

Annals of
Tropical Medicine
and **Public Health**

www.atmph.org



Official Publication of
Africa Health Research Organization

Volume 6 | Issue 3 | May-Jun 2013

 Wolters Kluwer
Health

Medknow

Autoimmune hemolytic anemia secondary to chicken pox

Abraham M. Ittyachen, Mohan B. Jose, Varghese Abraham

Department of Medicine, M.O.S.C Medical College and Hospital, Kolenchery, Ernakulam District, Kerala, India

ABSTRACT

Autoimmune hemolytic anemia (AIHA) is a rare complication of chicken pox. It is described mainly in children. Even in children it is a rare complication and the long-term prognosis remains to be elucidated. Herein we report an adult, a 23-year-old male who developed AIHA secondary to chicken pox.

Key words: Autoimmune hemolytic anemia, chicken pox, viral infection

Introduction

Autoimmune hemolytic anemia (AIHA) is an immune disorder characterized by the presence of auto antibodies against red blood cells. AIHA is one of the earliest known immune disorders described in the early twentieth century. About half of the patients with AIHA do not have any underlying cause and hence termed primary or idiopathic. Secondary causes of AIHA include lymphoproliferative disorders, collagen-vascular diseases, infections, tumors, inflammatory bowel disease, and drugs.^[1]

Chicken pox, a viral infection caused by the varicella-zoster virus is endemic in the population at large. The disease is often self limiting excepting in the new born and immunocompromised. AIHA is a rare complication of chicken pox in children.^[2,3] Here we describe an adult, a 23-year-old male who developed AIHA secondary to chicken pox.

Case Report

A 23-year-old male with chicken pox infection was admitted with complaints of passing 'dark colored' urine. He had a history of prodromal symptoms followed by eruptions, typical of chicken pox. The

eruptions were 8 days old at the time of admission and were in various stages of healing. There was a recent history of chicken pox in the family. On examination the patient also had icterus. Rest of clinical examination was unremarkable. The initial investigations were suggestive of indirect hyperbilirubenemia:

Hb: 10.7 g%, TC: $8.4 \times 10^9/L$, DC: N66% L28% E1% M4% B1%, Platelets: $115 \times 10^9/L$, ESR: 53/1st hour; Urine microscopy: WBCs 20–25, RBCs nil, Bile salts negative; Liver function tests: Total Bilirubin: 12.6 mg% (N: 0.2–1.2), Direct Bilirubin: 0.1 mg% (N: 0.2–0.7), SGPT: 109 IU/L (N: 20–60), SGOT: 196 IU/L (N: 8–40), ALP: 59 IU/L (N: 36–150); Hbs Ag: negative; IgM anti HAV: negative; HCV: negative; HIV: negative.

The next day a drop in the hemoglobin level was noted (8.9 g%). Subsequently he was also evaluated for hemolysis. Peripheral smear was suggestive of hemolytic anemia and lactate dehydrogenase (LDH) was also found to be markedly elevated: 8544 IU/L (N: 313–618). Direct Coombs test was also found to be positive. A diagnosis of AIHA was made. Patient was started on steroids—prednisolone (1 mg/kg/d); the dose was subsequently tapered. He was also transfused four units of packed red cells. After starting steroids improvement in all the biochemical markers of hemolysis (LDH, indirect bilirubin) was noted. The patient was discharged on the seventh day; steroids were stopped. On review after one week the hemogram was normal and the bilirubin and LDH levels had also normalized. Patient was reassured and sent home. Incidentally tests for other systemic immune disorders were negative.

Access this article online	
Quick Response Code:	Website: www.atmph.org
	DOI: 10.4103/1755-6783.121007

Correspondence:

Abraham M. Ittyachen, Department of Medicine, M.O.S.C Medical College and Hospital, Kolenchery, Ernakulam District, Kerala, India. E-mail: abylyz@rediffmail.com

Discussion

AIHA is a rare complication of chickenpox described in children. It characteristically occurs after the onset of vesicular eruptions. To our knowledge a pure case of AIHA secondary to chickenpox has not been reported in adults. A case of Evan's syndrome (hemolytic anemia with thrombocytopenia) has been described from Japan.^[4]

Though there is a risk of flare up of the underlying disease, high dose steroids remains the mainstay of treatment of AIHA. The clinical course remains variable. Some of the cases (not necessarily following chickenpox) may become chronic with exacerbations and remissions in between. Our patient had a good response to steroids. Since even in children this complication is rare, the long-term prognosis of AIHA secondary to chickenpox remains to be

elucidated.

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Cite this article as: Ittyachen AM, Jose MB, Abraham V. Autoimmune hemolytic anemia secondary to chicken pox. *Ann Trop Med Public Health* 2013;6:353-4.

Source of Support: Nil, **Conflict of Interest:** None declared.