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alcoholics, immunocompromised persons, and patients with concurrent illness with hepatitis C or B (D. R. Nalin, unpublished data).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 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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References

Fusococcum necrophorum Septicemia and Portal Vein Thrombosis

Like Fusobacterium necrophorum, Fusobacterium necrophorum is capable of causing hrombophlebitis of the internal jugular vein in previously healthy young adults, usually following pharyngo-tonsillar infection [1, 2]. All four complications of venous thrombosis associated with loca infections have been described in cases of F. necrophorum sepsis, including portal vein thrombosis which has never been reported. We describe a patient with F. necrophorum sepsis and portal vein thrombosis.

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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 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.


A 23-year-old man was hospitalized in February 1995 because of a 14-day history of abdominal pain, vomiting, rigors, and fever (empera ure 40°C). Five weeks before he onset of symptoms, he was re-admitted with heme icilin for oropharyngeal infection. Physical examination was remarkable. Laboratory tests showed an increased WBC count of 16.4 × 10^9/L, with 80% neutrophils and a left shift, and toxic changes. Liver function tests revealed mild elevations in levels of transaminases (aspartate and amino transaminase, 61 U/L; alanine aminotransferase, 113 U/L), alkaline phosphatase (192 U/L), and γ-glutamyl transferase (144 U/L), and a normal bilirubin level. Ultrasound examination of the abdomen demonstrated a right liver abscess and hepatic cirrhosis. After 5 days of imipenem therapy (2 g/d), his symptoms abated, and he was discharged. No pathogens were isolated from cultures of blood, urine, and stool. Fifteen days later, he was re-admitted with fever, abdominal pain, jaundice, and respiratory distress. Ultrasound examination of the kidneys demonstrated a kidney abscess and hepatic cirrhosis. Septicemia and portal vein thrombosis occurred. Ultrasound examination revealed hepatomegaly. After 5 days of imipenem therapy (2 g/d), his symptoms improved, and he was discharged. No pathogens were isolated from cultures of blood, urine, and stool. Fifty days later, he was readmitted with fever, abdominal pain, jaundice, and respiratory distress. Ultrasound examination of the abdomen demonstrated a kidney abscess and hepatic cirrhosis. Septicemia and portal vein thrombosis occurred. Ultrasound examination revealed hepatomegaly.
tum from an unknown source. Second, oropharyngeal infecion ion 5 weeks before he onse of sympms may be followed by F. nucleatum sep icemia resul ing in hrombophlebi is and hrombosis of he por al vein.

One argumen in favor of he second hypo hesis is he changing image of he por al vein a repea ed ul rasonographic examina ions. Firs , an echogenic hrombus wi hin a dila ed por al vein and he lack of varia ion in he diamne er of he por al vein wi h respira ion were demons ra ed, findings highly indica ive of ace e por al vein occlusion [3]; 2 mon hs la er, he diamne er of he por al vein was very small, as is he case wi h long-s anding hrombus [4]. Ano her argumen for he second hypo hesis concerns sep icemia due o F. nucleatum. Complica ions of venous hrombosis a various loca ions have been described in cases of fusobac erium sep icemia [2, 5]. The abili y of virulen Fusobacterium s rains o cause hrom- bophlebi is and me as a ic abscesses can probably be ascribed o

Figure 1. CT scan (wi h in ravenous con ras ma erial) of he abdo- men of a pa ien wi h Fusobacterium nucleatum sep icemia and por al vein hrombosis; he scan shows spleномegaly and colla er vessels (arrow) in he hili of he liver and spleen. The por al vein is no filled wi h con ras ma erial because of hrombosis.

Our pa ien had a clinical syndrome of fever, por al vein hrombosis, and ransien pleuropericardi is. Blood cul ures finally be- came posi ve for F. nucleatum af er prolonged incuba ion (7–10 days) and prolonged subcul uring (3 days). We considered wo hypo heses for he pa hogenesis of his clinical syndrome. Firs , preexis en por al vein hrombosis may be infec ed wi h F. nuclea-