

Infectious mononucleosis presenting as abdominal pain

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Received - 23 October 2019

Initial Review - 09 November 2019

Accepted - 23 November 2019

ABSTRACT

Infectious mononucleosis (IMN) is commonly seen with Epstein-Barr virus (EBV) infection. Severe abdominal pain can occur in IMN due to splenic rupture. However, abdominal pain alone is uncommon in IMN. Here, we report the case of a 42-year old male who presented with IMN induced abdominal pain in the absence of splenic rupture; which is an uncommon scenario.

Keywords: Abdominal pain, Epstein-Barr virus, Infectious mononucleosis, Splenomegaly.

Infectious mononucleosis (IMN) is characterized by sore throat, fever, lymphadenopathy and atypical lymphocytosis. It is commonly caused by the Epstein-Barr virus (EBV) belonging to the Herpes viridae family. It has an incubation period of 4 to 6 weeks and the symptoms usually last for 2 to 4 weeks. The infection is transmitted by contact with oral secretions and affects mainly the younger age groups [1]. Though abdominal pain is a feature of IMN, its occurrence in the absence of splenic rupture is uncommon. Here, we report the case of a 42-year-old male presenting with IMN induced abdominal pain in the absence of splenic rupture.

CASE REPORT

A 42-year-old male, with no prior comorbidities, presented to the emergency department with complaints of dull, aching abdominal pain of 2 hours duration, which was associated with nausea and non-projectile vomiting (3 to 4 episodes). He also had fever for the last 1 week. There was no history of infective contacts, recent travel or illicit drug use.

On examination, he was febrile (100°F) with vitals being stable. The oral cavity was normal with no lymphadenopathy. His abdominal examination revealed the left hypochondrial tenderness with no radiation of the pain. There was no abdominal guarding or rigidity. Splenomegaly of 2 cm was present. Kehr's sign was negative. Other systemic examinations were normal.

His complete blood counts showed lymphocytic leucocytosis (12500/mm³ with neutrophils 21% and lymphocytes 79%) and mild thrombocytopenia (125000/mm³). His liver function tests (LFTs) showed elevated liver enzymes (aspartate aminotransferase [AST] 185 IU/L, alanine transaminase [ALT] 220 IU/L) with total bilirubin 1.5 mg/dL, direct bilirubin 0.8 mg/dL, alkaline phosphatase (ALP) 195 IU/L and albumin: 3.3 g/dL. Serum

amylase and lipase were 110 U/L (20-96) and 50 U/L (3-43), respectively. His renal functions, plasma ammonia, prothrombin time (PT)/International normalised ratio (INR), activated partial thromboplastin time (aPTT) and electrolytes were normal.

Smear for malarial parasite, dengue and leptospira serology, Weil Felix test, and viral markers (HIV, HBsAg, anti HCV, anti-HEV IgM, HAV IgM, HSV 1 and 2 IgM) were negative. Ultrasound of the abdomen showed splenomegaly (14 cm) with no signs of rupture and mild hepatomegaly (16 cm). Chest X-ray and echocardiography were normal. Blood cultures were sterile. His peripheral blood smear showed large reactive lymphocytes with indented nuclei and abundant basophilic cytoplasm, suggestive of IMN (Fig.1). The heterophile antibody test was negative. EBV viral capsid antigen (VCA) IgM was positive (and IgG was negative) and EBV nuclear antigen (EBNA) IgG was negative. IgM and IgG for cytomegalovirus (CMV) were negative.

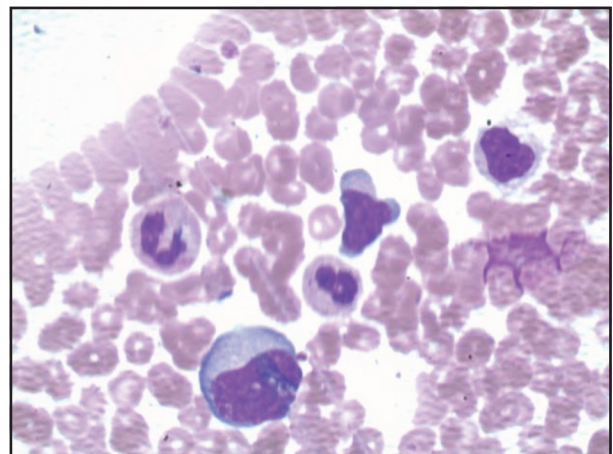


Figure 1: Peripheral blood smear showing large reactive lymphocytes with indented nuclei and abundant basophilic cytoplasm.

