

Judicious re-examinations documenting the sequential changes in echogenicity within the hematoma with its ultimate resolution is virtually diagnostic of NAH and prevents unnecessary, expensive and invasive investigations and surgical intervention. USG has hence become the modality of choice both for the initial diagnosis and clinical follow-up of these lesions(6).

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Kala Azar – Diagnostic Dilemma

T.S. Raghu Raman
S.A.M. Ahuja
K.V.S. Mani
Y.P. Jalpota
S.K. Adaval

There has been sudden resurgence of kala azar in recent times. Most of the chil-

dren present no difficulties in diagnosis. However it is important to be aware of atypical clinical manifestations, leading to diagnostic problems. Three cases being reported illustrate the diagnostic difficulties, atypical hematological features, and rare complications in kala azar.

Case Reports

Case 1: A 12-year-old girl presented with fever, headache, altered sensorium, and cough with expectoration of five days duration. Examination revealed a malnourished and toxic child. Other signs were moderate pallor, generalized lymphadenopathy, bilateral fine crepitations on chest examination, no hepatosplenomegaly, and meningeal irritation with no neurological deficit. Investigations showed the level of hemoglobin 10 g/dl, total leucocyte count 11700/cu mm, with polymorphs 84%. The blood and sputum cultures were sterile, and the X-ray chest showed bilateral non homogeneous opacities suggestive of bronchopneumonia.

Management included ampicillin (100 mg/kg/day) and gentamicin (5 mg/kg/day). At the end of three weeks, the child continued to be febrile; clinical and radiologic signs of lower respiratory tract infection persisted. On empirical grounds a therapeutic trial with anti tubercular drugs was started. At the end of fifth week of

From the Departments of Pediatrics, Obstetrics, Medicine, and Pathology, Command Hospital, Bangalore and No. 5 A.F. Hospital C/o 99 APO.

Reprint requests: Dr. T.S. Raghu Raman, Department of Pediatrics, Command Hospital (A.F.), Agaram, Bangalore 560 007.

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hospitalization, episodes of abdominal pain, splenomegaly of 1 cm, Grade III/VI early systolic murmur, and atrial ectopics were observed. Following were the investigations at this stage: hemoglobin 11 g/dl, total leucocyte count 10800/cu mm, polymorphs 80%, platelet count 100,000/cu mm, reticulocyte count 2%, peripheral smear revealed target cells, toxic granulation in white cells; and repeat blood cultures were sterile. Urine examination was normal and urine culture was sterile. An ultrasound examination of the abdomen was normal, echocardiography did not show any vegetations, and the bone marrow was normal. Lymph node biopsy revealed features suggestive of non-specific reactive hyperplasia.

A repeat bone marrow study done at the end of seventh week showed scanty *Leishmania donovani* (LD) bodies. The child was treated with intramuscular sodium stibogluconate (20 mg/kg/day) for 20 days. The child showed good response and at the end of therapy, clinical examination, peripheral smear and bone marrow studies were normal. The child was readmitted after three months with high fever, generalized lymphadenopathy; peripheral smear showed anemia, polymorphonuclear leucocytosis, polychromic RBC and target cells. Bone marrow showed large number of LD bodies. She was treated for relapse of kala azar. However the response was poor. The illness was further complicated by disseminated intravascular coagulation with gastrointestinal hemorrhage, prolonged prothrombin time, thrombocytopenia and increased fibrin degradation products. The child succumbed to the illness within ten days.

Case 2: A 3-year-old girl was admitted with history of intermittent fever and rigors of five days duration. Associated clinical

features included pallor, deep icterus, hepatomegaly (6 cm, firm), splenomegaly (8 cm, firm). Investigations revealed a hemoglobin of 5.5 g/dl, total leucocyte count 4400/cu mm, polymorphs 10%, platelet count 40000/cu mm, serum bilirubin 6.0 mg/dl, serum aspartate transaminase 115 IU/L, serum alanine transaminase 68 IU/L, alkaline phosphatase 100/L, prothrombin time 16 seconds (control 11 seconds), hepatitis B surface antigen and aldehyde test were negative. Bone marrow showed LD bodies. She was treated with intramuscular sodium stibogluconate (20 mg/kg/day) for 20 days. Initially, the child showed deterioration with increasing jaundice (bilirubin 11 mg/dl), signs of encephalopathy, raised transaminases and increased prothrombin time. However, with conservative management she recovered over the next two weeks. At the end of three weeks, she was afebrile, organomegaly had regressed, and biochemical and hematological parameters were normal. The child has been under observation for last two years and she has remained free of relapse.

Case 3: A 10-year-old boy presented with fever, lassitude, cough with mucopurulent sputum, and occasional epistaxis of three months duration. The clinical signs included pallor, hepatomegaly (4 cm), splenomegaly (10 cm), and signs of pneumonitis in right lung. Investigations revealed a hemoglobin of 3.5 g/dl, total leucocyte count 3800/cu mm, polymorphs 46%, platelet count 80000/cu mm, microcytic hypochromic anemia; and X-ray chest showed consolidation in right lung. The aldehyde test was positive and the bone marrow smear was positive for LD bodies. The case was managed with intramuscular sodium stibogluconate (20 mg/kg/day) for 20 days. Subjective improvement with

regression of organomegaly and normal hematological and bone marrow studies was observed within three weeks.

