Renal Vein Thrombosis*

Initial Manifestation of Goodpasture's Syndrome

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We report a patient who presented with renal vein thrombosis and nephrosis that progressed to alveolar hemorrhage and renal failure. Renal biopsy immunofluorescence and serum antiglomerular basement membrane antibody titer studies confirmed the diagnosis of Goodpasture's syndrome. To our knowledge, this is the first report of renal vein thrombosis as the initial presentation of Goodpasture's syndrome. (Chest 1991; 99:239-40)

Goodpasture's syndrome is an immunologic disorder characterized by glomerulonephritis, pulmonary hemorrhage and anti-glomerular basement membrane (GBM) antibody formation.1 Although most patients have some degree of proteinuria, frank nephrosis is distinctly unusual.1,2 Furthermore, renal vein thrombosis has not been reported, to our knowledge, as the initial presentation of Goodpasture's syndrome.

We report a patient who presented with renal vein thrombosis and nephrosis that progressed to pulmonary hemorrhage and renal failure. Results of renal biopsy and serum anti-GBM assay confirmed the diagnosis of Goodpasture's syndrome. This unique presentation contributed to the delay in correct diagnosis and initiation of appropriate therapy.

CASE REPORT

A 23-year-old woman presented with leg edema and left flank tenderness. Serum blood urea nitrogen was 13 mg/dl, creatinine 1.4 mg/dl, albumin 2.2 g/dl and cholesterol 276 g/dl. Urinalysis revealed 4+ proteinuria. A renal venogram demonstrated left renal vein thrombosis (Fig 1). Chest radiograph showed subtle bilateral alveolar infiltrates, which were ascribed to nephrosis and volume overload. Renal vein thrombosis secondary to the nephrotic syndrome was diagnosed and the patient was treated with intravenous heparin that was converted to coumadin at discharge.

Two weeks later, the patient was readmitted with severe dyspnea, anasacca, oliguria, cough productive of blood-tinged sputum, and nausea. A chest radiograph showed bilateral alveolar infiltrates most marked in the lower and mid lung zones (Fig 2). Blood urea nitrogen was 111 mg/dl; creatinine, 10 mg/dl; potassium, 7.2 mmol/L; and hematocrit, 10.7 percent. Urinalysis revealed 4+ proteinuria and hematuria. The patient was intubated because of progressive hypoxemia and transferred to the Medical University of South Carolina. Endotracheal secretions were blood-tinged and contained numerous hemosiderin-laden macrophages.

Following stabilization with hemodialysis, a renal biopsy specimen was obtained that showed 75 percent of glomeruli involved with crescentic glomerulonephritis and linear deposition of IgG and C3.
unknown, vascular thrombosis appears to be a consequence of the hypereosinophilic state that accompanies nephrosis rather than a cause, as was previously suspected, of the renal lesions. In adults, renal vein thrombosis most commonly occurs with membranous and membranoproliferative glomerulonephritis, although associations with renal sarcoidosis, amyloidosis, lupus nephritis and sickle cell anemia have also been described. To our knowledge, renal vein thrombosis has not been previously reported in a patient with nephrosis associated with Goodpasture's syndrome.

We suspect that renal vein thrombosis in our patient resulted from anti-GBM-related nephrosis that preceded the more typical features of Goodpasture's syndrome, such as frank alveolar hemorrhage and azotemia. Nephrotic syndrome has been noted infrequently as a complication of Goodpasture's syndrome, probably because of the typically fulminant course of renal insufficiency that occurs in anti-GBM disease. This rapid course may also underlie the apparent rarity of renal vein thrombosis in patients with Goodpasture's syndrome.

Other etiologies of pulmonary hemorrhage and glomerulonephritis have been described. These include Wegener's granulomatosis, systemic lupus erythematosus, Henoch-Schönlein purpura, and cryoglobulinemia. Although there is an immunologic basis to these disorders, circulating anti-GBM antibodies have not been demonstrated. This unique presentation of Goodpasture's syndrome contributed to the delay in correct diagnosis of the present patient. Furthermore, the administered anticoagulation therapy for renal vein thrombosis may have exacerbated the pulmonary hemorrhage and eventual degree of respiratory failure.

In summary, renal vein thrombosis secondary to nephrosis is an unusual but important initial presentation of Goodpasture's syndrome. Failure to detect the underlying disorder may prompt initiation of inappropriate therapy for thrombosis, such as anticoagulant drugs, thereby aggravating the degree of respiratory failure from subsequent alveolar hemorrhage.

REFERENCES
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Lightning Injury with Lung Bleeding in a Tracheotomized Patient

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A 68-year-old man, who had undergone laryngectomy six months earlier, was struck by lightning and developed pulmonary hemorrhage. This was attributed to pulmonary barotrauma due to a lightning blast via his tracheostoma. (Chest 1991; 99:240-42)

Lightning injuries occur only rarely, but one third of the patients die. The reported manifestations of this kind of electrical trauma are: burns, unconsciousness and amnesia of variable degree, barotrauma to the middle ear, temporary paralysis of peripheral nerves, and cardiac arrhythmias. Lung manifestations of lightning injury appear to be rare. In the literature we found only one well-documented case of blast injury to the lungs due to lightning. We report a case of lung injury and tympanic rupture due to lightning in a tracheotomized patient. To our knowledge, no reported similar case exists.

CASE REPORT

When the 68-year-old patient was struck by lightning while fishing during a thunderstorm, he remained conscious, heard a tremendous blast, was thrown to the ground, and realized that his clothes had exploded, his boots and fishing gear were missing, that he had fallen on his right side and could neither move nor feel his left leg. He saw that he was coughing fresh blood.

He had a smoking history of 50-pack-years and six months earlier had undergone uncomplicated total laryngectomy with irradiation for supraglottic cell carcinoma and was not carrying a tracheal cannula.

On admission, we found a smoky smelling, well oriented but tachycardic patient with his heavy clothing torn into pieces (Fig 1). He had 1st and 2nd degree burns mainly on his left side (Fig 2) and his legs with a burnt surface of 22 percent. We could not detect any typical entry burns. His tracheostoma showed several linear centrilobular blood marks and a few fresh blood clots (Fig 3). He was able to move all extremities. Sensibility and peripheral pulses were intact. Suspected rupture of the left tympanic membrane was otoscopically proven. His ECG showed sinus tachycardia, but no arrhythmia. His chest x-ray film revealed a diffuse, ill-defined density all over the left lower field. Laboratory results were normal except for mild metabolic acidosis. After initial evaluation and fluid resuscitation, bronchoscopy was performed, which revealed the left lower lobe as the source of hemorrhage. The exact bleeding spot, however, could not be pinpointed and the tracheobronchial mucosa appeared normal. Cytology was negative for recurrent malignancy. Hemothysis subsided spontaneously after 4 h. His chest x-ray

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