There is growing evidence that fluid accumulation in the neck as a consequence of overnight rostral fluid shift contributes to the pathogenesis of obstructive sleep apnea (OSA).\(^1\) Superior vena cava syndrome (SVCS) includes various symptoms due to external compression or intrinsic obstruction of the superior vena cava (SVC), including cyanosis, distension of subcutaneous vessels, and edema of the upper extremities, head, and neck. Fluid accumulation in the neck may compromise the function of the larynx or pharynx, causing dyspnea, stridor, cough, hoarseness, and dysphagia.\(^2\) This is a report of a catheter-related SVC thrombosis revealed by worsening of snoring and rapid onset of typical OSA related symptoms.

**REPORT OF CASE**

A 67-year-old man was referred for clinical suspicion of OSA. His main medical history was multifocal motor neuropathy with conduction block treated with intravenous immunoglobulin every 6 weeks through a subcutaneously implanted port-chamber catheter in the right cephalic vein. Apart from a long history of loud snoring, the patient reported no typical OSA associated symptoms in the past. In recent weeks, his wife noted an increase in snoring intensity and described gasping episodes. The patient had severe excessive daytime sleepiness with an Epworth Sleepiness Scale (ESS) score of 19/24.

His body mass index (BMI) was 26 kg/m\(^2\) with no recent history of weight gain. Physical examination revealed typical signs of SVC obstruction with facial and neck edema, jugular venous distension, and dilated superficial thoracic collateral vein. Upper airway exam revealed no evident septal deviation, no dental anomalies, and no adenotonsillar hypertrophy. The patient had a relatively large tongue with a Mallampati II presentation of the palate’s velum. Chest computed tomography (CT) revealed a large occlusive thrombus in the SVC, in the brachiocephalic vein and in the right internal jugular vein.

Overnight respiratory monitoring with continuous recording of oxygen saturation (Sp\(O_2\)), nasal-oral airflow, and chest and abdominal wall motion showed severe OSA with an apneahypopnea index (AHI) of 64 events/h (Table 1). The patients received low-molecular-weight heparin followed by fluindione oral anticoagulation and the central venous catheter was removed. Continuous positive airway pressure (CPAP) was initiated with self-adjusted pressure support between 4 and 14 cm H\(2\)O. At 6 weeks, mean CPAP compliance was 4.5 h/night, the patient was no longer sleepy (ESS = 4/24), and physical examination showed a partial reduction of the signs of SVCS. Overnight respiratory recording after 5 nights of CPAP withdrawal showed a marked decrease in the apnea index but only a slight decrease in the AHI (Table 1). A repeat chest CT showed the disappearance of the brachiocephalic vein thrombosis but persistence of 70% occlusion of the SVC near its junction with the right atrium. A percutaneous transluminal balloon angioplasty was performed, and the patient was maintained on oral anticoagulant therapy. Three months later, BMI was unchanged, mean CPAP compliance was 6.1 h/night, the ESS was 6/24, and physical examination showed a complete resolution of the signs of SVCS. A third overnight respiratory recording after 5 nights of CPAP withdrawal demonstrated a 58% reduction in AHI (Table 1).

**DISCUSSION**

Complex anatomic and physiologic factors are involved in upper airway (UA) collapse in patients with OSA. Recent advances in the understanding of the pathogenesis of OSA has provided strong evidence that fluid accumulation in the neck as a consequence of overnight rostral fluid shift contributes to increase UA collapsibility.\(^1\) In humans with obstruction of the SVC, the cervical venous pressure is usually increased to 20 to 40 mm Hg (normal range, 2 to 8 mm Hg).\(^3\) The increased fluid accumulation in the neck may contribute to the pathogenesis of OSA in patients with SVC obstruction. This is the first report of catheter-related SVC thrombosis associated with worsening of OSA. Further studies are needed to assess the role of fluid accumulation in the neck as a risk factor for OSA in patients with SVC obstruction.
hydrostatic pressure in the cervical veins is likely to cause transudation of fluid into the interstitial space surrounding the UA.\textsuperscript{1} Intrathoracic malignancies are responsible for 60% to 85% of SVCS cases.\textsuperscript{2} A few cases of OSA associated with SVCS due to malignant or non-malignant mediastinal compression of the SVC have been previously described,\textsuperscript{4–9} with some cases reporting relief of OSA after radiation, chemotherapy, and/or stent insertion into the stenotic SVC.\textsuperscript{5–9} A case of OSA associated with SVC thrombosis has also been previously described in a patient with familial Mediterranean fever.\textsuperscript{10} The modern practice of medicine has seen increased use of intravascular catheters. Although accounting for a substantial proportion of non-malignant SVCS, the incidence of catheter-related SVC thrombosis appears to be low.\textsuperscript{11} A recent report described severe OSA requiring continuous positive airway therapy as a complication of catheter-related SVC thrombosis in a 15-year-old girl with infantile nephrotic syndrome.\textsuperscript{12}

In the present report we describe a case of catheter-related SVC thrombosis revealed by worsening of snoring and rapid onset of typical symptoms of OSA. Although our patient did not report typical OSA related symptoms in the past, the persistence of moderate sleep disordered breathing (SDB) with an AHI of 27 events/h after resolution of SVCS indicates that he had at least moderate OSA prior to presentation. However, the contribution of SVC thrombosis to the worsening of OSA was supported by a marked decrease in SDB severity and related symptoms after central venous catheter removal, anticoagulant therapy, and SVC angioplasty. It seems highly unlikely that any confounding factor contributed to the improvement of SDB, as the patient had no weight loss and took no drug except oral anticoagulation during the follow-up period.

Clinicians should be aware that SVC obstruction might be revealed by symptoms of OSA.  

Table 1—Clinical and nocturnal polygraphic evaluation at baseline and during follow-up.

<table>
<thead>
<tr>
<th></th>
<th>Baseline (07/01/13)</th>
<th>6 weeks after venous catheter withdrawal (09/17/13)</th>
<th>3 months after SVC angioplasty (12/19/13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index, kg/m(^2)</td>
<td>26</td>
<td>26</td>
<td>26</td>
</tr>
<tr>
<td>Epworth Sleepiness Scale score</td>
<td>19</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>Overnight respiratory recordings</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apnea-hypopnea index, n</td>
<td>64</td>
<td>50</td>
<td>27</td>
</tr>
<tr>
<td>Apnea index, n</td>
<td>52</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>% ODI, n</td>
<td>65</td>
<td>42</td>
<td>21</td>
</tr>
<tr>
<td>Mean (\text{SpO}_2), %</td>
<td>92</td>
<td>92</td>
<td>91</td>
</tr>
<tr>
<td>T90, %</td>
<td>15</td>
<td>8</td>
<td>10</td>
</tr>
</tbody>
</table>

SVC, superior vena cava; ODI, oxygen desaturation index; \(\text{SpO}_2\), oxygen saturation; T90, time spent with oxygen saturation below 90%.

REFERENCES


SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication January, 2015
Submitted in final revised form February, 2015
Accepted for publication February, 2015
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DISCLOSURE STATEMENT

This was not an industry supported study. The authors have indicated no financial conflicts of interest.