Epstein-Barr virus (EBV) is a member of the herpesviridae family and one of the most common human viruses. It has been established as the etiological agent of infectious mononucleosis (IM). IM is common worldwide in distribution and appears in all age groups especially late adolescents or early adulthood. It is largely subclinical in early childhood. In addition EBV appears to play an important role in Burkitt’s lymphoma and nasopharyngeal carcinoma. Largely of self-limited elevations of hepatocellular enzyme levels are shown present in 90 percent of the cases of EBV infection-induced IM but EBV causes acute hepatitis rarely. Jaundice is distinctly uncommon so it isn’t presentation symptom generally. The jaundice is seen 5 percent of the cases (1,2). We describe in a patient who presented with jaundice due to serologically confirmed acute infection with EBV.

CASE
A 20 years old male patient who had weakness, fatigue, yellowness of sclera and right hipocondrium pain for two weeks admitted to the Infectious Disease clinic. He had no significant past medical history. Jaundice of sclera and skin and tenderness of fist percussion over the liver were found on physical examination. Laboratory studies showed midly elevated levels of alanine aminotransferase (165 U/L), aspartate aminotransferase (60 U/L), gamma glutamile transferase (118 U/L), conjugated bilirrubine (1.6 mg/dl), total bilirrubine (4 mg/dl) and normal level of alkaline phosphatase (272 U/L). The hemogram was found normal. Serological markers for hepatitis A, B, C, D and CRP were negative. Protrombine time was normal. Brucella agglutination tests (Rose Bengal and Wright) and Gruber Widal were negative. Because the patient refused hospitalization, he was told to rest at home. He returned to the clinic seven days later. He had had the additional complaint of a sore throat for past two days. His temperature was 37.0°C. Jaundice of sclera and skin, hepatomegaly and erythema on phrynx were found on physical examination. Laboratory studies did not show important alteration. Ultrasonography (USG) of abdomen showed reduced echogenicity on the liver. The hemogram showed normal white blood cell (10x10³/mm³) but 15 % atypical lymphocytes. Serological markers for CMV (anti-CMV IgM ve IgG) were negative but EBV (anti-EBV VCA IgM and IgG, Paul Bunnel and monospot tests ) were positive. So the diagnosis of HBV hepatitis...
was established. He was told to rest at home for ten days and to come again for a control. He returned to the clinic 15 days later. The following results were revealed: ALT: 35 U/L, AST: 25 U/L, ALP: 250 U/L, conjugated bilirubine 0.5 mg/dl, total bilirubine 1.2 mg/dl and white blood cell count 7840/mm$^3$. No atypical lymphocytes were seen on the hemogram.

**DISCUSSION**

EBV infection is common all over the world. In the United States 50 % of the children up to the age of five and 95 % of adults carry the disease. In children these infections usually cause no symptoms. When the infection with EBV occurs during adolescents or young adulthood it causes IM at the rate of 35 % to 50 %. Classic EBV infection-induced IM usually begin with weakness, sweats, anorexia and myalgias. This period is called the prodrome period and lasts several days. Then fever, sore throat and swollen lymph glands develops. In 50 % of the cases splenomegaly and 10 % of the cases hepatomegaly may be detected (1-3).

In our patient first symptoms were weakness and jaundice. So we didn’t think IM at first application. But seven days later sore throat and atypical lymphocytes on peripheral blood smear appeared so we thought IM. In patients with IM the lymphocyte count constitutes 50% of the total white blood cell count. Of this 10-30 % is atypical lymphocytes (4). In our patient’s peripheral blood smear were 55 % lymphomonocytosis and atypical lymphocytes. And then serological tests for EBV infection performed. The result of these tests were confirmed the diagnosis of IM. On four cases of EBV infection presented with jaundice, Albornoz et al. reported that the most useful laboratory finding was atypical lymphocytes (5). In adults and adolescents IM is frequently symptomatic (1). Our patient was an adult and IM was symptomatic in him, too. So in patients that presented with symptoms of jaundice, peripheral blood smear must be examined. We think this simple examination is very useful for diagnosis.

Posterior cervical adenopathy is present 80-90 % of the cases of IM (1). But we didn’t detect it on our patient. Likewise Albornoz et al, didn’t detect adenopathy on their four patients (5). Adenopathy does not occur in 10-20 % of the cases of IM so if other symptoms and signs are considered to cause IM, lack of adenopathy shouldn’t cause to avoid the diagnosis of IM.

Liver involvement is nearly universal in healthy persons with EBV infection-induced IM. But severe hepatitis as in two patients described by Ghosh et al, is uncommon (6). Presentation of IM with only jaundice and hepatitis and without other symptoms is very rare. Cholestatic hepatitis due to EBV infection is infrequently reported (4,7,8). IM began on our patient with jaundice, weakness and right hipocondrium pain and didn’t lead to cholestatic and fulminant hepatitis. It was self-limited. However, patients at older ages should be closely examined considering the fulminant hepatitis and long-term cholestasis (9,10).

In conclusion; in differential diagnosis of jaundice and hepatitis, IM associated EBV hepatitis should be kept in mind. The patient may not have typical symptoms for IM and may have only hepatitis clinic. In a condition as above, IM should be taken into consideration even if physical examination results aren’t parallel. So, peripheral blood smear must be examined carefully while drawing a result.

**REFERENCES**


7. Hinedi TB, Koff RS. Cholestatic hepatitis induced by Epstein-Barr virus infection
