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Case Report

ANCA glomerulonephritis in a patient with active tuberculosis

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Abstract TANCA vasculitis is a necrotizing vasculitis that commonly affects the medium and small vessels. Characterized by necrotizing granulomatous inflammation and the presence of anti neutrophil cytoplasmic antibodies (ANCA). However, tuberculosis may sometimes lead to elevation of serum ANCA titers, making it hard to diagnose and should always rule out ANCA vasculitis in the differential diagnosis, to ensure fast response within a short period of time. Here we presented a case manifested initially with symptoms suggestive with active tuberculosis, remission noted within appropriate treatment. However worsening renal failure findings and serum ANCA positive titers, suggest actual ANCA vasculitis, which confirmed by renal biopsy.

Keywords: ANCA, Tuberculosis, Glomerulonephritis, ANCA vasculitis

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Introduction

ANCA associated vasculitis is a type of autoimmune disease that causes necrotizing vasculitis, usually affects the medium and small arteries. It can be associated with ANCA specific for myeloperoxidase (MPO-ANCA/P-ANCA) or proteinase 3 (PR3-ANCA/C-ANCA). Symptoms depend on what organ has been involved. When ANCA vasculitis involves the kidney, these ANCA antibodies attack small blood vessels in the glomeruli and damage it. ANCA glomerulonephritis is the term used when ANCA vasculitis has involved the kidney.

Major variants of ANCA associated vasculitis include Microscopic polyangiitis, Granulomatosis with polyangiitis (Wegener's), and Eosinophilic granulomatosis with polyangiitis (Churg- Strauss). However, some infectious diseases, such as bacterial endocarditis and tuberculosis may sometimes show high titers of ANCA, mimicking vasculitis. Tuberculosis is an infection caused by Mycobacterium tuberculosis and is believed to induce the development of antibodies such as positive (ANCA) that can be indicators for the diagnosis of other diseases. Based on reports, conflicting results found on tuberculosis patients with a positive ANCA.

Case Report

A 77-year-old Guatemalan male with no known past medical history, presented with dyspnea and dry cough for 4 weeks. CXR and CT chest revealed fibrosis, nodules and a small cavitary lesion in the upper lobe. BAL confirmed active pulmonary TB with positive acid-fast bacilli stain. Patient was started on Anti-tuberculosis (TB) drugs. However, his creatinine began to worsen from 2.3mg/dl on admission to 4.6mg/dl. Multiple urinalysis revealed microscopic hematuria. He had 5 grams of proteinuria based on protein to creatinine ratio. Workup for AKL was unrevealing. P-ANCA was elevated to 1:640 (normal, <3.5 U/mL). Renal biopsy was performed, and revealed active chronic pauci-immune crescentic glomerulonephritis.

Figure 1: see proliferation of cells occupying the entire Bowman's space (green arrows) and compressing the tuft (red arrows). The blue arrows indicate the Bowman's capsule. This is the characteristic aspect of an epithelial crescent, in this case circumferential. Cells forming the crescent may be epithelial, monocytes or other inflammatory cells. (Masson's trichrome, X400).

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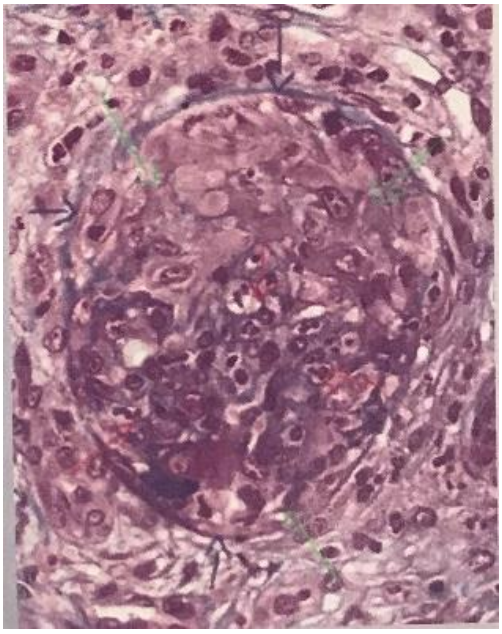

Figure 1 cites: Mualla, N. M. (n.d.). Bowman's space. photograph, LA.

Figure 2: This immunofluorescence micrograph of a glomerulus demonstrates positivity with antibody to fibrinogen. With a rapidly progressive GN, the glomerular damage is so severe that fibrinogen leaks into Bowman's space, leading to proliferation of the epithelial cells and formation of the bright crescent shown.

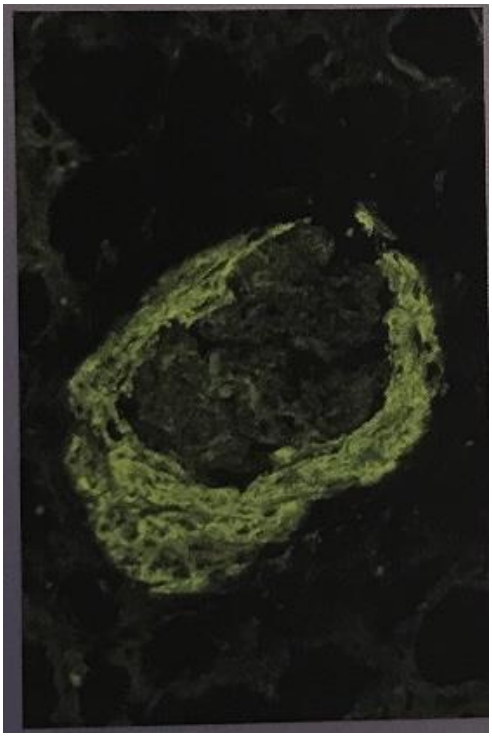

Figure 2 cites: Mualla, N. M. (n.d.). Immunofluorescence micrograph of a glomerulus. photograph, LA.

Discussion

From our search of the literature, we found only one other case of ANCA glomerulonephritis in a patient with active tuberculosis.

There are many reported cases in the literature of patients with tuberculosis who had positive ANCA titers, but none had actual ANCA vasculitis. However, in this case, remission of TB was noted after initiating the anti TB treatments, while the ANCA titer remained high, with worsening creatinine level and renal failure findings, all indicating the presence of actual ANCA vasculitis.

We can not be certain if our patient had two unrelated disease processes, or if the elevated ANCA titers as seen in other cases of tuberculosis, actually lead to an ANCA vasculitis in our patient. The other uncertainty is whether or not tuberculosis can actually predispose patients to ANCA vasculitis.

Of note, the management is difficult for these patients as immunosuppression could potentially worsen the outcome of a patient with active tuberculosis.

The etiology underlying remains unclear. The most common etiology was "anti-TB drug-induced vasculitis".

Based on previous case reports showing various anti- TB drugs have been reported as causative agents for vasculitis, such as **propylthiouracil (PTU)**, **isoniazid (INH**) and **rifampicin**. These agents might interrupt the resolution of the neutrophil extracellular trap, acting as the mechanism behind vasculitis. The other explaining etiology is that secondary elevation of ANCA induced by a chronic inflammation by TB infection lead to development of vasculitis.

Hospital course

While the patient was receiving treatment for TB, we instituted 60mg prednisone daily. Once repeat BAL was negative for acid-fast bacilli, we then added weekly Rituximab infusions. By his third dose, his creatinine had

decreased to 2.8mg/dl and proteinuria had decreased to 3.2 grams. Unfortunately due to the patient's financial constraints he was later lost to follow up.

Conflicts of Interest

None

Acknowledgments

None

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