he complained of headaches, anorexia, abdominal pain and vomiting, and he developed a mild-to-moderate fever 2 days later.

On initial physical examination, his temperature was 38.5 °C, his heart rate was 98 beats/min, and mild jaundice of the skin and sclera was present. The abdomen was distended, and fluid was detected by abdominal percussion. The liver was palpable 4 cm below the costal margin, but the spleen was not palpable. There were no other abnormal findings in the physical examination.

Laboratory values were as follows: total bilirubin 6 mg/dL, direct bilirubin 2.4 mg/dL, aspartate aminotransferase 3660 U/L, alanine aminotransferase 2855 U/L, alkaline phosphatase 1306 U/L, lactate dehydrogenase 1010 U/L, total protein 6.0 g/dL, albumin 3.6 g/dL, sodium 134 mmol/L, potassium 4.1 mmol/L, serum amylase 22 U/L, 1-antitrypsin 3.8 g/L (normal range 2.0–4.0 g/L), and ceruloplasmin 300 mg/dL (normal range 150–550 mg/L). The results of urine analysis showed a +3 value for bilirubin, increased levels of urobilinogen but no protein. Sediment was normal. Both anti-HAV IgM and anti-HAV IgG antibodies were detected. Hepatitis B surface antigen and antibodies to hepatitis B, C and E were absent. Serologic analysis for cytomegalovirus and Epstein–Barr virus revealed no evidence of recent infection. Bilateral moderate pleural effusions were shown on chest X-ray on the sixth day of hospitalization but were not detected on initial chest radiogram. Abdominal ultrasonography showed ascites. An ultrasound study 15 days later showed complete resolution of both ascites and the pleural effusion. Levels of liver transaminases became normal 1 month later and the child recovered completely.

Ascites in liver diseases may occur as a result of venous and lymphatic obstruction or decreases in the osmotic pressure of plasma colloid, such as in hypoalbuminemia. A transient increase in portal venous or lymphatic pressure due to the compression of hepatic sinusoids may explain the occurrence of ascites in this case. Pleural effusion may be secondary to ascites due to fluid transport through the diaphragmatic lymphatics or direct passage through a diaphragmatic defect. Our patient developed bilateral pleural effusion 6 days after the diagnosis of ascites associated with type A viral hepatitis.

In conclusion, ascites and pleural effusion may exceptionally accompany hepatitis A infection in children, and presumably the same may occur in adults. Its appearance, however, does not indicate an unfavorable outcome.

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REFERENCES

Disseminated Nocardia asteroides complex infection in an immuno-compromised child

Nocardiosis is a rather frequent infectious disease that usually involves the respiratory tract [1–5]. Predisposing factors include malignancies, AIDS, organ transplantation, tuberculosis, drugs or alcohol abuse, lupus erythematosus, diabetes and chronic lung disease [2,4]. Very few cases have been reported in Greece, therefore we report a case of pulmonary nocardiosis in an 8-year-old immunocompromised female.

The girl was admitted to our hospital because of fever and cough. She had been suffering from autoimmune hemolytic anemia and thrombopenia (Evans’s syndrome) from the age of 1 year, receiving corticosteroids and cyclosporin periodically. A Mycobacterium avium complex (MAC) disseminated infection had taken place a year ago, which was treated with a combination of ethambutol, rifampicin, cycloserine and clarithromycin.

On admission, the laboratory studies showed WBC count of 27800/μL, with 87% neutrophils, haemoglobin 17.6 g/dL, erythrocytes sedimentation rate (ESR) 15 mm/h, CRP 55 mg/L, AST 62 U/L and ALT 64 U/L, urine protein 300 mg/dL with a few granular and rare waxy casts in the urinalysis. Blood and urine cultures were sterile and exclusive growth of Candida albicans was found in fecal cultures. The main roentgenographic findings included a centrally placed circumscribed shadow in the left middle lobe with mediastinal lymph node enlargement to the same side. Candida albicans was isolated in urine culture 2 weeks after the admission. Some subcutaneous scalp abscesses were noted 1 month after the admission and the ESR had increased to 110 mm/h.

Nocardia asteroides complex (NAC) was then isolated from blood, sputum and the exudate of the subcutaneous abscesses. The initial Gram stain preparations revealed Gram-positive, beaded, partially acid-fast, branched filaments. An empirical course of treatment was given, which consisted of amikacin, trimethoprim-sulphamethoxazole and ceftriaxone. In the following days a final identification of NAC was made, based on colony morphology and biochemical characteristics [4]. The susceptibility testing was performed by a modified disk diffusion...
method [6] and the isolate was found sensitive to amikacin, netilmicin, imipenem and resistant to trimethoprim-sulfamethoxazole, ampicillin, amoxicillin-clavulanic acid, all cephalosporins, tobramycin, gentamicin, chloramphenicol, clindamycin and ciprofloxacin. The therapy given initially was not changed because of its proved clinical efficacy in similar infections. Moreover, amikacin was included, to which the Nocardia isolate was highly sensitive. *Candida albicans* was isolated from multiple sites, including urine and blood, so amphotericin B was added to the therapeutic scheme.

The infection was not controlled and the child was moved to the Paediatric Intensive Care Unit (PICU) with severe respiratory failure, for which supportive care was supplied. Fever of 39 °C, gastroplegia, hepatomegaly and increase of WBCs to 41700/μL was found. Unfortunately the clinical condition did not improve, and 1 month after admission to the PICU, the child developed septic shock with multi-organ dysfunction, with a fatal outcome.

NAC is mostly isolated from lungs and *Nocardia brasiliensis* from primary skin lesions [2,4]. Pulmonary infections have been reported in children with serious prognosis such ours [7,8] and bacteremic nocardiosis disclosed here is rarely reported [9].

Many diagnostic difficulties and a delay in exact diagnosis of nocardiosis have been described [3]. In the present case, the Gram stain was very useful, orientating the diagnosis to nocardial infection and suggesting an empirical therapy. The administration of corticosteroids, because of the Evans’s syndrome, was a predisposing factor for nocardiosis, negatively affecting the cell-mediated immunity of the host.

The mortality from nocardiosis is high so this entity must always be seriously considered [2,3,5,8]. However, the fatal outcome observed in our child could not be exclusively attributed to the disseminated nocardial infection, but to the development of a systemic fungal infection, as well as to this child’s serious underlying condition.

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**REFERENCES**