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The coexistence of hepatitis A and infectious mononucleosis

Hepatit A ve infeksiyoz mononükleoz birlikteliği

Gül KARAGÖZ, Öznur AK, Serdar ÖZER

Department of Infectious Diseases and Clinical Microbiology, Kartal Training and Research Hospital, İstanbul

Hepatitis A is an acute, self-limited disease that spreads predominantly by the fecal-oral route. Hepatitis A characteristically has an acute, sudden influenza-like onset with a prominence of myalgia, headache, fever and malaise. Infectious mononucleosis is an acute illness characterized clinically by sore throat, fever and lymphadenopathy. The virus usually spreads from person to person by close contact with nasopharyngeal secretions. In this case the coexistence of both diseases in the same patient is found interesting.

Key words: Hepatitis A, infectious mononucleosis

Hepatit A başlıca fekal - oral yolla bulaşan, akut, kendini sınırlayan bir hastalıktır. Hepatit A karakteristik olarak kas ağrısı, baş ağrısı, ateş ve yorgunluk gibi semptomlarla influenza benzeri başlangıç gösterir. İnfeksiyoz mononükleoz, klinik olarak boğaz ağrısı, ateş ve lenfadenopati ile karakterize akut bir hastalıktır. Virus genellikle yakın temasta olan insanlarda nazofarengeal sekresyonla bulaşır. Bu vakada aynı hastada iki hastalığın birlikte görülmesi ilginç bulunmuştur.

Anahtar kelimeler: Hepatit A, enfeksiyoz mononukleoz

INTRODUCTION

Hepatitis A is an acute, self-limited disease that spreads predominantly by the fecal-oral route. Transmission is generally limited to close contacts. Three other epidemiologic sources have been shown to be important in the spread of hepatitis A: exposure to children in day care centers, male homosexuality and intravenous drug addiction. Hepatitis A characteristically has an acute, sudden influenza-like onset with a predominance of myalgia, headache, fever and malaise (1). Hepatitis clinically occurs after this prodromal phase. These nonspecific symptoms may sometimes cause problems in differential diagnosis. Infectious mononucleosis is an acute illness characterized clinically by sore throat, fever and lymphadenopathy; serologically by the transient appearance of heterophile antibodies; and hematologically by a mononuclear leukocytosis that consists partly of atypical lymphocytes. The virus usually spreads from person to person by close contact with nasopharyngeal secretions (2). The diagnosis of both diseases is confirmed by serologic tests. In this case we describe a patient whom both hepatitis A and infectious mononucleosis existed concomitantly.

CASE REPORT

A 22-year-old male patient working as a discjockey was admitted to our hospital with symptoms of fatigue, myalgia, fever, nausea, vomiting, dark urine, and loss of taste for cigarettes for 10 days.

The patient had type 1 diabetes mellitus from 19 years of age, for which he was receiving insulin treatment. Alcohol consumption had been approximately 30-35 ml once a week for the past four years, and he had smoked two packs of cigarettes per year for about six years. He had no history of dental treatment, blood transfusion or sexual activity in the last six months.

On physical examination, temperature was 39°C, pulse 84 beats/min and respiratory rate 16/min; blood pressure was 110/60 mmHg. Bilateral posterior cervical adenopathy was found, and they were mobile and not tender. Tonsils were enlarged

Address for correspondence: Gül KARAGÖZ
Kartal Training and Research Hospital, Department of Infectious Diseases and Clinical Microbiology, 81327 Kartal, İstanbul, Turkey
Phone: +90 216 441 39 00/1827
E-mail: gulkaragozmd@hotmail.com

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and hyperemic. Scleral jaundice was prominent. The auscultation of heart and lung were normal. The abdomen was soft. The liver edge descended 1-2 cm below the right costal margin; the spleen was not felt. The results of laboratory tests are reported in Tables 1 and 2; alanine transaminase (ALT) and aspartate transaminase (AST) enzyme

Table 1. Hematologic laboratory values in the patient with hepatitis A and infectious mononucleosis

	1st day	4th day	Normal Ranges
Hematocrit (%)	37.8	34	37-47
White cells (per/mm^3)	2300	4200	4800-10800
Differential count (%)			
Neutrophiles	44	40	43-65
Lymphocyte	42	48	5.5-20.5
Monocytes	12	12	5.5-11.7
Platelets (per/mm^3)	64,000	117,000	130,000-400,000
Prothrombin time (second)	10.3	9.6	11.3-16.1
Partial-thromboplastin time (second)	23	27	24.35-31.25

