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Kidney Case Conference: How I Treat

120 Pregnancy in a Kidney Transplant Patient
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What’s the diagnosis?
In 2014, a 37-year-old white man previously healthy presented with an increasing abdominal girth over two weeks and blood pressure of 160/85 mmHg. Physical exam was significant for abdominal distention with dullness to percussion. Ultrasound confirmed ascites and paracentesis was consistent with exudative fluid. Computed tomography (CT) scan revealed extensive peritoneal and omental thickening with diffusely enlarged kidneys (Figure 1). Ultrasound-guided omental, peritoneal, and renal biopsies were diagnostic of a sclerosing fibro-inflammatory proliferation with foamy histiocytic infiltrate. Histopathology was reviewed and verified by three independent laboratories. The cellular infiltrate was considered nonmalignant, and no specific treatment was initiated, and the lesions abated spontaneously. During the clinic follow up, his hypertension workup included serum aldosterone and renin activity which were elevated at 30.6 ng/dl and 14.2 ng/ml/hr respectively, when his blood pressure was 173/87 mmHg; suggestive of renovascular-mediated hypertension. His repeat CT scan showed interval development of subcapsular densities surrounding both kidneys. A diagnosis of secondary hypertension due to Page kidney process was made, and spironolactone was initiated, which controlled his blood pressure to 133-144/76-85 mmHg (Table 1). Page kidney is analogous to the renal wrap hypertension model that is also renin-dependent [1,2]. He sustained recurrent acute kidney injuries due to partial obstruction, primarily due to the encapsulation of the renal pelves and ureters that required ureteral stenting. In these instances, spironolactone was discontinued due to the obstruction-associated hyperkalemia; his blood pressure needed multiple high doses of alternative anti-hypertension medications to achieve comparable blood pressure control. In June 2015, the infiltrate recurred, and the histology was deemed to have developed into more aggressive pre-malignant form. He was treated with prednisone with stabilization. He remained on spironolactone until late 2017, but due to recurrent episodes of acute kidney injury with hyperkalemia and progressive loss of function (eGFR in 2019, 33 ml/min per 1.73m²), his blood pressure has been treated with less-than-optimal success on four medications plus a diuretic. (Cover Image and text provided by Nashat Imran, Internal Medicine Department, Nephrology Division, Assistant Professor, Wayne State University; Nadine Abdallah, Internal Medicine Department, Medical Resident, Wayne State University; and Noreen F. Rossi; Internal Medicine Department, Nephrology Division, Professor, Wayne State University.)

Table 1. Renin and Aldosterone Levels and Treatment Effect on Outpatient Blood Pressure over Time

<table>
<thead>
<tr>
<th>Month</th>
<th>Systolic</th>
<th>Diastolic</th>
<th>Renin ng/ml/hr</th>
<th>Aldosterone ng/dl</th>
<th>Spironolactone (on/off)</th>
</tr>
</thead>
<tbody>
<tr>
<td>May 2014</td>
<td>173</td>
<td>87</td>
<td>14.2</td>
<td>30.6</td>
<td>Time of diagnosis (off)</td>
</tr>
<tr>
<td>July 2014</td>
<td>144</td>
<td>85</td>
<td></td>
<td></td>
<td>on</td>
</tr>
<tr>
<td>November 2014</td>
<td>133</td>
<td>76</td>
<td></td>
<td></td>
<td>on</td>
</tr>
<tr>
<td>March 2015</td>
<td>140</td>
<td>80</td>
<td></td>
<td></td>
<td>on</td>
</tr>
<tr>
<td>June 2015</td>
<td>155</td>
<td>91</td>
<td></td>
<td></td>
<td>off</td>
</tr>
<tr>
<td>December 2015</td>
<td>154</td>
<td>90</td>
<td>6.1</td>
<td>38.3</td>
<td>off</td>
</tr>
<tr>
<td>December 2016</td>
<td>151</td>
<td>85</td>
<td></td>
<td></td>
<td>on</td>
</tr>
<tr>
<td>August 2017</td>
<td>145</td>
<td>85</td>
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<td></td>
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<tr>
<td>July 2018</td>
<td>137</td>
<td>71</td>
<td></td>
<td></td>
<td>on</td>
</tr>
<tr>
<td>January 2019</td>
<td>173</td>
<td>97</td>
<td></td>
<td></td>
<td>off</td>
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References