Recurrent Symptomatic Pleural Effusion
Due to a Ventriculopleural Shunt

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Ventriculopleural shunts are uncommonly used for the treatment of normal pressure hydrocephalus in adults. Pleural effusion has been reported to complicate the course of these ventriculopleural shunts in children. The pleural effusion should typically resemble the cerebrospinal fluid unless frankly infected. There are few good data on the nature of the pleural effusion. We report a case of recurrent right-sided pleural effusion, 2 years after a ventriculopleural shunt insertion, for normal pressure hydrocephalus with no evidence of an underlying infection. The effusion abated after ligation of the shunt. We discuss the possible mechanisms in the development of the effusion. It is important to be aware of this unlikely complication of an uncommon procedure. Recognizing the origin of the pleural effusion can help in instituting close follow-up and early referral for revision of the ventriculopleural shunt. Key words: ventriculopleural shunt, pleural effusion, exudates, recurrence. [Respir Care 2009;54(8):1112–1114. © 2009 Daedalus Enterprises]
Fusion was noted on the chest radiograph. A computed tomogram scan of the chest confirmed a large right pleural effusion with right-lower and middle-lobe atelectasis, and a ventriculopleural shunt in the lower-right hemithorax (Fig. 1). There was no evidence of pleural thickening, loculations, or masses.

A diagnostic and therapeutic thoracentesis was undertaken, draining 1,500 mL of clear fluid with a pH of 7.44, a total protein of 1.5 g/dL (normal range 0.6–4.9 g/dL), lactate dehydrogenase of 109 units/L, and a white-blood-cell count of 166 cells/µL, with 91% lymphocytes. Serum total protein and lactate dehydrogenase were 5.1 g/dL (normal range 6–8 g/dL) and 112 IU/L (normal range 98–192 IU/L), respectively. The pleural fluid was positive for β2 transferrin, indicating the presence of cerebrospinal fluid. A simultaneous lumbar puncture was undertaken to rule out an infected shunt system. It revealed normal cerebrospinal fluid characteristics. The pleural and cerebrospinal fluid cultures were negative.

Stool studies confirmed *Clostridium difficile* colitis that was appropriately treated, with resolution of diarrhea and fever. A shunt series indicated that the tip of the catheter

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**Table 1. Pleural Fluid and Serum Values on Each Admission.**

<table>
<thead>
<tr>
<th>Admission</th>
<th>Pleural Fluid</th>
<th>Serum</th>
<th>Pleural Fluid</th>
<th>Serum</th>
<th>Pleural Fluid</th>
<th>Serum</th>
<th>Pleural Fluid</th>
<th>Serum</th>
<th>Pleural Fluid</th>
<th>Serum</th>
<th>CSF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amount drained (L)</td>
<td>2</td>
<td>NA</td>
<td>1.4</td>
<td>NA</td>
<td>1.3</td>
<td>NA</td>
<td>1.5</td>
<td>NA</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.45</td>
<td>ND</td>
<td>7.39</td>
<td>ND</td>
<td>8.0</td>
<td>ND</td>
<td>7.44</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total protein (g/dL)</td>
<td>0.6 (normal 0.6–4.9)</td>
<td>6.6 (normal 6–8)</td>
<td>2.1</td>
<td>5.5</td>
<td>1.5</td>
<td>4.9</td>
<td>1.5</td>
<td>5.1</td>
<td>230 mg/dL (normal 20–45)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactate dehydrogenase (units/L)</td>
<td>90</td>
<td>97</td>
<td>110</td>
<td>142</td>
<td>97</td>
<td>103</td>
<td>109</td>
<td>112</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Albumin (g/dL)</td>
<td>&lt;1.0</td>
<td>2.3 (normal 3.1–4.7)</td>
<td>&lt;1.0</td>
<td>1.7</td>
<td>&lt;1.0</td>
<td>1.9</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol (mg/dL)</td>
<td>29</td>
<td>ND</td>
<td>26</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White blood cells (cells/µL)</td>
<td>86</td>
<td>ND</td>
<td>13</td>
<td>ND</td>
<td>231</td>
<td>ND</td>
<td>127</td>
<td>ND</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lymphocytes (%)</td>
<td>60</td>
<td>ND</td>
<td>86</td>
<td>ND</td>
<td>86</td>
<td>ND</td>
<td>91</td>
<td>ND</td>
<td>11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Red blood cells (cells/µL)</td>
<td>30</td>
<td>ND</td>
<td>49</td>
<td>ND</td>
<td>43</td>
<td>ND</td>
<td>53</td>
<td>ND</td>
<td>5,000</td>
<td></td>
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</tbody>
</table>

