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# A case of membranoproliferative glomerulonephritis associated with psoriasis vulgaris

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#### **Abstract**

**Background:** Psoriasis is a chronic inflammatory disorder of the skin; the hallmark of psoriasis is sustained inflammation that leads to uncontrolled keratinocyte proliferation and dysfunctional differentiation. Psoriasis shows clear autoimmune-related pathomechanisms, in which autoantigen-specific T cells contribute to the development, chronification, and overall course of the disease. Psoriasis is primarily a skin disease, but other internal organs may be involved; affliction of the joints is well established, but kidney involvement is yet to be universally agreed upon, although some recent studies have linked psoriasis to kidney disease and certain glomerular diseases including secondary renal amyloidosis, Ig A nephropathy, and membranous glomerulopathy.

**Case presentation and conclusion:** In this paper, we report a case of primary membranoproliferative glomerulo-nephritis (MPGN) with nephrotic impure syndrome in a psoriatic patient. The etiological investigation was negative with normal serum complement and ASO titers and were negative for HBV, HCV, HIV, and ANA. The patient improved on prednisolone and angiotensin-converting enzyme inhibitors. To our best of knowledge, cases of MPGN associated with psoriasis are rare and we would like to shed light on this association.

Keywords: Psoriasis, glomerulonephritis, Nephropathy

#### **Case report**

A 23-year-old male patient was referred to our hospital for limb swelling, fatigue, and a skin rash evolving for 3 months. The medical history showed risky sexual behavior, chronic weaned smoking, and occasional alcoholism; he was not diabetic nor had hypertensive history and had promiscuity for 8 months in prison. There was no other considerable morbidities, with no family history of kidney disease. The patient was conscious and hypertensive with a blood pressure reading 150/90 mmHg, without signs of dehydration with preserved diuresis. Upon examination, the patient presented edema of the lower limbs, abdominal distension with ascites, and a

hydrocele; further inspection showed multiple erythematous, hyperkeratotic plaques (Fig. 1) with silvery squames on the extensor surfaces of elbows, knees, back, and scalp and a scar of chancre on the penis with 2 achromic spots (Fig. 2). There were no signs of arthritis on joint examination.

Upon admission, serum albumin level was low at 19 g/l, serum urea at 1.01 g/l, AKI 1 at 23mg/l, and severe vitamin D deficiency at 3ng/ml; his hemoglobin level was 12.4 g/dL with a mean corpuscular volume of 81.7 fL. White blood cell count and platelet count were within normal range, with a lymphocyte count of 5207. Liver enzyme tests, serum bilirubin, and alkaline phosphatase levels were within normal range. Electrolyte assessment showed normal natremia at 136 mmol/l, kalemia at 3.8 mmol/l, low calcium level at 1.7 mmol/l, and a slight increase in phosphorus at 1.74 mmol/l. Serum albumin level was low at 19 g/l, serum urea at 1.01 g/l, serum

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Fig. 1 Erythematous plaques with silvery squames on extensor surface of the knees and scale



Fig. 2 Scar of chancre and achromatic spots on penis

creatinine at 23 mg/l, and severe vitamin D deficiency at 3 ng/ml. Urine analysis revealed mild leukocyturia and hematuria with sterile cultures. Proteinuria in 24 h urine was at  $4.4 \, \text{g/day}$ .

Abdominal ultrasound showed ascites, with well-differentiated kidneys of normal size.

Patient's immunological assessment was negative for antinuclear antibody (ANA), anti DNA, serum

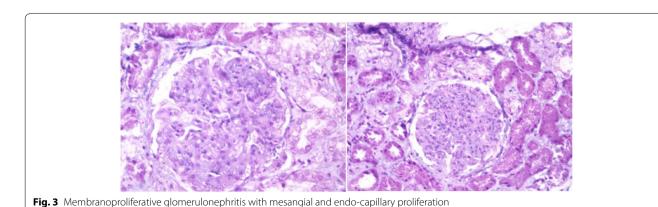
complement C3 and C4, and CRP were within normal range.

Serological markers of hepatitis B and C and syphilis (TPHA-VDRL) were all negative. Given the endemic nature of tuberculosis in the country, and promiscuity within the prison, the patient underwent a QuantiF-ERON test, which came back negative. Serum protein electrophoresis and serum free light chain analysis were normal as well as electrocardiography.

An ultrasound-guided renal biopsy was performed revealing glomerulonephritis with mesangial and endocapillary proliferation of inflammatory cells (Fig. 3), with interstitial edema and inflammatory infiltrate made essentially of mononuclear lymphoplasmacytic cells; direct immunofluorescence finds peripheral membrane deport of IgA, IgG C3, and C1q, all compatible with membranoproliferative glomerulonephritis (Fig. 4).

Before we made the diagnosis of MPGN with psoriasis, we excluded other causes such as connective tissue diseases, chronic infections including tuberculosis, liver diseases, and thrombotic microangiopathy.

Treatment was initiated with ACE inhibitors, diuretics, and local corticosteroids; skin lesions have healed; blood pressure is back within normal range; there was decrease of



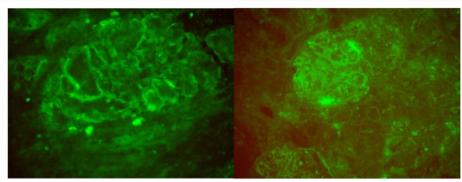


Fig. 4 Peripheral membrane deposit of IgA and IgG

edema; serum creatinine concentration is down to 13 mg/l; and urine analysis showed improvement in proteinuria of 0.37 g/day.

#### **Discussion**

This case widens the spectrum of glomerulonephritis in association with psoriasis. Renal glomerular diseases has already been described in several articles [1-5].

The clinical presentations are very polymorphic, ranging from asymptomatic microalbuminuria to massive nephrotic syndrome. Generally, the association between psoriasis and renal disease is rare, but new studies [6, 7] have shown that psoriasis can be accompanied with various glomerulopathy, and if left untreated, it can evolve into CKD and thus is considered a risk factor for CKD.

This new entity is called psoriatic nephropathy [1], but it is a new entity for which a consensus is not yet reached.

Amyloidosis secondary to chronic inflammation is well understood, but the glomerular involvement is not yet well explained and theories suggest a T cell-mediated immunological phenomenon.

IgA nephropathies are the most common in psoriasis [1, 3] or nephropathies secondary to nephrotoxic treatment of psoriasis vulgaris or articular psoriasis.

In this article, we describe a case of psoriasis that appeared concomitantly with membranoproliferative glomerulonephritis; any other cause of MPGN was ruled out, and the diagnosis of a form associated with psoriasis was retained.

MPGN generally have a poor prognosis if left untreated, with a 50% risk of developing into CKD; the therapeutic strategy was symptomatic treatment of the nephrotic syndrome and topical treatment of the psoriasis, pending the results of the etiological investigation.

#### Conclusion

We cannot say for sure that MPGN shown in this case should be considered as psoriatic nephropathy, but it is to shed the light on the subject and suggest that more studies and research, from clinical to histological findings, is needed to consider a real link between renal involvement and psoriasis. And until we have a clear definition of psoriatic nephropathy, it is in our modest opinion that patients with psoriasis should be subject to regular urine analysis and renal function monitoring for early diagnosis and treatment of coexisting lesions.

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#### Authors' contributions

MJ and AW and analyzed and interpreted the patient data. MJ, AW, and AM have contributed in the writing and correction of the article. All authors read and approved the final manuscript.

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#### Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

#### **Declarations**

#### Ethics approval and consent to participate

Not applicable.

#### Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

#### **Competing interests**

The authors declare that they have no competing interests.

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