

A diagnostic dilemma.

Anukul A*, Nikam A*, Jain KC**, Chatterjee R***

Abstract :

Fulminant Meningococcal disease is an acute medical emergency with varied presentations including life threatening complications. We report a four years male child who presented as a case of meningoencephalitis but was finally diagnosed as a case of severe meningococcal septicemia and glomerulonephritis with hospital acquired Klebsiella infection.

Key Words : Meningocci, Glomerulonephritis, Septicemia, Purpura Fulminans.

Case Report

A four year old male child second product of non-consanguineous marriage was brought with complaints of:

1. Fever of 4 days duration
2. Rash of 2 days duration

Fever was high grade, continuous, acute in onset and associated with a rash which developed on the 2nd day of fever; initially present over the trunk followed by involvement of both limbs and face. It was associated with drowsiness since night before admission and two episodes of vomiting. He was treated for the above complaints by a private practitioner before admission to our hospital. His past history, family history and birth history were not significant. His developmental history was essentially normal. He was immunized for age, having received MMR vaccine at 18 months. His anthropometric findings were commensurate for his age (weight: 14.3 kgs. and height: 98 cms).

On clinical examination, patient was arousable on verbal commands as per (AVPU Scale) and restless. He had fever (100 °F), with commensurate tachycardia and tachypnea. His blood pressure was normal at 110/70 mm of Hg. He had severe pallor with a fine petechial rash all over the body sparing the palms and soles.

* PG Student, ** HOD and Proff., *** Professor
Department of Pediatrics, Rural Medical College, Loni

Corresponding Author :

Dr. Nikam A.
PG, Student, Department of Pediatrics,
Rural Medical College, Loni, Tal. Rahata, Dist. Ahmednagar (MS)

Examination of the central nervous system showed that he was drowsy, had equally reacting pupils, increased power in all limbs and brisk reflexes. Plantars were positive but signs of meningeal irritation (Brudzinski's/ Kernig's) was present. No features of raised intracranial tension was found. Hepatosplenomegaly was present in the abdomen. Other systems were essentially within normal limits.

In view of the presentation of fever with rash, and associated CNS signs a diagnosis of viral exanthematous fever / meningoencephalitis / severe bacterial sepsis was made. He was investigated and the following results were received:

Hb : 7gm/dl, PCV: 20.6%.

TLC : 22000 /cu mm,

DLC : Polymorphonuclear leucocytes: 84%

Lymphocytes: 12 %

Eosinophils: 2%

Monocytes: 2%. Platelet Count: 30000 /cumm, Bleeding Time: 7m 20s, Clotting Time: 6m 10s ProthrombinTime: 15 sec Liver Function Tests: Serum bilirubin (total): 1.6 mg / dl, Conjugated: 1.2 mg / dl

Serum GPT: 97 u /l.

Serum Alkaline Phosphatase: 124 u/l

Urine: Routine examination: Within normal limits

Renal function tests :

Serum urea: 22 mg / dl

Serum Creatinine: 0.96 mg / dl

Serum sodium: 136 meq/l

Serum potassium: 4.6 meq/l

Cerebrospinal fluid:

Cytology : 384 cells / cumm, predominantly neutrophils

Proteins: 127mg/dl, Glucose: 29 mg/dl, LDH:84u/l

Gram stain : Occasional pus cells, no organism, negative for Acid Fast Bacilli.

HIV status: Negative

Chest X ray findings: Within normal limits

In view of convulsions, rash, severe thrombocytopenia and CSF pleocytosis, the following treatment was started: : Intravenous fluids (IV), platelet transfusion and antibiotics in the form of ampicillin and cefotaxime and IV Phenytoin sodium for convulsions. On the second day of hospitalization the patient had two episodes of generalized convulsions which were controlled with IV diazepam. Rash had now increased to involve the palms and soles as well, with resultant.