CSF = cerebrospinal fluid
NA = not applicable
ND = no data collected

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**Fig. 1.** Computed tomogram showing predominant right pleural effusion with the ventriculopleural shunt catheter in the right hemithorax (arrows).

**Fig. 2.** Ventriculopleural shunt series revealing the tip of the catheter in the right pleural space (arrow), with moderate right pleural effusion.
was in the right pleural space (Fig. 2). The distal shunt catheter was ligated, as the patient declined a revision surgery. No symptomatic re-accumulation of the pleural effusion was noted after 6 months of follow-up.

**Discussion**

Ventriculo-pleural shunts were introduced as a management option for hydrocephalus by Ransohoff in 1954.1 The next few decades witnessed reports of pleural effusions complicating the course of ventriculo-pleural shunts in infants.2,3 This led to the relegation of ventriculo-pleural shunting as a “backup” measure. Some reports suggested that these shunts should preferably be avoided in younger children.3 Incorporation of anti-siphon devices and valves into the shunt system has been recommended in an attempt to reduce the incidence of pleural effusions.4

Ventriculo-pleural shunts are rarely utilized in middle-age adults.5 Despite the good absorptive capacity of the pleura in adults, some patients can develop pleural effusions.5 The pleural fluid typically resembles the cerebrospinal fluid, with low protein and lactate dehydrogenase values.6

In our patient, the diversion of the cerebrospinal fluid into the pleural space was utilized as a first-line measure, due to his abdominal size. Recurrent pleural effusions developed around 2 years after the shunt insertion. The pleural fluid consistently demonstrated high lactate dehydrogenase levels and lymphocytosis. No further re-accumulation of the effusions was noted after ligation of the shunt. His fever on the current admission was due to *Clostridium difficile* infection, which responded to appropriate antibiotic treatment.

Pleural effusions develop due to the alterations in dynamics of net pleural fluid production and absorption. The mechanisms for accumulation of pleural effusions with ventriculo-pleural shunts remain speculative. The presence of a shunt catheter in the pleural space may produce local irritant effects, inducing a chronic sub-clinical inflammatory response, as supported by the predominant lymphocytosis in the pleural fluid. Inflammation leads to increased pleural fluid production and impaired lymphatic flow, causing pleural fluid accumulation and lung collapse. This further reduces the pleural surface area, resulting in a decrease in the net absorption of the pleural fluid.7 Continuous addition of cerebrospinal fluid compounds the problem, leading to rapid accumulation of large pleural effusions.7

The elevated pleural fluid lactate dehydrogenase could be due to chronic shunt catheter inflammation, and partly as result of repeated thoracentesis causing cell injury.8 The possibility of a local immune response cannot be excluded.5 In adults there is probably a gradual diminution in the pleural resorptive capacity over time; hence, symptoms may not manifest for several months.

The presence of β_2_ transferrin in a body fluid has 94% sensitivity and almost 100% specificity for the presence of cerebrospinal fluid.9 Hence, it should be considered in all patients with a ventricular shunt and a pleural effusion.

Some degree of pleural effusion is noted in most patients with ventriculo-pleural shunts. Asymptomatic or minimally symptomatic effusions mandate close follow-up, with elective referral to the neurosurgeon. Therapeutic thoracentesis can be utilized as a temporizing measure for large symptomatic effusions. Revision of the shunt remains definitive treatment.10

In conclusion, ventriculo-pleural shunt insertion, though uncommon, is occasionally utilized in adults. Clinicians need to be alert to the possibility of pleural effusions complicating the course of these ventriculo-pleural shunts, and that those could present a few years later. They could be transudative or exudative in character.6 Revision of the shunt may be considered in these cases.

**REFERENCES**