He was managed mainly with intravenous fluids and tramadol. Oral paracetamol (500 mg) was given during fever episodes. On day 3 of admission, his WBC got elevated (22400/mm³ with neutrophils 20% and lymphocytes 80%) with thrombocytopenia (11400/mm³) and deranged LFTs (total bilirubin 3.5 mg/dL, direct bilirubin 1.8 mg/dL, AST 205 IU/L, ALT 142 IU/L, ALP 178 IU/L and albumin: 3.0 g/dL). PT/ INR were normal.

He continued to have fever spikes (101°F) 2 to 3 times daily. By day 9, he became afebrile with no abdominal pain. His WBC showed decreasing trend (11200/mm³ with neutrophils 40%, lymphocytes 57% and eosinophils 3%) along with platelet count (151000/mm³) and LFTs (total bilirubin 2.0 mg/dL, direct bilirubin 1.2 mg/dL, AST 120 IU/L, ALT 79 IU/L, ALP 170 IU/L and albumin: 3.3 g/dL). His repeat ultrasound abdomen continued to show splenomegaly (13.5 cm). He was discharged; and on review after 1 week, the patient was asymptomatic with normal complete blood counts and LFTs. Ultrasound abdomen after 1 month showed normal spleen.

DISCUSSION

Epstein-Barr virus (EBV) infections are common in childhood and can be asymptomatic or present as pharyngitis with or without tonsillitis. Adults usually present as IMN with fever, malaise, myalgia and fatigue. Pharyngitis, lymphadenopathy, splenomegaly and atypical lymphocytes are uncommon among elderly patients. The skin manifestations include erythema nodosum, erythema multiforme and morbilliform or papular rash [1].

The blood investigations will show leucocytosis with lymphocytosis (>10% atypical lymphocytes), along with neutropenia and thrombocytopenia. The atypical lymphocytes are enlarged with abundant cytoplasm and vacuoles. LFTs show elevated aminotransferases and alkaline phosphatase. Cholestatic hepatitis can result in direct hyperbilirubinemia [2]. A positive heterophile antibody test (titers \geq 40 fold) is indicative of acute EBV infection. The test is positive in about 40% of patients during the first week of infection and in 80 to 90% during the third week and continues to remain positive for about 3 months after the onset of illness. About 90% of patients show raised titers of VCA IgM and IgG antibodies. The seroconversion to positive EBNA is also suggestive of acute infection. About 70% of IMN patients will test positive for early antigen diffuse (EA-D) antibodies, especially in severe cases. The association of the virus with several malignancies can be demonstrated with the help of EBV DNA, RNA or proteins [1].

The condition is usually self-limiting, and managed with supportive measures, rest and analgesics. Excessive physical activity should be avoided during the first month in view of the possibility of splenic rupture. Severe airway obstruction due to tonsillar hypertrophy, thrombocytopenia, autoimmune hemolytic anemia and hemophagocytic lymphohistiocytosis are managed with prednisolone. The role of steroids in EBV

hepatitis is controversial [3,4]. Acyclovir has been effective in oral hairy leukoplakia but has no significant clinical impact in the treatment of IMN [1]. Ganciclovir may be administered in cases of severe IMN hepatitis [5]. The complications include splenic rupture, meningitis and encephalitis, hemiplegia, upper airway obstruction, pneumonia, psychosis, autoimmune hemolytic anemia, myocarditis or pericarditis, genital ulcerations, vasculitis, bacterial superinfection, hepatitis and fulminant hepatic failure [1,6].

