

CASE REPORTS

5. Sung D II, Harisiadls L, Chang CH. Midline pineal tumors and suprasellar germinomas: Highly curable by irradiation. *Radiology* 1978; 128: 745-751.
6. Fujita T, Yameda R, Saitoh H, Itoh S, Nakai O. Intracranial germinoma and Down's syndrome: Case report. *Neurologia Medico-chirurgica* 1992; 32: 163-165.
7. Johnsen DE, Woodruff WW, Allm IS, Cera PJ, Funkhouser GR, Coleman LL. MR imaging of the sellar and juxtaseellar regions. *Radiographics* 1991; 11: 727-758.
8. Sanders WP, Chundi VV. Extra axial tumors including pituitary and parasellar. *In: William W, Orrison Jr MD, editor. Neuroimaging. Philadelphia: WB Saunders Company. 2000; p 660-696.*

Multiple Hypoechoic Lesions in Spleen and *Mycoplasma Pneumoniae* Infection

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An 8-year-old boy was admitted because of recurrent fever for 1 month with increased CRP and ESR. Ultrasound reviewed multiple, small, hypoechoic, rounded and wedge-shaped nodules with diffuse blood flow in spleen and enlarged abdominal lymph nodes. The spleen was enlarged and no echoic space was found in the largest lesion on 5th day. After a positive mycoplasma pneumoniae (MP) IgM was reported on 6th day, azithromycin was used intravenously. The temperature returned to normal and CRP and ESR improved in a short period. The

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*Manuscript received: October 23, 2003;
Initial review completed: January 23, 2004;
Revision accepted: September 27, 2004.*

lesions and lymphadenopathy disappeared and MP IgM antibody became negative 6 months later.

Key words: *Mycoplasma pneumoniae, Splenic lesions, Ultrasound.*

Case Report

An 8-year-old boy was admitted to hospital because of recurrent fever (normal in the morning and febrile during night, ranged from 39.5° C to 40.0° C) for 1 month. There was no cough except on the first and second day. No angina, tetter, arthralgia, dyspepsia, vomiting, night sweats, or abdominal pain were noted. He had no history of chronic liver disease or foreign travelling and no family history of serious illness. The patient had received intravenous cephalosporins or penicillins for 20 days, but did not recover.

Physical examination showed normal vital signs except for a temperature of 40° C. There was no skin lesion or peripheral stigmata of chronic liver disease. Several cervical lymph nodes were palpable with 0.5-1.0 cm in diameter, and no axillary or inguinal nodes were noted. The head, ears, eyes, nose, throat, cardiac and pulmonary examinations were all normal. The spleen was not palpable. The liver span was 9-10 cm by percussion, easily felt

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1cm with soft and smooth edge below the right costal margin in the midclavicular line. The neurological examination was normal.

Laboratory findings

He had increased C-reactive protein (CRP: 30 mg/L) and erythrocyte sedimentation rate (ESR: 72 mm/hr). Other results of routine laboratory studies revealed: Hemoglobin, 104-105g/L; WBC, $5.3-7.5 \times 10^9/L$; lymphocytes, 21.3-39.2%; polymorphonuclear leukocytes, 76.4 - 52.5%; Platelets, $349-359 \times 10^9/L$; eosinophils, 75/mL. Flow cytometry analysis results: CD3, 30.22%; CD4, 11.48%; CD8, 12.18%; CD2, 49.23%; CD5, 0.15%; CD7, 37.84%; CD1a, 0.40%; CD25, 3.83%; CD56, 18.56%; CD19, 0.9%; CD34, 1.01%; HLA-DR, 25.02%; TCRa/b, 20.31%; TCRr/d, 7.54%. No malignant clone was identified.

The bone marrow seen with light microscopy had normal cellularity; the erythrocytic series increased and significant granulocytic hyperplasia was present. Granules increased and vacuolization was found in the cytoplasm of some granulocytes. Lymphocytic series were hypoplastic, but the numbers of megakaryocytes were normal.

