Fusobacterium nucleatum septicemia and portal vein thrombosis [brief report]
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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alcoholics, immunocompromised persons, and patients with concurrent illness with hepatitis C or B (D. R. Nalin, unpublished data).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 low rates of seroconversion. One of our patients is the use of inhaled steroids by our patient, which may have been associated with the lack of seroconversion.

### 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 tests</th>
<th>13/7/98</th>
<th>14/7/98</th>
<th>16/7/98</th>
<th>20/7/98</th>
<th>22/7/98</th>
<th>11/8/98</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total protein level (g/L)</td>
<td>64</td>
<td>63</td>
<td>65</td>
<td>74</td>
<td>78</td>
<td>76</td>
</tr>
<tr>
<td>Albumin level (g/L)</td>
<td>33</td>
<td>32</td>
<td>31</td>
<td>34</td>
<td>35</td>
<td>43</td>
</tr>
<tr>
<td>Total bilirubin level (µmol/L)</td>
<td>50</td>
<td>57</td>
<td>81</td>
<td>86</td>
<td>96</td>
<td>14</td>
</tr>
<tr>
<td>SAP level (U/L)</td>
<td>356</td>
<td>284</td>
<td>280</td>
<td>489</td>
<td>470</td>
<td>114</td>
</tr>
<tr>
<td>ALT level (U/L)</td>
<td>2,273</td>
<td>2,037</td>
<td>2,097</td>
<td>427</td>
<td>271</td>
<td>54</td>
</tr>
<tr>
<td>AST level (U/L)</td>
<td>3,247</td>
<td>1,762</td>
<td>1,541</td>
<td>186</td>
<td>114</td>
<td>35</td>
</tr>
</tbody>
</table>

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

### References

### Fuso acterium 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 pharyngosascular infection [1, 2]. Allopha h composes of venous thrombosis a variety of loca tions have been described in cases of F. nucleatum sepsis, por al vein thrombosis has never been reported. We describe a patient with F. nucleatum sepsis and portal vein thrombosis.

A 23-year-old man was hospitalized in February 1995 because of a 14-day history of abdominal pain, vomiting, rigors, and fever (temperature of 40°C). Five weeks before he onset of symptoms, he was readmitted with a fever of 38°C. On physical examination, he had a hepatomegaly. The laboratory results showed an increased WBC count of 16.4 x 10^9/L, with 80% neutrophils and a left shift, and a normal C-reactive protein. Liver function tests revealed mild elevations in levels of transaminases (aspartate aminotransferase, 61 U/L; alanine aminotransferase, 113 U/L), alkaline phosphatase (192 U/L), and y-glutamyl transpeptidase (144 U/L), and a normal bilirubin level. Ultrasound examination of the abdomen demonstrated a 10-cm mass in the liver, which was enlarged, and an extensive collateral venous system in the hepatic hilum. Hepinem and heparin were
One argument in favor of the second hypothesis is the changing image of the portal vein at repeated ultrasound examinations. First, an echogenic thrombus within a dilated portal vein and the lack of variation in the diameter of the portal vein with respiration were demonstrated, findings highly indicative of acute portal vein occlusion [3]; 2 months later, the diameter of the portal vein was very small, as is the case when long-standing thrombus [4]. Another argument for the second hypo thesis concerns sepsis pecia icemia due to \textit{F. nucleatum}. Complications of venous thrombosis at various locations have been described in cases of fusobacterium sepsis pecia icemia \cite{2, 5}. The ability of virulent \textit{Fusobacterium} species to cause thrombophlebitis and septic abscesses can probably be ascribed to the lipid A component of the lipopolysaccharide endogenous of \textit{Fusobacterium} species. This virulence factor has been shown to be capable of in vivo acyclic thrombosis and sepsis, and also a virulence property \cite{7, 8}.

Our patient had a clinical syndrome of fever, por al vein thrombosis, and ransfererd 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 splenomegaly, and empyema pericardial and pleural frictions. Repeated CT of the abdomen (figure 1) showed portal vein thrombosis and an increasing spleen size (maximum span, 25 cm). Upper gastrointestinal endoscopy demonstrated varices. Laboratory investigations showed no autoimmune or systemic disease or hypocoagulable state. The patient's condition worsened, and he was transferred to the Academic Medical Center of the University of Amsterdam.

References