done and sample was collected for toxicological analysis. After he was stabilized he was shifted to medical ICU and put on invasive ventilator support. Literature was reviewed after obtaining the poison container. As there was no antidote available, the patient was treated symptomatically with IV fluids, antibiotics as prophylaxis against aspiration pneumonia and supportive care provided with mechanical ventilation and general nursing care. After 12 hours of ventilation the patient was fully conscious and started developing neuropsychiatric manifestations like agitation and delirium. Due to severe agitation he self extubated so he was sedated and reintubated. After 96 hours, neuropsychiatric manifestations subsided and he was weaned off from the ventilator and extubated on day five. Patient was shifted to the ward on the next day and after psychiatric counseling he was discharged. After a week the patient was followed up in outpatient department, he was found fit to resume his work.

Discussion

Imidacloprid was developed in 1985 with the aim of combining compounds with high potency against insects with low mammalian toxicity and favorable persistence. On the basis of animal studies, it is classified as a “moderate toxic” (class II by WHO and toxicity category II EPA). It is not banned, restricted, canceled, or illegal to import in any country.

A few cases of significant human toxicity due to imidacloprid have been reported in medical literature. In a prospective human case series of 68 cases, the majority of the cases developed mild gastrointestinal symptoms and only one case required mechanical ventilation for respiratory failure.

Our patient during the clinical course of toxicity developed gastrointestinal irritation, respiratory failure and severe neuropsychiatric symptoms. With symptomatic and supportive care our patient recovered completely. Till date neuropsychiatric symptoms in imidacloprid poisoning has been reported in one case with inhalation exposure,5 mainly due to central nicotinic stimulation. Biochemical abnormality like metabolic acidosis in these cases may develop due to acidic metabolites such as 6-chloronicotinic acid and other metabolite. Cardiovascular manifestations were also described in different case reports like tachycardia, bradycardia, arrhythmia, cardiac arrest but were not present in our case.

Acknowledgment

We are thankful to Dr. Himanshu Pandya, Professor of Medicine, Pramukhswnami Medical College, Karamsad, Gujarat.

References


Nephropathy in Association with Annular Psoriasis

Gouranga Santra*, Pradip Kumar Sinha**, Dibyendu De***

Abstract

Occurrence of glomerular diseases in psoriasis is rare, although the number of reports is increasing in recent years. Different types of glomerular involvement have been reported but mesangio-proliferative glomerulonephritis with IgA deposits, AA amyloidosis and membranous nephropathy are relatively common in association with psoriasis. The term ‘psoriatic nephropathy’ has been introduced recently. We contribute a case to the ongoing discussion regarding psoriatic nephropathy. Our patient had mesangio-proliferative glomerulonephritis (with IgG and C3 deposition) in association with annular psoriasis (rare variety of chronic plaque psoriasis). Presence of mesangio-proliferative glomerulonephritis with IgG deposition is rare in association with psoriasis. The patient responded well to weekly methotrexate (15 mg) injection. Methotrexate has not been tried previously in psoriatic nephropathy or reported to be effective in it.

Introduction

Psoriasis is an immune-mediated chronic inflammatory disorder of the skin with distinct microvascular changes. It can involve joints but involvement of internal organs is uncommon. Psoriatic nephropathy is a recently described clinical disorder. Nephropathy in association with psoriasis is increasingly being reported in recent years.

Case Report

A 32 years old Hindu male from rural West Bengal presented with wide spread psoriasis with hair and nail involvement for last two years, which was aggravated since last four months. The patient presented to us with complaints of swelling of face and both lower limbs for last six days, along with decreased urine output for same duration. There was no history of fever, sore throat, and joint pain or swelling. He had no history of haematuria. He was previously non-diabetic and normo-tensive and had no past history of nephropathy.

On examination patient was conscious, oriented, and...
hypertensive. His BP was 160/110 mm of Hg. The patient had puffiness of face, pedal oedema and mild pallor. He had wide spread skin lesions distributed over both upper and lower extremities as well as on the trunk. Skin lesions were typically whitish and scaly dry. Skin lesions over the trunk were annular in shape having well defined raised margins with scaling. Auspitz sign was positive. Nail pitting was also present. He was diagnosed to have plaque psoriasis with annular variety (Fig. 1). There was no organomegaly. Examination of other systems revealed no abnormality.

His peripheral blood picture revealed Hb 11.0 gm%, TLC-11200/ cu mm with neutrophil 75%, lymphocyte 20%, monocyte 3%, eosinophil 2%, and 11200/ cu mm with neutrophil 75%, lymphocyte 20%, monocyte 596

was found to be trace, and urea and creatinine levels became normal.

After six weeks of follow up urine albumin was 1270 mg. A granular casts -present. 24 hours urinary protein excretion was 212 mg% and triglyceride 190 mg%. Urine examination revealed PH 6.0, albumin ++, RBC +, pus cells 12-15/ HPF, sugar- nil, granular casts -present. 24 hours urinary protein excretion was 1270 mg. ASO titer was within normal range (164 U/mL). Kidney biopsy revealed diffuse mesangioproliferative glomerulonephritis (Fig. 2) with immunofluorosence study showing IgG and C3 deposition. ELISA for HIV I and II was negative. ANF, p ANCA and c ANCA were negative. HBsAg and anti-HCV antibodies were nonreactive. Immunofluorescent study of skin biopsy from psoriatic lesions revealed presence of Ig G and C3 deposits in the stratum corneum.

