A Surprising Cause of Chest Pain
Rudolf Benz*, Kurt Seiler+, Markus Vogt++

Abstract
We present the case of a 19 years female who was referred to our institution because of severe left-sided chest pain. While the clinical picture suggested acute mononucleosis, low O2 saturation, slightly altered ECG, and pain in the left hemithorax could not be explained by an acute EBV infection. Using a CT scan, we ruled out a pulmonary embolism. However, we found two fresh splenic infarcts. Diseases of the (left) upper abdomen can cause symptoms in the left hemithorax, which explains the atypical presentation. In the rare instance of splenic infarction in acute mononucleosis, we recommend supportive treatment. However, careful observation is needed because some data suggest a potential progression to splenic rupture. Additionally, it is debatable whether NSAID – the standard treatment of acute EBV – should be avoided in this situation because it inhibits thrombocytic functions.

INTRODUCTION
Epstein-Barr virus infection primo infections are predominantly seen during childhood and are often clinically asymptomatic. In contrast, if the infection occurs during adolescence or adulthood, acute mononucleosis presents often with the classical triad of hepatosplenomegaly, pharyngitis and lymphadenopathy. Additional symptoms are rare, but of interest for general physicians. Here, we describe the case of a young woman with acute mononucleosis presenting with thoracic pain.

CASE
A 19 years woman with an ordinary medical history was referred to our emergency department because of recent severe left-sided pleuritic chest pain, which was not accompanied by a cough or hemoptysis. Four days prior to admission, her treating physician had noted fever, tonsillitis, pharyngitis, and generalized lymphadenopathy with no hepatosplenomegaly. The laboratory workup was noteworthy, with a white blood cell-count of 3,1x10^9/L (neutrophils, 50%; lymphocytes, 31%) and a negative group A streptococcal carbohydrate antigen test on the throat swab. The clinical picture strongly suggested infectious mononucleosis and symptomatic analgesic treatment with Acetaminophen was instituted.

On admission, the young woman appeared ill and complained of severe pain in the entire left hemithorax with limiting respiratory excursions. Auscultation of heart and lungs was normal, with a pulse rate of 86 beats/minute and blood pressure of 130/64 mmHg. Temperature was 38.9°C axillary. The liver edge was tender on palpation and splenomegaly was now noticed. There were no signs of right heart failure. Peripheral edema and calf tenderness were absent. She had no family history of thrombophilia.

The electrocardiogram showed a slight S in I and a Q in III. Oxygen saturation on pulse oxymetry was 94% on room air. Routine laboratory examination showed a slightly increased white cell-count of 11,4x10^9/L (75,5% large polymorphic lymphocytes), a haemoglobin of 134 g/l, platelets of 135000/µL, a LDH of 899U/L (normal range 210-425 U/L), an AST 113U/L (11-36 U/L), an ALT 116 U/L (10-37 U/L), a CRP of 58mg/L (<5 mg/L), and a negative Monotestâ as well as D-Dimers of 900 ng/mL (68-500 ng/mL).

Chest pain, ECG findings, and low oxygen saturation could not be explained by the suspected mononucleosis. In order to rule out a pulmonary embolism or a pleuritic inflammatory process, a contrast-enhanced helical-computed tomogram of the chest was performed. As a result, an acute pulmonary embolism could be ruled out but two recent splenic infarcts were identified in an otherwise only slightly enlarged spleen that measured 11 cm (Fig. 1).

Although unexpected, this finding easily explained the documented symptoms. By day six of her illness, a positive IgM-response consistent with acute EBV infection was noted (Table 1).

The patient’s condition improved gradually following symptomatic treatment with Acetaminophen. She was discharged without residual left-sided pain and she was advised to avoid contact-sport and heavy lifting for three months. The typical antibody sequence after an acute EBV infection could be found in the follow up controls.

*Senior Resident, Division of Hematology, University Hospital of Zurich, Switzerland. +Senior Resident; ++Head of Department of Internal Medicine; Medical Clinic, Zuger Kantonsspital, Switzerland.
Received : 14.7.2006; Accepted : 1.8.2007
Background Section

Splenic infarction is a rare clinical picture causing a variety of symptoms, ranging from asymptomatic to strong abdominal pain.\(^1,2\) Similar to other pathologies of the (left) upper abdomen, pain can radiate to the left hemithorax, as was the case with our patient. This clinical sign is named Kehr’s Sign. While there are several causes of splenic infarction, two main groups can be identified: embolic and hematologic.\(^1\) Besides septic emboli in endocarditis, embolic aetiology is mainly seen in the elderly population with irregular heart rhythm and arteriosclerosis. Hematologic causes of splenic infarction are hemoglobinopathies (most often sickle cell disease) and lymphoproliferative as well as myeloproliferative disease occurring in young and old patients. Splenic infarction in acute EBV – as was the case with our patient – has only been reported in medical literature three times.\(^3-5\) Nothing specific can be said about the spontaneous progression of this complication. Splenic infarction in non-EBV cases results in splenic rupture in a small proportion of cases.\(^1\) Nevertheless, a splenectomy is not initially recommended, but careful observation is mandatory. Regarding this potentially lethal complication, we recommend avoiding non-steroidal anti-inflammatory drugs (NSAID) in patients suffering acute mononucleosis with palpable spleen or symptoms indicating a possible infarction or rupture. This recommendation is based on knowledge of the effect NSAID have on platelets and personal experience (fatal in-hospital splenic rupture in a patient with acute EBV infection on NSAID).

10% of adolescents with an acute EBV infection have negative heterophile antibodies, as seen in our patient.\(^6\) If clinically suspected, a positive EBV-VCA IgM confirms an acute infection. For additional information, we recommend the publication of Tsaparas et al.,\(^7\) which includes a useful algorithm for patients with negative monospot and a clinic picture suggestive for mononucleosis.

The reduced oxygen saturation in our patient was likely due to compromised respiratory excursions.

### REFERENCES


### Table 1: Evolution of EBV-Serology

<table>
<thead>
<tr>
<th></th>
<th>day 1</th>
<th>day 6</th>
<th>7 month</th>
<th>9 month</th>
<th>normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EBV-VCA-IgG IF</strong></td>
<td>1:80</td>
<td>1:320*</td>
<td>1:1280</td>
<td>1:640</td>
<td>&lt; 1:20</td>
</tr>
<tr>
<td><strong>EBNA-1 ACIF</strong></td>
<td>&lt; 1:10</td>
<td>&lt; 1:10*</td>
<td>&lt; 1:10</td>
<td>&lt; 1:10</td>
<td>&lt; 1:10</td>
</tr>
<tr>
<td><strong>EBNA-2 ACIF</strong></td>
<td>&lt; 1:10</td>
<td>&lt; 1:10*</td>
<td>1:40</td>
<td></td>
<td>&lt; 1:10</td>
</tr>
<tr>
<td><strong>EBV-VCA-IgM IF</strong></td>
<td>neg.</td>
<td>pos.*</td>
<td>neg.</td>
<td></td>
<td>neg.</td>
</tr>
<tr>
<td><strong>Heterophile Anti-neg.</strong></td>
<td>neg.*</td>
<td>neg.</td>
<td>neg.</td>
<td></td>
<td>neg.</td>
</tr>
</tbody>
</table>

* tested together with probe from day 1.

Fig. 1: Contrast-enhanced helical-computed tomography demonstrating two recent splenic infarcts.