EBV accounts for about 90% of IMN cases, while 5 to 10% are due to CMV. The differential diagnosis includes rubella, herpes infection, lymphoma, viral hepatitis, streptococcal pharyngitis, HIV and toxoplasmosis [1]. Splenic rupture can present as an acute abdomen [7]. However, abdominal pain in uncomplicated IMN, even in the presence of splenomegaly, is an uncommon scenario; and is seen in only 1 to 2 % of cases [8]. The patients with splenic rupture, generally, undergo emergency splenectomy. Histologically, the spleen may remain abnormal for a long time, even after IMN symptoms improve. Hence, patients should be advised to avoid physical activity for 2 to 3 months after infection, and up to 6 months in case of athletes [8]. Acute acalculous cholecystitis can occur in EBV infection due to the direct invasion of the gallbladder mucosa and bile stasis, leading to gallbladder inflammation [9]. EBV may cause mesenteric adenitis either directly through infection of target B cells or indirectly by promoting immune T-cell expansion [10]. Acute appendicitis and severe gastritis have also been reported with IMN infection [11,12].

CONCLUSION

Infectious mononucleosis can present with severe abdominal pain due to splenic rupture. Our patient had presented with the left hypochondrial pain, nausea and vomiting. His ultrasound abdomen revealed splenomegaly with mild hepatomegaly. On further investigation, he was found to have IMN due to EBV infection. Abdominal pain in the absence of splenic rupture in IMN infection is an uncommon scenario. Since the spleen takes time to become normal, patients should be advised to restrain from physical activity in order to avoid splenic rupture.

REFERENCES

1. Cohen JI. Epstein-Barr Virus Infections, Including Infectious Mononucleosis. In: Kasper, Fauci, Hauser, Longo, Jameson, Loscalzo (ed). Harrison's Principles of Internal Medicine. 19th ed. McGraw Hill education. pp. 1186-1190.
2. Salva I, Silva IV, Cunha F. Epstein-Barr virus-associated cholestatic hepatitis. *BMJ Case Rep.* 2013;doi: 10.1136/ber-2013-202213.
3. Khoo A. Acute cholestatic hepatitis induced by Epstein-Barr virus infection in an adult: a case report. *J Med Case Rep.* 2016;10:75.
4. Kang MJ, Kim TH, Shim KN, Jung SA, Cho MS, Yoo K, *et al.* Infectious mononucleosis hepatitis in young adults: two case reports. *Korean J Intern Med.* 2009;24:381-7.
5. Vine LJ, Shepherd K, Hunter JG, Madden R, Thornton C, Ellis V, *et al.* Characteristics of Epstein-Barr virus hepatitis among patients with jaundice or acute hepatitis. *Aliment Pharmacol Ther* 2012;36:16-21.

6. Zhang W, Chen B, Chen Y, Chamberland R, Whyte AF, Craig J, *et al.* Epstein-Barr Virus-Associated Acute Liver Failure Present in a 67-Year-Old Immunocompetent Female. *Gastroenterology Res.* 2016;9:74-8.
7. Farley DR, Zietlow SP, Bannon MP, Farnell MB. Spontaneous rupture of the spleen due to infectious mononucleosis. *Mayo Clin Proc.* 1992;67:846-53.
8. Rutkow IM. Rupture of the spleen in infectious mononucleosis: a critical review. *Arch Surg.* 1978;113:718-20.
9. Prassouli A, Panagiotou J, Vakaki M, Giannatou I, Atilakos A, Garoufi A, *et al.* Acute acalculous cholecystitis as the initial presentation of primary Epstein-Barr virus infection. *J Pediatr Surg.* 2007;42:E11-3.
10. Yarchoan M, Flynn D, Hyder I, Bose S. An Uncommon Presentation of a Common Illness: Epstein-Barr Virus (EBV) Infection Case Report and Review of Literature. *Cureus.* 2011;3:e23.
11. Navidad AL, Domingo P, Cadafalch J, Farrerons J, Allende L, Bordes R. "Acute Appendicitis Complicating Infectious Mononucleosis: Case Report and Review." *Reviews of Infectious Diseases.* 1990;12:297-302.
12. Zhang Y, Molot R. Severe gastritis secondary to Epstein-Barr viral infection. Unusual presentation of infectious mononucleosis and associated diffuse lymphoid hyperplasia in gastric mucosa. *Arch Pathol Lab Med.* 2003;127:478-80.

Funding: None; Conflict of Interest: None Stated.

How to cite this article: Manappallil RG, Praveen A, Chandramana AD. Infectious Mononucleosis presenting as abdominal pain. *Indian J Case Reports.* 2019;5(6):554-556.

Doi: 10.32677/IJCR.2019.v05.i06.016