The patient was treated with topical beclomethasone, coal tar and salicylate ointment for skin lesions. Renal problem was treated with diuretics and immunosuppressive (injection methotrexate 15 mg/ week). Antihypertensive (prazopressin) was added. Patient improved gradually, oedema subsided, and blood pressure came to normal. Nutritional supports including vitamins and minerals were provided to improve the common immune mechanism. After six weeks of follow up urine albumin was found to be trace, and urea and creatinine levels became normal.

**Discussion**

Psoriatic nephropathy is a recently described entity. Few cases of psoriasis with different types of glomerular involvement have been reported in literature. Mesangioproliferative glomerulonephritis, AA amyloidosis and membranous nephropathy are usually described in association with psoriasis.\(^1\)\(^2\) Focal proliferative glomerulonephritis, focal segmental glomerulosclerosis, mesangiocapillary glomerulonephritis and minimal change disease have also been reported in association with psoriasis but relatively rarely.\(^1\)\(^2\) Glomerulonephritis in psoriasis is mainly of a mesangioproliferative type with fixation of IgA and C3 on the mesangium and basal membranes of glomerular capillaries. Amyloid nephropathy in psoriasis has all the morphological features of acquired amyloidosis (AA-amyloidosis).

Increasing reports of nephropathy in psoriasis are arguing against chance association. But the exact relationship of psoriasis and nephropathy remains unclear, but an autoimmune mechanism most likely links the two. Probably the same inflammatory mechanism of psoriatic arthritis is responsible for nephropathy. Drug related nephropathy in psoriasis should be kept in mind because of high usage of offending drugs (e.g., NSAIDs in psoriatic arthritis).

Psoriatic patients have an increased risk of developing microalbuminuria due to subclinical glomerular disease.\(^4\) Creamer et al suggested that enhanced urinary albumin excretion in psoriasis could be mediated by plasma vascular endothelial growth factor/ vascular permeability factor (VEGF/VPF) derived from lesional skin during relapse of the disease.\(^5\)

Mesangioproliferative glomerulonephritis with IgA nephropathy and focal proliferative glomerulonephritis in psoriatic patients have active urinary sediment and reported to be improved with prednisolone therapy.\(^2\)\(^3\) Angiotensin-converting enzyme (ACE) inhibitors are beneficial.\(^3\) Amyloidosis in psoriasis is an aggressive disease with sporadic cases having favourable response to colchicine or infliximab.\(^6\)\(^7\) Rapidly progressing glomerulonephritis in psoriasis needs institution of early therapy. Protracted course is seen in subclinical nephropathy with microalbuminuria.

Our patient had nephropathy in association with annular psoriasis. Annular psoriasis is associated with ring-shaped plaques with central clearing. This is an uncommon type of chronic plaque psoriasis. Primary annular plaque-type psoriasis shares features of both typical plaque-type and annular pustular psoriasis.\(^8\)

---

**Fig. 1:** Showing psoriasis involving the extensor surface of forearm and trunk.

**Fig. 2:** Showing mesangioproliferative glomerulonephritis (H and E stain).
Our patient had mesangioproliferative glomerulonephritis with Ig G and C3 deposition in mesangium and basal membranes of glomerular capillaries. Presence of mesangioproliferative glomerulonephritis with Ig G deposition is rare in association with psoriasis. The patient responded well to weekly methotrexate (15 mg) injection. Methotrexate had not been tried previously in psoriatic nephropathy or reported to be effective in it.

Conclusion
Psoriatic nephropathy is a recently described disorder. Routine urinalysis, kidney function assessment and a wider application of renal biopsy in psoriatic patients may be helpful to detect renal involvement (subclinical or overt nephropathy). Psoriatic nephropathy may respond to methotrexate like psoriatic arthritis. Large trials are needed to establish the role of methotrexate in psoriasis associated nephropathy.

References

Radial Arterio-venous Fistula following Transradial Coronary Angiography
SR Mittal*

Abstract
A case of clinically detectable arterio-venous fistula following coronary angiography by right radial approach is reported. It is a rare complication. It may be clinically detectable only days or weeks following the procedure.

Introduction
Arteriovenous fistula formation is a known complication of percutaneous catheterization procedure from femoral approach. Clinically manifest arterio venous fistula is a rare complication of radial approach. This is due to easy compressibility of radial artery and absence of major veins around it.1, 2

Case Report
A 70 years hypertensive female underwent coronary angiography from right radial route three months back. It revealed critical stenosis of proximal right coronary artery. She underwent angioplasty and stenting from right femoral route.

Fig. 1: Colour flow imaging showing close proximity of radial artery and vein with communication between the two vessels

Fig. 2: Pulsed Doppler evaluation of radial artery showing high velocity diastolic flow