4. Nagababu E, Rifkind JM. Heme degradation by reactive oxygen species. Antioxid Redox Signal 2004; 6: 967-978.

5. Reynolds CA. DNA-technology-based cellular assay used to measure specific biological activity in a wheatgrass extract. J Australasian Integrative Medicine Association, 2005.

Hepatic Abscess Caused by *Salmonella typhi*

Hepatic abscess, a rare complication of Salmonella infection is associated with high mortality(1). This is the second documented culture positive report of liver abscess due to *Salmonella typhi*(2).

An eight-year-old male was admitted with history of fever with chills for ten days and severe intermittent pain in the epigastrium for three days. On examination he had a temperature of 37.2ºC, no pallor, icterus or cyanosis. Vitals were stable.

Systemic examination revealed presence of fullness and lump in the epigastrium over the liver, accompanied by pain, tenderness and guarding. The liver was enlarged to 3.5 cm below the costal margin. Rest of the systemic examination was normal.

Ultrasound abdomen revealed multiple heterogeneous, hypoechoic spherical lesions in both lobes of the liver. A guided needle aspirate (6 mL) from the largest lesion (5.5 × 3.8 cm) in the left lobe was sent for microbiological evaluation. Empirical therapy was started keeping a possibility of a mixed bacterial and parasitic infection.

Liver function test during the second week revealed: total bilirubin; 0.4 mg/dL, AST; 174 IU/L, ALT; 112 IU/L, alkaline phosphatase; 294 mg/dL. Widal test result in the first week were: somatic O antigen (T_o) = 50, flagellar antigen of serotype Typhie (T_H):>800, flagellar antigen of serotype Paratyphi A (A_H) <50, B_H <50 and in the second week T_o = 100, T_H >800, A_H <50; B_H <50. Amebic sero-conversion observed during second week of illness and was negative thereafter. The abscess aspirate was reddish brown and on direct examination revealed multiple pus cells but no pathogens. Culture yielded *S. typhi.* The abscess became sterile on day fourteen. The bacterium was not isolated from any other sample from the patient. During the hospital stay the patient remained an-icteric. Epigastric tenderness and guarding disappeared after ten days; he became afebrile after twenty-four days of hospitalization and was discharged after six weeks. Thereafter, the patient remained asymptomatic during follow up.

Isolation of *S. typhi* from the aspirate in pure culture and presence of multiple abscesses, reddish brown aspirate and positive qualitative serology for *E. histolytica* were responsible for diagnosis of a mixed bacterial and parasitic infection (3).

However, a repeat serology for *E. histolytica* in the third week was non-reactive, which proved earlier sero-conversion to be an anamnestic response and use of color of the aspirate as contributory to provisional diagnosis, a questionable criterion.

Failure to perform a quantitative test (to observe four fold rise in antibody titers) was

responsible for the misdiagnosis and continuation of antiparasitic chemotherapy. Thus, making quantitative serology the only dependable test, as antigen detection kit in pus is not available and detection rate of trophozoites in cases with mixed amebic and parasitic infection is poor(3,4).

Sandhya Kabra,
Vishal Wadhwa,
Department of Microbiology,
Lok Nayak Hospital and
Maulana Azad Medical College,
New Delhi 110 002,
India.
E-mail: vishaI2870@yahoo.com

REFERENCES

1. Ronovito V, Bonanno CA. Salmonella hepatic abscess: An unusual complication of Salmonella carrier state? Am J Gastroenterol 1982; 77: 338-339.
2. Lasch EE. Liver abscess due to salmonellosis. A report on two cases in upper Volta. Israel J Med Sci 1966; 2: 377-379.
3. Soni PN, Hoosen AA and Pillay DG. Hepatic abscess caused by Salmonella Typhi. A case report and review of literature. Dig Dis Sci 1994; 39: 1694-1696.
4. Marr J, Haff R. Super infection of an amebic abscess by *Salmonella enteritidis*. Arch Intern Med 1971; 128: 291-297.

Allogeneic Peripheral Blood Stem Cell Transplantation in Aplastic Anemia

Allogeneic hematopoietic stem cell transplantation (HSCT) is the definitive therapy for severe aplastic anemia (SAA)(1,2). The major factors that limit the success of HSCT are graft rejection and graft-versus-host diseases (GVHD)(3,4). Engraftment depends on the conditioning regimen, GVHD prophylaxis, number of donor marrow cells infused and alloimmunization of the patient(3,4). Taking an aplastic anemia with septicemia for transplant is a very high-risk proposition.

A 11-year-old boy was diagnosed to have aplastic anemia and treated with ATG followed by Cyclosporin for 6 months. He was admitted at our center with high-grade fever of one week's duration with pancytopenia. Blood culture grew *E. coli* on two occasions and *Klebsiella* on one occasion. *Staphylococcus aureus* was grown from central line tip on two occasions. He was started on meropenem and teicoplanin for 3 weeks, when he developed maxillary sinusitis, for which he was started on amphotericin B. He continued to have intermittent fever. He was also given granulocyte infusions for one week. Blood culture was sterile after 3 weeks when a Hickman catheter insertion was done. During this time HLA typing of his 3-year-old sister was done and found to be 6 antigens matched. During the afebrile period patient was started on conditioning with fludarabine (30 mg/m^2 IV daily on day 2-4 busulfan (4 mg/kg/d q6h on day 5,6) and cyclophosphamide. 350 mg/m^2 IV daily on day 2-4. A peripheral blood stem cell (PBSC) harvest was done from the donor after 5 days of G-CSF at 10 mcg/kg/d. A volume of 110 mL was collected with an MNC of 3.65 × 108 cells/kg. The CD 34 cell dose was 1.34 × 106 cells/kg. GVHD prophylaxis was IV cyclosporin (CsA) at a dose of 3.0 mg/kg/d. G-CSF was started from