

CASE REPORT

# Henoch-Schönlein Purpura and Elevated Hepatitis C Virus Antibody in a Girl With Nasopharyngeal Diffuse Large B-Cell Lymphoma

Jen-Yin Hou<sup>a,b</sup>, Hsi-Che Liu<sup>a,c,d</sup>, Der-Cherng Liang<sup>a</sup>, Yin-Sum Choi<sup>a</sup>,  
Chia-Ying Lin<sup>a</sup>, Ting-Chi Yeh<sup>a,b,e,\*</sup>

<sup>a</sup> Division of Pediatric Hematology-Oncology, Mackay Memorial Hospital, Taipei, Taiwan

<sup>b</sup> Mackay Medicine, Nursing and Management College, New Taipei City, Taiwan

<sup>c</sup> Mackay Medical College, New Taipei City, Taiwan

<sup>d</sup> School of Medicine, Taipei Medical University, Taipei, Taiwan

<sup>e</sup> Department of Applied Cosmetology, Lee-Ming Institute of Technology, New Taipei City, Taiwan

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## Key Words

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Henoch-Schönlein purpura (HSP) or hepatitis C virus (HCV) infection was reported in association with malignancies. However, HSP and HCV infection rarely present in pediatric patients with non-Hodgkin's lymphoma. We describe an 8-year-old girl with Stage-IV diffuse large B-cell lymphoma who presented with HSP and elevated HCV antibody titer at diagnosis and at relapse. After treatment, purpura disappeared and HCV antibody titer returned to normal range. There was no recurrence of HSP or elevated HCV antibody during a follow-up of 2 years. Copyright © 2011, Taiwan Pediatric Association. Published by Elsevier Taiwan LLC. All rights reserved.

## 1. Introduction

Henoch-Schönlein purpura (HSP) is a systemic vasculitis affecting small vessels with immunoglobulin A (IgA)-dominant immune deposits; characteristic involvement with skin, gastrointestinal tract and glomeruli; and association with arthritis or arthralgia.<sup>1</sup> HSP is more frequent in children than adults. Vasculitis has been described in association with neoplasm.<sup>2</sup> However, HSP associated with malignancy

\* Corresponding author. Division of Pediatric Hematology-Oncology, Mackay Memorial Hospital, No. 92, Section 2, Chung-San North Road, Taipei 10449, Taiwan.

E-mail address: [yeh@ms1.mmh.org.tw](mailto:yeh@ms1.mmh.org.tw) (T.-C. Yeh).

has rarely been reported and is especially uncommon in children.<sup>3</sup> The role of hepatitis C virus (HCV) infection in non-Hodgkin's lymphoma (NHL) is still controversial.<sup>4-6</sup> Herein, we report an 8-year-old girl with Stage-IV diffuse large B-cell lymphoma (DLBCL) who presented with HSP and elevated HCV antibody titer at diagnosis and at relapse. She has been in continuous second remission for 24 months.