Six months later the child came with fever, pallor, splenomegaly (22 cm) and hepatomegaly (5 cm). Investigations revealed a hemoglobin of 5.5 g/dl, total leucocyte count 2000/cu mm, polymorphs 24%, platelet count 50000/cu mm, and positive aldehyde test, bone marrow was hypercellular with large number of LD bodies. The child was managed for relapse of kala azar including intramuscular sodium stibogluconate (20 mg/kg/day) for 20 days and blood transfusions. At the end of three weeks he was afebrile. However, there was persistent splenomegaly (20 cm), pancytopenia with a total leucocyte count 2000/cu mm, hemoglobin 5 g/dl (despite repeated blood transfusions), polymorphs 20%, platelet count 50000/cu mm, reticulocyte count 4%, and markedly hypercellular bone marrow with absent LD bodies. Hypersplenism was diagnosed, and the poor response to conservative therapy prompted splenectomy. Post-operatively the hematological abnormalities reverted to normal. The histopathology of spleen did not show any LD bodies. The patient has remained asymptomatic for last two years.

Discussion

Majority of infections with visceral leishmaniasis present with irregular chronic fever, organomegaly and leucopenia. As in every other parasitic infection, the diagnosis is confirmed by presence of LD bodies in bone marrow, spleen or liver.

During epidemics, kala azar may present as an acute hemorrhagic condition with severe pancytopenia and absence of splenomegaly(1). Considerable leucopenia is typical; white blood cells are reduced

below 3000/cu mm in 95%, below 2000/cu mm in 75%, and below 1000/cu mm in 42% cases. There is associated eosinopenia(2). A case of kala azar has been reported with increasing leucocytosis and eosinophilia(3). However, persistent leucocytosis, polychromic RBCs and large number of target cells as seen in the first case has not been documented.

Generalized lymphadenopathy (as in Case 1) is unusual in Indian kala azar. It is a more common feature in Mediterranean, African, and Chinese kala azar. The histological picture may resemble tuberculosis or Hodgkin's disease. LD bodies may be very few and difficult to identify(2). Talukdar *et al.*(4) have however reported a case of kala azar with cervical lymphadenopathy.

Hyperbilirubinemia (as in Case 2) as a presenting feature in Indian kala azar has been documented only in 3% cases(5). All these cases presented with mild jaundice. Severe jaundice with hepatic decompensation is a feature of Italian kala azar and is a bad prognostic sign(2). Clinical profile, therapeutic response to antimonials, and absence of serologic evidence of hepatitis B suggest that hepatitis was secondary to kala azar. However, we did not screen for concomitant infections with hepatitis A and C viruses.

Secondary hypersplenism may occur in kala azar (as in Case 3) and the reported incidence is extremely low. In three studies involving 1495 cases of kala azar, only three cases of hypersplenism were documented(6-8). Splenectomy is useful in cases where conservative therapy has failed.

The diagnosis of kala azar proves difficult in early stages of sporadic infections due to atypical features. LD bodies may not be detectable, formal gel test negative,

and leucopenia absent. In endemic areas a high index of suspicion and appropriate investigations are required to establish the diagnosis.

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Acute Glomerulonephritis in Multi-Drug Resistant *Salmonella Typhi* Infection

A. Dhawan
R.K. Marwaha

Typhoid fever still remains a perplexing public health problem in the developing world. In 25-30% of cases, the illness is

complicated by involvement of one or more major organs of the body(1,2). The emergence, in recent months of a multi-drug resistant (MDR) strain of *Salmonella typhi*(3,4) is likely to increase the frequency of encounters with atypical manifestations of this disease. This report of a child with acute renal failure in association with MDR *S. typhi* infection aims to focus attention on renal involvement in enteric fever.

Case Report

A 10-year-old school-going boy, presented to the Pediatric services of Nehru Hospital, P.G.I.M.E.R, Chandigarh with an acute illness, characterized by remittent fever, of a fortnight's duration. A few days after the onset of the fever, he had intermittent, ill-localised pain in the abdomen and occasional vomitings. The appearance of hematuria and oliguria, 5 days prior to hospitalization, necessitated a referral from the local physician, who, however, failed to provide specific treatment details.

Examination revealed a febrile, ill-looking child with periorbital puffiness of both eyes, edema feet and deep acidotic breathing. He was normotensive (90/60 mm of Hg in the supine position) and had a hepatosplenomegaly of 3 cm and 1 cm below the costal margin, respectively.

The hemoglobin was 6.9 g/dl, white blood cell count $8.0 \times 10^9/L$ with 70% neutrophils and the platelet count was nor-

From the Departments of Pediatrics, Postgraduate Institute of Medical Education and Research, Chandigarh 160 012.

Reprint requests: Dr. R.K. Marwaha, Associate Professor, Department of Pediatrics, Postgraduate Institute of Medical Education and Research, Chandigarh 160 012.

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