Purpura Fulminans (Fig 1), more marked over the buttocks. One fresh whole blood transfusion was administered and steroids in the form of IV dexamethasone was started. By the fourth day he had no fresh lesions. His repeat CSF cytology showed 68 cells / cumm, with 60% neutrophils. CSF glucose: 49 mg /dl, Proteins: 24mg /dl and LDH: 30.8 u/l. Repeat platelet count showed an increase to 80000/cumm and blood culture sent previously was sterile.



Fig 1: Purpura Fulminans -Buttocks.

On day five, he had hyperpyrexia and digital gangrene (hands & feet) (Fig 2) as well as gangrene of ear lobule.



Fig 2: Gangrene of the Fingers.

IV antibiotics were continued and low molecular weight heparin with Tab Pentoxiphylline started. Another two units of platelets were transfused. By eighth day he showed improvement and was conscious and oriented. Purpura had reduced, digital and ear lobe gangrene started drying, but fever was persistent (Fig 3).



Fig 3: Temperature chart of the patient,



Fig 4: Auto Amputation of the Fingers.



Fig 5 : Auto amputation of the toes

Syrup chloroquine was started empirically and blood culture was repeated. At this time he tested negative for Widal test, Weil Felix Test, Dengue (IgG and IgM).

His platelet count rose to 3.2 lakh/cumm. With this treatment he showed initial recovery but soon developed high fever with chills, tachycardia acidotic breathing and features of shock. Blood gas analysis showed metabolic acidosis together with respiratory acidosis. His acidosis was corrected with sodium bicarbonate infusion. Repeat urine examination now showed gross hematuria with 30-35 red cells / HPF. Oliguria was dealt with dopamine infusion at 5 µg /kg /min.

By day twelve he started showing signs of recovery. CSF examination was repeated and was normal but he had persistent hematuria with hypertension (Acute glomerulonephritis). He was now put on Tab Depin 5mg sublingually with regular blood pressure monitoring. On the twentieth day he became normotensive but microscopic hematuria persisted. Patient was discharged for domestic reasons and asked to follow up on OPD basis where he was regularly followed up weekly for three months. Hematuria and hypertension persisted for two months. Tab. Depin was continued for 6 weeks. He was readmitted after one month for blood transfusion.

Discussion

A four year old male child presenting with fever and rash develops convulsions, followed by severe gangrene of the hands, toes and ear lobes, responds well to initial therapy for eight days, then suddenly develops shock followed by persistent hematuria and hypertension. Initial investigations reveal a septicemia blood picture with severe thrombocytopenia and CSF pleocytosis.

Later blood culture shows growth of klebsiella spp. associated with clinical picture of shock followed by features of acute glomerulonephritis.

Was this a case of meningococcal meningitis with secondary hospital acquired klebsiella sepsis or was it a case of Henoch-Schonlein purpura with secondary septicemia?

1. Meningococcal meningitis:

(a) Features in favor: Purpura Fulminans severe thrombocytopenia

(b) Features against:

- (i) No meningococci could be isolated from the CSF
- (ii) Glomerulonephritis cannot be accounted for

2. Vasculitis syndrome (Henoch-Schonlein Purpura) with secondary septicemia:

(a) Features in favour:

- (i) Hematuria (ii) Purpura (iii) Hepatosplenomegaly.
- (iv) Seizures though rare are a fatal complication of Henoch-Shonlein purpura (HSP)

(b) Features against:

- (i) HSP is essentially nonthrombocytopenic.
- (ii) Edema, arthritis, upper respiratory tract infection were not present.
- (iii) Rash did not occur in crops but appeared together all over the body.

Having weighed all available evidence a retrospective diagnosis of **meningococcal meningitis with secondary hospital acquired sepsis due to Klebsiella infection, was made.**

CSF and blood cultures negative for meningococci can be explained by the fact that they are fastidious organisms with exacting growth requirements and cultures are often negative if patient has received prior antibiotic therapy. Our patient had received treatment for two days before admission.

Glomerulonephritis is most commonly caused by Group B streptococcal infection but other organisms including a wide variety of bacteria, viruses and parasites are also known. Meningococci is one of the bacterial organisms known to cause this disease.

References

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