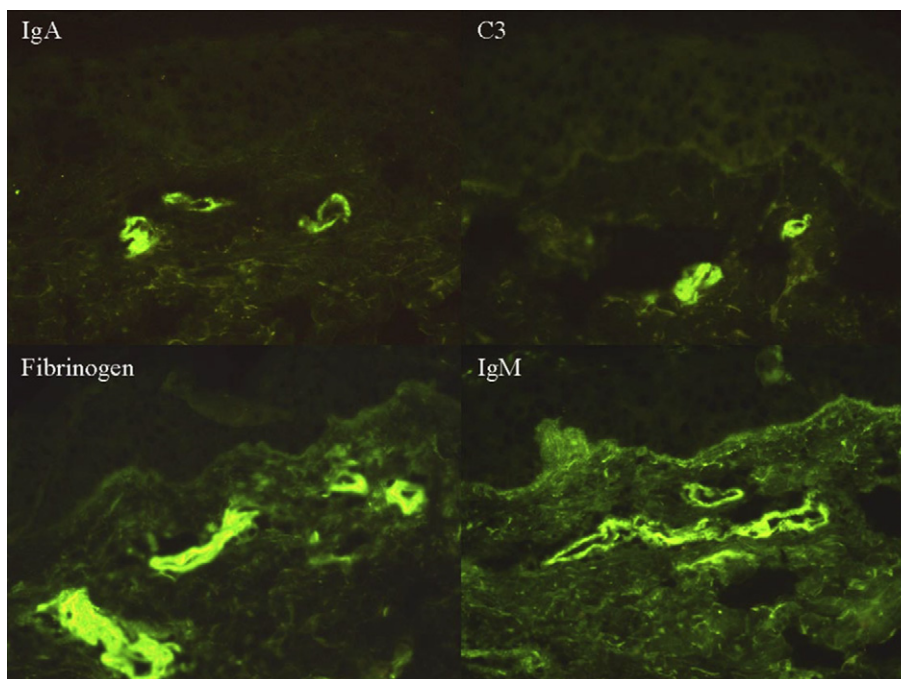
## 2. Case Report

An 8-year-old naturally healthy girl was referred to our pediatric department for intermittent epistaxis for 1 month. Also, she had had multiple palpable purpura on both lower legs off and on for 6 months. Fever, cough, and rhinorrhea had developed for the preceding 2 days. Physical examination showed hepatosplenomegaly and nasal mass with active bleeding. Complete blood counts revealed hemoglobin 109 g/L, mean corpuscular volume 82.4 fL, white blood cell count  $8.1 \times 10^9$ /L, normal differential counts, and platelets  $187 \times 10^9$ /L. Prothrombin time, activated partial thromboplastin time, and bleeding time were within normal range. The urine analysis was normal. Brain computed tomography scan showed a tumor mass (2.5 cm  $\times$  4 cm  $\times$  3.5 cm) in the nasopharynx. Nasopharyngeal biopsy confirmed DLBCL, which showed a panel of immunoreactivity, positive for CD79a and CD20 and negative for CD56, TdT, or CD45RO. Bone marrow biopsy revealed tumor cell involvement. Cerebrospinal fluid analysis and cytospin were normal. Tc99m whole-body bone scan showed increased uptake in the skull base, probably reflecting local tumor invasion. Serology studies showed serum lactate dehydrogenase: 397 U/L, aspartate transaminase: 43 U/L, alanine transaminase:

9 U/L, blood urea nitrogen: 6 mg/dL, creatinine: 0.3 mg/dL, and IgA: 355 mg/dL (normal range: 45–236 mg/dL). HCV antibody was 7.77 (reference range was less than 1.00), whereas HCV-RNA titer was negative (less than 10 IU/mL).

This patient was considered Stage-IV DLBCL according to the St. Jude Children's Research Hospital Staging System.<sup>7</sup> She was treated with Taiwan Pediatric Oncology Group 98 B-NHL protocol, which is based on the BFM-90 protocol,<sup>8</sup> with minor modification. Risk group was classified as BFM-R3. Purpura disappeared soon after treatment was initiated. After cytoreductive prephase, two more courses of chemotherapy (AA, BB) were used. Bone marrow biopsy revealed remission, whereas nasopharyngeal biopsy still displayed residual tumor. Because of unsatisfactory response, CC course was followed. Then, nasopharyngeal biopsy disclosed no residual tumor. Consecutive treatment with AA, BB, and CC courses proceeded. Post-treatment evaluation revealed free of lymphoma.

However, 4 months after completion of treatment, fever, and purpura on both of the patient's lower legs recurred. Serum IgA was 231 mg/dL. C3, C4, and antinuclear factor titer were within normal limits. HCV antibody titer was 1.45 and HCV-RNA titer was negative. Lactate dehydrogenase titer was 170 U/L. Skin biopsy of purpura on the right dorsal foot revealed leukocytoclastic vasculitis and tissue immunofluorescence demonstrated IgA, IgM, C3, and fibrinogen depositions on blood vessels of the superficial dermis while not for IgG (Figure 1). Brain computed tomography scan showed thickening of the bilateral posterosuperior wall of nasopharynx. Nasopharyngeal biopsy revealed relapse of DLBCL, whereas bone marrow biopsy showed still in remission. Marrow donor search failed to find an HLA-match donor. R-CHOP regimen<sup>9</sup> (rituximab



**Figure 1** Immunofluorescent stain of skin biopsy revealed IgA, IgM, C3, and fibrinogen depositions on blood vessels of the superficial dermis. IgG was negative (not shown). The findings were consistent with vasculitis, including Henoch-Schönlein purpura. IgA = immunoglobulin A; IgM = immunoglobulin M.

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