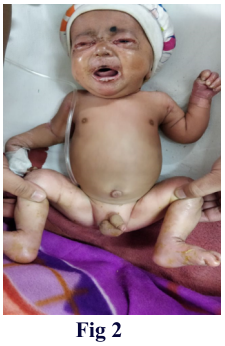

Fig 2

(Figure 1 showing purulent discharge from both eyes and involvement of the rash in mucocutaneous junction .Figure 2 showing involvement of the rash and peeling of skin in the dorsum of legs and hands)

INVESTIGATIONS:

At admission , routine investigation of infant revealed values (Table 1)

investigations	values
Hemoglobin	5.3 g/dl
Total Leucocyte count	32000/mm^3
Differential leucocyte count	N66L30E4
Platelet count	32000/cu mm
Manual Platelet count	43000/cu mm
Peripheral smear	dimorphic blood picture with atypical lymphocytes along with toxic granules
Blood urea nitrogen/ creatinine	21/0.9 mg/dl
SGOT/SGPT/AlK	32/27/270 IU/ml
PT/INR	1.2
Random Blood Sugar	95mg/dl
Tzanck smear	Gram positive cocci

As per history and examination done, possibity of SSSS (Staphylococcal skin scalded syndrome) was kept .After collecting blood culture ,IV antibiotic cloxacillin was started and tobramycin eye drops for local ophthalmic use were started.USG abdomen confirmed hepatomegaly andand splemomegaly Neurosonogram was normal.

Eye and fundus examination was normal.Hearing assessment of the baby was with in normal limits.In view of persistent anemia and no regression in hepatosplenomegaly along with sepsis following differential diagnosis was kept

1. TORCH infection
2. Atypical HLH
3. congenital leukemia

Bone marrow aspiration was aparticulate. Seerum triglyceride and fibrinogen levels were with in normal range and serum ferritin was 750 microgm/ml. TORCH report came on day 16 th of admission showed CMV for IgM POSITIVE (8.69) .URINE CMV DNA PCR was 2500 million copies /ml and mother serum CMV IgM was also positive.

TREATMENT

In view of reactive CMV serology revised diagnosis of disseminated CMV infection was made and baby initiated on oral valganciclovir (dose 15-16 mg/kg/day bd) .after 7 days of valganciclovir therapy ,follow up CBC and LFT were with in normal range and discharged on oral valganciclovir.

OUTCOME AND FOLLOW UP

During the follow up period,physical and haematological parameters of the patient observed to be improved with valganciclovir therapy and he is on regular follow up.

Fig 3

(figure 3 showing follow up clinical pictograph at 1 month of follow up)

DISCUSSSION

Currently,congenital CMV infection is the most prevalent infection-related cause of congenital neurological handicap .CMV infection is widely spread in developing countries and in communities with poor socio-economic status 3.In our study,the patient came from low economic background.Transmission sources of CMV include saliva ,urine,stool,tissue or organ transplant .the most common clinical features are hepatomegaly,splenomegaly jaundice and petechiae.Neurological symptoms are microcephaly, hydrocephalus,mental retardation ,cerebral palsy ,sensorineural hearing loss ,chorioretinitis and seizures.symptomatic congenital CMV infection has two presentation.baby with IUGR ,microcephaly or intracranial calcification. Other early manifestation include petechiae ,blueberry muffin spots consistent with extramedullary hematopoiesis. Neuroradiogical manifestations include pachygyria,polymicrogyria,lissencephaly,schizencepahly,heterotopia and cortical dysplasia including hippocampal dysplasia and migration anomalies.

CONCLUSION

CMV infection is increasing and every attending physician should be aware of it is the leading non-genetic cause of SNHL. Neonatal screening for hearing loss should be done on a routine basis .A prevalence of 65 per 1,00,000 children with prelingual hearing defects attributed to CMV infection 4.PCR DNA for CMV is very sensitive and specific for diagnosis 5 .Hand washing is an important prevention tool.

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