

CASE REPORT

ACUTE HEPATITIS A INFECTION - A RARE PRESENTATION WITH PERIPHERAL BLOOD EOSINOPHILIA

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Abstract. Viral hepatitis infection is the most common cause of hepatobiliary tract infection, with hepatitis A virus (HAV) infection still being a health problem in developing countries. There are no available clinical data for HAV infected patients presenting with eosinophilia in the peripheral blood. From a review of medical records and laboratory investigations at Hospital for Tropical Diseases and Rajavithi Hospital, one male HAV-infected patient was recorded presenting a mild degree of eosinophilia. Other secondary causes of reactive eosinophilia including parasite infection, medication and allergic conditions were ruled out. There was no complication from the HAV infection and high eosinophilia. After an icteric phase, the patient's condition improved within a few weeks with only supportive treatment. This rare hematological condition might have been due to an inflammatory process of liver injury from the viral infection.

Keywords: hepatitis A, clinical outcome, eosinophilia

INTRODUCTION

Hepatitis is an inflammatory process of the hepatobiliary tract, resulting from infectious and noninfectious causes. The most common cause of acute hepatitis is viral infection, the majority from hepatitis A (HAV) and hepatitis E (HEV) viruses

(Clemente and Schwarz, 2011). HAV infection is often associated with consumption of contaminated food or water. There is a low seroprevalence (<50%) among Thais with HAV infection (Theamboonlers *et al*, 2002; Chatproedprai *et al*, 2007; Sa-Nguanmoo *et al*, 2016). Clinical characteristic of HAV infection is a gastrointestinal problem that develops to jaundice, which usually self-resolves within a few weeks.

To the best of our knowledge, there are no available data on HAV-infected patients with concomitant eosinophilia during the clinical course (Chowdry *et al*, 2012). This report describes a rare HAV infection and eosinophilia.

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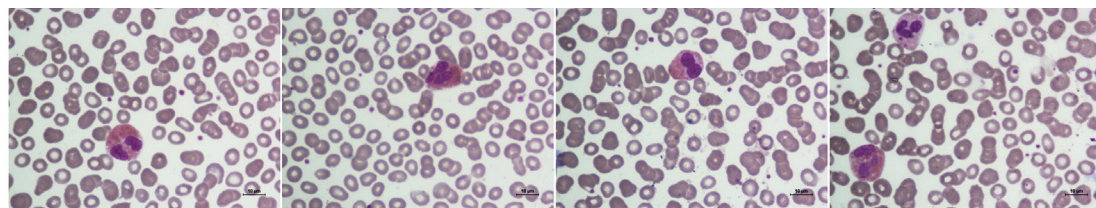
CASE REPORT

A 59-year-old Thai man was hospitalized for abrupt onset of jaundice, low-grade fever, myalgia and watery diarrhea. Jaundice was noticed a few days after defervescence. The patient is a carrier of alpha-thalassemia, with no history of blood transfusion. There was no history of drug abuse or alcohol intake. Physical examination revealed marked icteric sclera, mild tenderness at the right upper abdomen and hepatomegaly by palpation. A complete blood count revealed a hemoglobin level of 14.8g/dl, a hematocrit of 44.8%, a normal white blood cell count ($5,600 \text{ cells/mm}^3$), with 45% neutrophils, 38% lymphocytes, 12% eosinophils, 4% monocytes, 1% basophils, and a normal platelet count ($164 \times 10^9/l$). The peripheral blood smear showed an increase in mature eosinophils of normal morphology (an absolute eosinophil count of $672/\text{mm}^3$) (Fig 1); the cut-off value of absolute eosinophil count is $500/\text{mm}^3$ (Gotlib, 2015). The severity of eosinophilia was categorized as mild

(absolute eosinophil count of $500\text{-}1,500/\text{mm}^3$) (Curtis and Ogbogu, 2016; Gotlib, 2015). The total bilirubin was 4 mg/dl, direct bilirubin 3.7 mg/dl, serum aspartate aminotransferase 816 IU/l, alanine aminotransferase 1,430 IU/l, and coagulogram test (prothrombin time and activated partial thromboplastin time) in the normal limit. Tests for HBs antigen, anti-HCV and anti-HAV IgG antibodies were negative, but that for anti-HAV IgM antibody was positive (SD Bioline HAV IgG/IgM rapid test, Gyeonggi-do, Republic of Korea).