levels are shown in Figure 1. At this point we considered the case as viral hepatitis. Hepatitis A virus antibody IgM (Anti-HAV IgM), hepatitis B surface antigen (HbsAg), hepatitis B core antibody IgM (Anti-HBcIgM), and hepatitis C antibody (Anti-HCV) were searched. Anti-HAV IgM was found positive. At follow up, all symptoms except fever improved. Temperature increased to 38.5-39°C on the third hospital day. Because of this peak in fever, ampicillin/sulbactam 375mg b.i.d was started empirically. A maculopapular rash, especially on the trunk, developed after a dose of antibiotic, and splenomegaly was found as a new sign on physical examination, which was confirmed by abdominal ultrasonography. Epstein-Barr virus (EBV) infection was considered because of fever, splenomegaly, cervical lymphadenopathy and rash after ampicillin/sulbactam treatment. Heterophile antibodies were negative, but serum IgM antibodies against EBV capsid antigen (EBV VCA IgM) was positive.

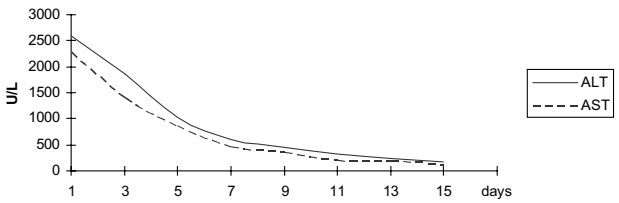

Figure 1. Transaminase levels decreasing in time (days)

Table 2. Blood chemical values in the patient with hepatitis A and infectious mononucleosis on the 1st and 10th day

	1st day	10th day	Normal Ranges
Glucose (mg/dl)	234	291	76-110
Protein (g/dl)	5.6	6.6	6.6-8.7
Albumin	2.6	2.7	3.4-4.8
Globulin	3	3.9	1.9-4.5
Bilirubin (mg/dl)			
Total	8.7	7.3	0.0-1.1
Conjugated	7.2	3.7	0.00-0.30
AST (U/L)	2277	203	0-32
ALT (U/L)	2587	331	0-31
ALP (U/L)	1094	1134	0-270
GGT (U/L)	647	337	7-32
Urea nitrogen (mg/dl)	18	20	10-50
Creatinine (mg/dl)	0.5	0.5	0.00-0.95
Na (mmol/L)	134	134	133-150
K (mmol/L)	2.6	5.1	3.3-5.1

EBV VCA IgM and HAV IgM were both positive in this patient. It was accepted that the cause of this clinical picture was HAV, but that the next step of the new finding was due to concomitant EBV infection. Treatment with ampicillin/sulbactam was stopped after the development of rash and the patient was followed by symptomatic treatment.

On the 15th hospital day his physical examination was nearly normal and ALT, AST, and bilirubin values had decreased and he was discharged from the hospital. Two weeks later his physical examination and laboratory findings were normal.

DISCUSSION

HAV and EBV infections occur most frequently in children and adolescents. IgG antibodies to EBV and HAV can be found positive in more than 80% of the population in Turkey (3). Clinically, hepatitis A has an influenza-like onset. Fever (up to 40°C) may be accompanied by chills, mild headache, malaise and fatigue. Patients with hepatitis A often lose their taste for cigarettes. Hepatocellular enzyme levels, especially levels of ALT and AST, are sensitive for parenchymal liver damage but are not specific for hepatitis A. Atypical lymphocytes account for less than 10% of the leukocytes. Diagnosis of acute hepatitis A is most commonly confirmed by detection of specific IgM antibodies (4). However, in infectious mononucleosis, sore throat, fever and lymphadenopathy are dominant. Liver functions consist largely of self- limited elevations of hepatocellular enzyme levels in 80%, and jaundice is present in approximately 5% of cases in infectious mononucleosis (5).

In this case, the existence of nausea, vomiting, dark urine, jaundice, loss of taste for cigarettes and positive HAV IgM was thought to be due to hepatitis A. But persisting fever for three days up to 39°C, lymphadenopathy, sore throat and pruritic maculopapular rash with ingestion of ampicillin/sulbactam, seen in 90%-100% of patients, led us to consider infectious mononucleosis. As anticipated, EBV VCA IgM was found positive. Heterophile antibodies were negative at the first week but at the third week they became positive. In this patient, infectious mononucleosis and hepatitis A coinfection was considered, because the incubation period of hepatitis A is 15-45 days and for EBV infection 30-50 days. In spite of differences in their transmission routes, their incubation periods are similar to one other. For this reason we thought that these viruses were acquired at nearly the same time. Hepatitis A infection in chronic carriers of hepatitis C virus may be more severe and more likely to result in fulminant hepatitis A (6). But there is no published data about the prognosis of coinfection of HAV and infectious mononucleosis in the literature. However, this case improved without any complications. Finally, the coexistence of these infections was found interesting.

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