Fusobacterium nucleatum septicemia and portal vein thrombosis [brief report]
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Table 1. Summary of results of liver function tests for a patient with acute hepatitis A who had received preexposure inactivated hepatitis A vaccine.

<table>
<thead>
<tr>
<th>Date of Test</th>
<th>Toal Protein (g/L)</th>
<th>Albumin (g/L)</th>
<th>Total Bilirubin (μmol/L)</th>
<th>SAP (U/L)</th>
<th>ALT (U/L)</th>
<th>AST (U/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>13/7/98</td>
<td>64</td>
<td>33</td>
<td>50</td>
<td>356</td>
<td>2,273</td>
<td>3,247</td>
</tr>
<tr>
<td>14/7/98</td>
<td>63</td>
<td>32</td>
<td>57</td>
<td>284</td>
<td>2,037</td>
<td>1,762</td>
</tr>
<tr>
<td>16/7/98</td>
<td>65</td>
<td>31</td>
<td>81</td>
<td>280</td>
<td>2,097</td>
<td>1,541</td>
</tr>
<tr>
<td>20/7/98</td>
<td>74</td>
<td>34</td>
<td>86</td>
<td>489</td>
<td>427</td>
<td>186</td>
</tr>
<tr>
<td>22/7/98</td>
<td>78</td>
<td>35</td>
<td>96</td>
<td>470</td>
<td>271</td>
<td>114</td>
</tr>
<tr>
<td>11/8/98</td>
<td>76</td>
<td>43</td>
<td>14</td>
<td>114</td>
<td>35</td>
<td>35</td>
</tr>
</tbody>
</table>

NOTE. ALT = alanine aminotransferase; AST = aspartate aminotransferase; SAP = serum alkaline phosphatase.

A seroconversion failure rate of 0.1% has been found, and these failures occurred for smokers, alcoholics, immunocompromised persons, and patients with concurrent illness with hepatitis C or B (D. R. Nalin, unpublished data). Our patient did not have any of these risk factors associated with a low rate of seroconversion. One considers infection is the use of inhaled steroids by our patient, which may have been associated with the lack of seroconversion.

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3. McMahon BJ, Beller M, Williams J, Schloss M, Tanilla H, Bulkow L. An incidence of 0.1% has been found, and these failures occurred for smokers, alcoholics, immunocompromised persons, and patients with concurrent illness with hepatitis C or B (D. R. Nalin, unpublished data). Our patient did not have any of these risk factors associated with a low rate of seroconversion. One considers infection is the use of inhaled steroids by our patient, which may have been associated with the lack of seroconversion.

Fusobacterium nucleatum Septicemia and Portal Vein Thrombosis

Like Fusobacterium necrophorum, Fusobacterium nucleatum is capable of causing hrombophlebitis of the internal jugular vein in previously healthy young adults, usually following pharyngosinusillar infection [1, 2]. Allochomplex infection of venous hrombosis a various loci has been described in cases of Fusobacterium nucleatum septicemia, poral vein hrombosis has never been reported. We describe a patient with a hepatitis A vaccine.

Fusobacterium nucleatum (F. nucleatum) is an obligate anaerobe, capable of causing hrombophlebitis of the internal jugular vein, usually following pharyngosinusillar infection [1, 2]. Allochomplex infection of venous hrombosis a various loci has been described in cases of Fusobacterium nucleatum septicemia, poral vein hrombosis has never been reported. We describe a patient with a hepatitis A vaccine.

A 23-year-old man was hospitalized in February 1995 because of a 14-day history of abdominal pain, vomiting, rigors, and fever (empyema urethral). Five weeks before his onset of symptoms, he was treated with diclofenac for oropharyngeal infection. Physical examination was unremarkable. Laboratory tests showed an increased WBC count of 16.4 x 10^9/L, with 80% neutrophils and a lefshift, and oxiec changes. Liver function tests revealed mild elevations in levels of transaminases (aspartate and alanine aminotransferase, 61 U/L; alanine amino transferase, 113 U/L), alkaline phosphatase (192 U/L), and y-glutamyl transferase (144 U/L), and a normal bilirubin level. Ultrasound examination revealed infection of the abdomen. The patient was treated with intravenous antibiotics and heparin.

After 5 days of imipenem treatment (2 g/d), his symptoms abated, and he was discharged. No pathogens were isolated from cul ure of blood, urine, and stool. Ten days later, he was readmitted to a hospital with abdominal pain, jaundice, and respiratory distress. Ultrasonographic examination revealed a large, solitary, echogenic mass in the liver, which was enlarged, and consistent with the diagnosis of hepatitis A. The patient was discharged.

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administered. The patient's condition worsened, and he was transferred to the Academic Medical Center of the University of Amsterdam. At the time of physical examination, the patient was ill-appearing; findings included normal vital signs, hepatic and splenomegaly, and empyema pleural and pleural friction rubs. Repeated CT of the abdomen (figure 1) showed portal vein thrombosis and an increasing spleen size (maximum span, 25 cm). Upper gastrointestinal endoscopy demonstrated esophageal varices. Laboratory investigations showed no autoimmune or systemic disease or hypercoagulable state. The patient's condition worsened, and he was transferred to the Academic Medical Center of the University of Amsterdam.

A 6-week course of therapy with intravenous penicillin (12 million U/d) resulted in recovery, although portal vein thrombosis persisted.

Our patient had a clinical syndrome of fever, portal vein thrombosis, and ransient pleuropneumocardiitis. Blood cultures finally became positive for *F. nucleatum* on the 10th day (7–10 days) and prolonged subculture (3 days). We considered the hypothesis that the patient had *F. nucleatum* from an unknown source. Second, oropharyngeal infections prior to the onset of symptoms may be followed by *F. nucleatum* sepsis resulting in thrombophlebitis and thrombosis of the portal vein.

One argument in favor of the second hypothesis concerns sepsis due to *F. nucleatum*. Complications of venous thrombosis at various locations have been described in cases of *fusobacterium* sepsis [2, 5]. The ability of virulent *Fusobacterium* species to cause thrombophlebitis and mesenteric abscesses can probably be ascribed to the lipid A component of the lipopolysaccharide endotoxin of *Fusobacterium* species. This virulence factor has been shown to be capable of inducing an in vivo and in vitro coagulation response [6]. Aggregation of platelets by *F. necrophorum* has been demonstrated in vivo and is also a virulence property [7, 8].

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References