TEACHING FILE

Grand Rounds

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FEVER, RASH AND CONVULSIONS

Clinical Problem : A two and a half years old boy born of third degree consanguineous marriage presented with fever for 6 days and maculopapular rash starting from face and then spreading to rest of the body for 2 days. He had a generalized tonic clonic convulsion half an hour back and was treated with IV Midazolam, Phenytoin and Phenobarbitone after which seizure subsided. He now had post itcal drowsiness. There is no otorrhoea, contact with TB. He is fully immunized till Booster 1 and milestones are normal. On examination, he has tachycardia {heart rate = 120/min} with hypotension {B.P. = 90/60 mm of Hg} and prolonged capillary refill time. He is drowsy with normal power and reflexes. There are no meningeal signs. He has a maculopapular erythematous rash all over the body. He has hepatomegaly and other examination findings are normal.

Question : What is the diagnosis?

Expert Opinion: This child has presented with fever, rash and a convulsion without meningeal signs suggestive of infection involving the brain in form of encephalitis. Among the common infections, bacterial and viral infections would be on the forefront. The common bacterial infections affecting the brain at this age are H.influenza b, meningococcus and streptococcus pneumoniae and pneumococcal. Of these, meningococcus is the only one which leads to rash. However, the rash is usually petechial and the child would have toxicity and shock and multisystem organ dysfunction. Also bacterical infections will have meningeal signs. Thus, meningococcal infection or other bacterial infection seems unlikely. Viral infections commonly occur with fever, rash and encephalitis and common viral infections that can lead to brain involvement are enteroviruses, herpes virus, measles, mumps, arenavirus, dengue, poliovirus. Herpes and enteroviruses usually do not cause generalized rash. Mumps may be accompanied by parotid enlargement. The child has already received measles and polio vaccine and thus, they seem to be unlikely. Dengue may lead to fever, rash and brain involvement. Also, dengue can lead to hypotension too. Thus, in this child, one may suspect dengue encephalitis. In this child, complete blood count showed thrombocytopenia {platelet count = 97,000, cumm} with CSF showing 8 lymphocytes, cumm with normal proteins and sugar. CSF culture was negative. Dengue IgM by Panbio test was 3 {Normal is Less than 0.9 AI}. His electrolytes, blood sugar and calcium were normal. He was treated with IV Fluids and he responded on his own with subsequent normalization of platelet count.

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CMV INFECTION

Clinical Problem: A pregnant woman at 8 months gestation underwent a regular antenatal ultrasound where it was found that the fetus had mild ventriculomegaly. She then underwent a malformation scan and it was found that the child had Dandy Walker variant with subependymal calcifications in the brain with normal abdominal scan. The mother had fever at 3 months of gestation. A TORCH titre was done in the mother and CMV IgG was positive with absent CMV IgM. Karyotype was not advised as there were no other abnormal features.

Question : What should be further management?

Expert Opinion : Prenatal diagnosis of CMV is difficult and complicated. The mother is CMV IgG positive suggesting that she had CMV in the past. However it does not tell us whether she had CMV in the recent past or earlier, one way to know is to repeat CMV IgG after 4 weeks and see for 4 fold rise or by doing the CMV IgG avidity test. Four fold rise is suggestive of recent infection whereas same titre or mildly elevated, decreased time is suggestive of past CMV infection. Measurement of CMV-specific IgG avidity has proven to be a powerful tool for distinguishing primary from nonprimary CMV infection. Defined as the strength with which the IgG attaches to antigen, IgG avidity matures with the length of time following primary infection. Thus, IgG produced within the first few months following primary infection exhibits low avidity, whereas IgG produced several months or years later exhibits high avidity. Several groups of investigators have shown that detection of CMV specific IgG of low avidity is a reliable indicator of infection within the previous 6-8 months.

What is important is to know whether the fetus is infected with CMV infection. Spread of CMV infection to the fetus occurs with placental infection. Once fetal infection occurs, the virus replicates in the renal tubular epithelium, passes into urine and amniotic fluid. Hence amniocentesis or percutaneous umbilical blood sampling can be done for isolating virus by PCR or CMV IgM. However this is possible only after 32 weeks of gestation. This is because of necessary time interval from maternal infection through transmission, fetal infection and viral shedding. The amniotic fluid is most likely to be positive if tested 7 weeks or more after the maternal infection. However, doing these procedures are fraught with risk and there are currently no well-studied, effective therapies for CMV in pregnancy. Intravenous ganciclovir injection in fetus has been tried but response was not good. Thus, here since the pregnancy is already is 8th months of gestation, one can wait till the mother delivers, monitor the child for signs and laboratory features of CMV infection and then treat accordingly. Also, other system involvement can be checked for and if any dysmorphism, then even karyotype can be done.

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WHAT IS THE CAUSE OF CHOREOATHETOSIS?

Clinical Problem: A 12 years old HIV infected boy presented with sudden onset left sided choreoathetoid movements which have remained same for past 4