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Idiopathic Membranoproliferative Glomerulonephritis, Leukocytoclastic Vasculitis: A Case Report on a 67-Year-Old Female with Chronic Sinusitis

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Authors' contributions

This work was carried out in collaboration between all authors. Author ET formally initiated documenting the case report, wrote the first draft of the manuscript and provided approval of the final manuscript. Authors EDZ, RZ and VST managed the analyses of the case report. Authors IDZ and MT managed the literature searches, wrote and edited the final manuscript. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

This case report is about a 67-year-old female patient who presented with an unusual case of acute renal failure and leukocytoclastic vasculitis. The patient required emergency dialysis; an ultrasound-guided kidney biopsy revealed membranoproliferative glomerulonephritis type one, a condition where the immune system damages both the membrane and the mesangium of the glomerulus. This condition was treated with therapeutic plasma exchange, pulse steroid therapy, and Rituxan, which lowered creatinine levels and stimulated greater urine output. These treatments resulted in attaining acceptable creatinine levels and the patient's subsequent discharge from the facility.

Keywords: Idiopathic; membranoproliferative; glomerulonephritis; cryoglobulinemia; vasculitis.

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1. INTRODUCTION

"Membranoproliferative Glomerulonephritis (MPGN) is an uncommon cause of glomerulonephritis" [1]. It accounts for 6 to 12% of all causes of glomerulonephritis [2]. Most of the cases are type two and three which are associated with Hepatitis C viral infection (HCV) [3]. We herein report an unusual case of acute renal failure and leukocytoclastic vasculitis in a 67-year-old female with chronic sinusitis.

2. CASE REPORT

A 67-year-old female presented to the hospital with weakness, swelling in legs, periorbital puffiness, and diffuse purpuric skin rash involving upper and lower extremities (Fig. 1). She had a longstanding history of hypertension, chronic obstructive pulmonary disease, hypothyroidism, and suffered from chronic sinusitis. The patient exhibited sinusitis symptoms for approximately six weeks before her visit and showed no improvement despite multiple regimens of antibiotics.

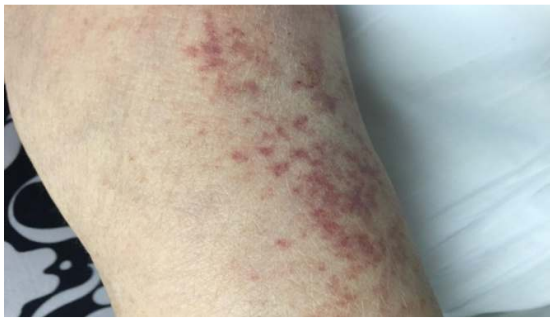

Fig. 1. Diffuse purpuric skin rash

On presentation, the patient's blood pressure was 160/95, no fever was evident. Diffuse crackles over both lung bases indicated excess fluid in the airway, and the patient had plus two pitting edema. Her serum white blood cell count was 13,000 microliters, hemoglobin was 10 grams per deciliter, and platelet count was 150,000 per microliter. The patient displayed a Blood urea nitrogen of 80 milligrams per deciliter (mg/dl), creatinine of 4 mg/dl, and bicarbonate level of 17 millimoles per liter. Urine analysis showed nephrotic nephritic syndrome with red blood cell casts.

The patient was oliguric and began having shortness of breath. Because of worsening symptoms and impending respiratory failure, the patient required emergency dialysis. An ultrasound-guided kidney biopsy was obtained which revealed MPGN type one associated with exudative component and hyaline capillary thrombi with cryoglobulinemic nephropathy. Immunofluorescence showed staining of tubular casts for both kappa and lambda light chains as well as immunoglobulin A and immunoglobulin M (IgM) (Fig. 2). Serum C3 and C4 complements were low. Serum immunoelectrophoresis showed slightly elevated immunoglobulin G (IgG) level. Hepatitis C antibody, as well as Hepatitis C RNA PCR, was negative. Cytomegalovirus, Human immunodeficiency virus (HIV), Epstein-Barr virus serology was also negative. Serum cryoglobulin level 200 mg/L (reference range 0-60 mg/L).

The patient underwent therapeutic plasma exchange five times, in addition to pulse steroid therapy, followed by four doses of Rituxan. Over the following two weeks, creatinine decreased with a significant increase in urine output, and dialysis was stopped because of renal recovery. The patient was subsequently discharged with a creatinine of 1.35 mg/dl.