The routine stool examination was negative for parasites and protozoa. Automated blood cultures for the detection of microorganisms were negative including blood immunoblot tests for tissue parasites (angiostrongyliasis, filariasis, gnathostomiasis, paragonimiasis, toxocariasis, and trichinellosis). Presence of HAV in stool was demonstrated by RT-PCR (Namsai *et al*, 2011; Ngaosuwanikul *et al*, 2013). The patient was treated with intravenous fluid for hydration replacement during the anorexia period, and

A)



B)

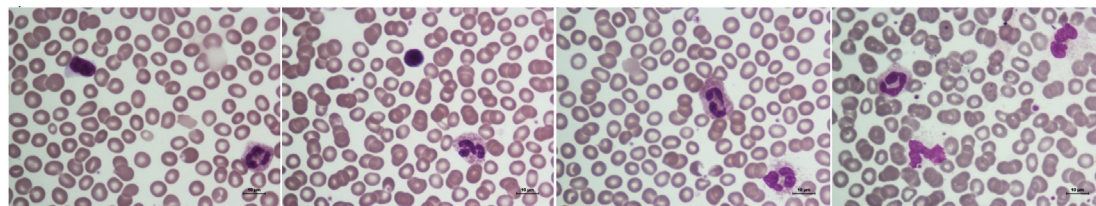


Fig 1 - A) Peripheral blood smears of the patient during hospitalization show mature eosinophilia 12-17 μm in diameter. B) The patient's peripheral blood smears performed at the outpatient clinic show normal morphology of white blood cells.

symptoms improved during the five-day hospitalization during which eosinophilia became normal. Three weeks later, when liver function tests returned to normal levels, the patient was normal (Table 1).

This case report was approved by the Ethics Committee of Rajavithi Hospital (approval No. 228/2559). The patient has provided his written consent for the publication of this report and any accompanying information.

DISCUSSION

HAV infection is a self-limiting disease following an inflammatory period of the liver, general clinical characteristics of viral hepatitis (Namsai *et al*, 2011; Ngaosuwankul *et al*, 2013; WHO, 2010). The majority of HAV cases present with leukopenia or thrombocytopenia (Tomida *et al*, 1996). However, there were a few reports of unusual presentations and

Table 1
Laboratory findings of the patient.

Parameter	Date of investigation (2016)					
	25/Jan	2/Feb	3/Feb	7/Feb	21/Mar	21/Apr
Complete blood count						
Hemoglobin (g/dl)	14.7	14.8	N/A	14.7	13.9	14.6
Hematocrit (%)	45.7	44.8		44.5	42.5	45.8
White blood cells count (WBC/ μ l)	6,400	5,600		5,700	5,500	6,500
Neutrophils (%)	55.0	45.0		51.0	45.0	55.0
Lymphocytes (%)	33.0	30.0		23.0	42.0	34.0
Atypical lymphocytes (%)	0	8.0		8.0	6.0	0
Eosinophils (%)	5.0	12.0		8.0	6.0	4.0
Monocytes (%)	7.0	4.0		10.0	0	7.0
Basophils (%)	0	1.0		0	1.0	0
Platelets count (cells/ μ l)	184,000	165,000		258,000	242,000	210,000
Fasting blood sugar (mg%)			99.0			
Liver function test						
Total bilirubin (mg/dl)		4.0	3.9	1.4	1.0	0.48
Direct bilirubin (mg/dl)		3.7	3.2	1.1	0.5	0.24
AST (U/l)		816	622	204	26	22
ALT (U/l)		1,430	1,236	714	53	22
Alkaline phosphatase (U/l)		534	465	512	177	112
Total protein (g/dl)		7.4	6.9	7.3	7.3	7.6
Albumin (g/dl)		4.3	4.1	4.2	4.6	4.9
	Checkup period	Hospitalization period		Follow up period		

complications from HAV infection, such as aplastic anemia and hemophagocytic syndrome (Tomida *et al*, 1996; Tuon *et al*, 2008; WHO, 2010). The current case presented as a prodromal period with non-specific symptom, which shortly turned to jaundice and a mild degree of eosinophilia. The cause of eosinophilia can be primary (clonal or idiopathic) or secondary (tissue-invasive parasitosis) (Ehrhardt and Burchard, 2008; Ndao, 2009; Gotlib, 2015). Neither tissue nor intestinal parasite infection was detected, and the patient had no history of allergy or medication usage. Thus, eosinophilia was attributed to HAV infection.

There are little available data on the relationship between eosinophilia and gastrointestinal tract disorder (Curtis and Ogbogu, 2016). Eosinophilia was described in patients with organ infection or eosinophil-mediated inflammation (Nutmans, 2007). This might be the situation in the patient (inflammation of hepatocytic cells). The clinical course observed in this report is similar to other studies of HAV infection (Chodick *et al*, 2006).

To the best of our knowledge, this is the first case of a HAV-infected patient presented with mild eosinophilia during treatment. We suggest eosinophilia might be related HAV-associated hepatic inflammation, but this will require further investigation.

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CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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