Viral serologies were negative for hepatitis A, hepatitis B, hepatitis C, hepatitis D, hepatitis E and HIV markers. Other laboratory examination results were normal or negative, including chest X-ray, ECG, serum levels of antinuclear antibody, tuberculosis antibody, rheumatoid factor, antistreptolysin O antibody, Epstein-Barr virus antibody, Purified protein derivative, Chlamydia pneumoniae antibody, somatic O antigens and flagellar H antigens of Salmonella, blood culture, bone marrow culture, total protein, total bilirubin, alkaline phosphatase, alanine aminotransferase, total IgG, IgA, IgM, IgE, C3 and C4.

Imaging findings

An abdominal computerized tomography (CT) examination shown several low-attenuation lesions diffusely located in the spleen (*Fig 1*). Abdominal ultrasound (US) with 3.5 MHz convex transducer was also done. The length of the liver (9.1 cm) and the width of portal trunk (0.7 cm) were all in normal ranges. The length in the major axis (7.8 cm) and thickness of the spleen (3.2 cm) were in normal ranges as well. However, many small, hypoechoic, rounded, wedge-shaped and well-defined nodules in spleen were found as in diffuse infiltration (*Fig 2*). Lesion diameter was about 0.5-1.0 cm and the biggest was 1.20×1.06 cm. Blood flow was found in these lesions by color Doppler flow imaging. Enlarged abdominal lymph nodes, including para-aortic lymph nodes (the largest 1.54×0.91 cm) and mesenteric lymph nodes (the largest 1.03×0.65 cm), were also found.

Clinical course

On the fifth day of hospitalization, the spleen was found to be enlarged (the thickness of spleen was 3.6 cm) and no echoic space, which implied internal fluid, was found in these lesions. After a positive Mycoplasma pneumoniae (MP) IgM (1 : 32) was reported

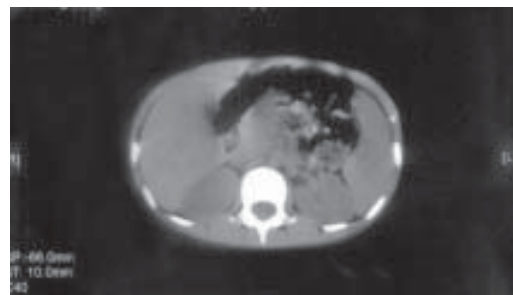


Fig. 1. Abdominal computerized tomography examination. Unenhanced CT image showing several low-attenuation lesions diffusely in spleen.

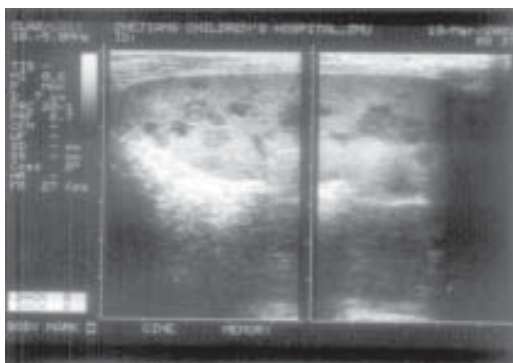


Fig. 2. Abdominal ultrasound examination showing many small, hypoechoic, rounded, wedge-shaped and well-defined nodules diffusely in spleen.

on the sixth day, azithromycin (10 mg/kg.day for 5 days) was used intravenously and the temperature was returned to normal in 3 days. CRP (<8 mg/dL) and ESR (15 mm/hr) recovered in 1 week. The size of spleen (the thickness of spleen was 2.9 cm) and splenic lesions (the biggest lesion was 1.05 × 0.74 cm) decreased in 2 month and 10 days later. The lesions and enlarged lymph nodes disappeared, MP IgM antibody became negative (<1 : 8) and IgG antibody was positive (1 : 32) in 6 months later.

Discussion

Mycoplasma pneumoniae (MP) is a frequent cause of community-acquired respiratory infections in children and adults, especially in school-aged children(1-2). It is also responsible for a wide spectrum of non-pulmonary manifestations including hematological, gastrointestinal, renal, cardiac and central nervous system diseases(3-6). Certain observations are suggestive and can be helpful to the physician, for example, pneumonia in young adults, serum cold hemagglutinins in a titer of 1 : 64, a positive IgM MP antibody, polymerase chain reaction or effective treatment with macrolides(7,8).