3. DISCUSSION

Cryoglobulinemia is associated with the presence of cryoglobulins in the serum. Cryoglobulins are abnormal protein antibodies that tend to precipitate in vitro at temperatures below 98.6. Cryoglobulinemia is responsible for membranoproliferative glomerulonephritis that is associated with the proliferation of the mesangial and endothelial cell, as well thickening of the capillary walls due to subendothelial deposits. Increased mesangial cellularity is also noted.

There are three types of cryoglobulinemia [4,5,6]. Type one or simple is usually associated with IgM monoclonal antibodies and IgG and to a lesser extent light antibodies. Whereas type two and three or mixed cryoglobulinemia which are more common contain the rheumatoid factor antibody that is mostly IgM and is associated with hypocomplementemia [7].

Mixed cryoglobulinemia is seen in up to 40% of patients with HCV [8]. Less frequently it can be secondary to connective tissue disorders including systemic lupus, rheumatoid, Sjogren's syndrome, lymphoproliferative disorders as well as infections like HIV and hepatitis B. Treatment includes addressing HCV and treating it with the latest antiviral therapy. Other treatments include corticosteroids, cyclophosphamide and plasmapheresis are reserved for severe cases.

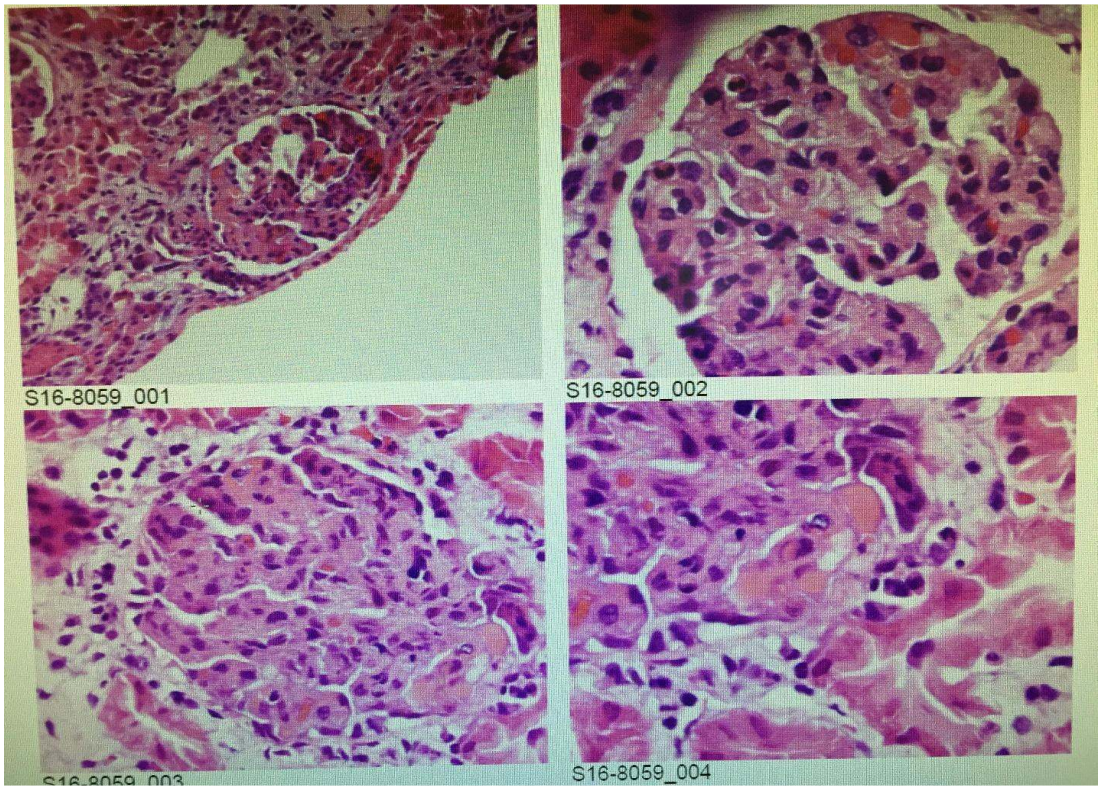

Fig. 2. Immunofluorescence showing staining of tubular casts

4. CONCLUSION

Idiopathic mixed cryoglobulinemia is uncommon but can present with an aggressive form of MPGN that can rapidly destroy the kidneys. As this case illustrated, prompt diagnosis and aggressive management with plasmapheresis, Rituximab [9,10], and supportive dialysis with pulse steroids can have a positive impact and slow the rate of kidney damage, which contributed to the opportunity of a longer, healthier life.

CONSENT

As per international standard or university standard, patient's written consent has been collected and preserved by the authors.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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