The cause of hospitalization in this case was mainly the persisting fever for 1 month. Imaging findings included splenomegaly and multiple hypoechoic nodules within spleen. To present knowledge, no previous report suggested that multiple hypoechoic nodules in spleen without respiratory manifestation are associated with MP infection. In this case, however, some clinical feature and laboratory results supported MP infection, including (1) a school-aged children, (2) fever and cough in the first and second days, (3) positive and increasing MP-IgM antibody in short period of time, and (4) effective treatment with azithromycin and resistance to penicillins and cephalosporins.

As we know, there are two mechanisms for the development of MP associated disease: direct invasion mechanism and immune-mediated mechanism(6,9). Because azithromycin was very effective in this case and the fever recovered in three days, we postulated that direct organism invasion rather than autoimmune mechanisms played an important role in the lesion of spleen. The limitation of this report was that pathologic biopsy of the lesions in spleen were not done, so we are not sure that these hypoechoic nodules in spleen is a lymphocytic proliferating lesion or an abscess due to cell-mediated immune response. As no abdominal pain and no capsule of these hypoechoic nodules were detected, we suspected these nodules were not abscess, but focal nodular hyperplasia as a result of direct MP invasion. Similar lesion in liver had been found by ultrasound because of focal nodular hyperplasia(10).

Contributors: Both authors were involved in case management, search of literature and writing the report.

Funding: None.

Competing Interests: None stated.

REFERENCES

1. Wattanathum A, Chaoprasong C, Nunthapisud P, Chantaratchada S, Limpairojn N, Jatakanon A, *et al.* Community-acquired pneumonia in southeast Asia: The microbial differences between ambulatory and hospitalized patients. *Chest* 2003; 123: 1512-1519.
2. Korppi M, Heiskanen-Kosma T, Kleemola M. *Mycoplasma pneumoniae* causes over 50% of community-acquired pneumonia in school-aged children. *Scand J Infect Dis* 2003; 35: 294.
3. Paz A, Postasman I. Mycoplasma-associated carditis. Case reports and review. *Cardiology* 2002; 97: 83-88.
4. Minami K, Maeda H, Yanagawa T, Suzuki H, Izumi G, Yoshikawa N. Rhabdomyolysis associated with *Mycoplasma pneumoniae* infection. *Pediatr Infect Dis J* 2003; 22: 291-293.
5. Smith R, Eviatar L. Neurologic manifestations of *Mycoplasma pneumoniae* infections: Diverse spectrum of diseases. A case of six cases and review of literature. *Clin Pediatr (Phila)* 2000; 39: 195-201.
6. Bitnun A, Ford-Jones E, Blaser S, Richardson S. *Mycoplasma pneumoniae* encephalitis. *Semin Pediatr Infect Dis* 2003; 14: 96-107.
7. Templeton KE, Scheltinga SA, Graffelman AW, Van Schie JM, Crielaard JW, Sillekens P, *et al.* Comparison and evaluation of real-time PCR, real-time nucleic acid sequence-based amplification, conventional PCR, and serology for diagnosis of *Mycoplasma pneumoniae*. *J Clin Microbiol* 2003; 41: 4366-4371.
8. Daxboeck F, Krause R, Wenisch C. Laboratory diagnosis of *Mycoplasma pneumoniae* infection. *Clin Microbiol Infect* 2003; 9: 263-273.
9. Chiba H, Pattanajitvilai S, Mitsuzawa H, Kuroki Y, Evans A, Voelker DR. Pulmonary surfactant proteins A and D recognize lipid ligands on *Mycoplasma pneumoniae* and markedly augment the innate immune response to the organism. *Chest* 2003; 123: 426S.
10. Shirkhoda A, Farah MC, Bernacki E, Mardrazo B, Roberts J. Hepatic focal hyperplasia: CT and sonographic spectrum. *Abdom Imaging* 1994; 19: 34-38.