Acute interstitial nephritis related to lansoprazole administration
Jimmy Jose1, Kavitha Saravu2, Kanav Khera3, Beena Jimmy4, Barkur Anantha Krishna Shastry5
Department of Clinical Pharmacy1,3,4, Department of Medicine2,5, Kasturba Medical College, Manipal University, Manipal, Karnataka, India.

Abstract
Acute interstitial nephritis (AIN) is a rare but serious adverse effect reported with proton pump inhibitors (PPIs). Only one report (2 cases) of AIN with lansoprazole is published in literature. A case of lansoprazole induced interstitial nephritis (biopsy supported) was reported in an elderly female patient admitted to the hospital with non specific symptoms after 45 days of treatment with the drug. A recovery was noticed upon withdrawal of the drug and treatment with corticosteroids. We consider this report as a valuable addition to the existing literature on this rare adverse effect with lansoprazole. This potentially serious adverse effect with PPIs including lansoprazole has to be given due consideration, especially taking into account the wide spread use of this group of agents.

Introduction
Acute interstitial nephritis (AIN) is often caused by hypersensitivity to drugs.1-2 It is a well-recognized, but rare and serious adverse reaction reported with proton pump inhibitors (PPIs).3 Most of the reports of PPI induced AIN are with the use of omeprazole,1-3 even though other PPIs are also implicated.3-5 Based on the literature search done, we could find only one report (2 cases) of suspected lansoprazole induced AIN.1 We describe a case of suspected AIN due to lansoprazole, which would be an useful addition to the existing literature on this rare reaction to this drug.

Case Report
A 67 year old female patient was admitted to the hospital with complaints of nausea and dizziness for past 15 days. Medication history revealed diabetes mellitus from 1 year and hypertension from last 10 years. Medication history included metformin 500 mg BID from last 1 year and amlodipine 2.5 mg OD from last 2 years. Patient was recently started (45 days) on lansoprazole 30 mg OD for her gastrointestinal complaints. Laboratory investigations on the day of admission (day 1) revealed a raised serum creatinine (SCR); 3.8 mg/dl and blood urea nitrogen; 65 mg/dl. Liver function tests and blood counts were normal. Twenty hour urine protein done on day 3 was raised and urine analysis revealed, RBCs 15-20 cells/hpf, WBC 3-4 cells/hpf, and presence of proteins and granular casts. Ultrasonography demonstrated renal parenchymal changes and an increase in resistive index value was reported with renal artery doppler.

Lansoprazole induced AIN was suspected and the drug was withdrawn on day 6. Renal biopsy report was supportive of AIN. A course of prednisolone (1 mg/kg) was started and the patients renal function tests showed a progressive improvement with SCr levels on day 19 and day 25 being 3.2 mg/dl and 2.0 mg/dl. Rechallenge with lansoprazole was not done. Subsequent follow ups showed a complete recovery, with SCr level 1.1 mg/dl and 0.9 mg/dl after a period of 8 and 15 months.

Discussion
Even though PPI induced AIN is a well recognized adverse reaction, we consider the present report as significant due to rarity in reports (2 cases) where lansoprazole was the suspected drug. Drug induced AIN was considered in our case based on the temporal relationship with the administration of lansoprazole, absence of any other etiological factors, and significant improvement upon dechallenge. Rechallenge with the drug was not clinically warranted. Assessment using Naranjos probability scale showed a probable association between drug administration and development of AIN.6 Similar to our case, patients with PPI induced AIN often present with non-specific symptoms of illness (e.g. weight loss, malaise, fever and nausea)3 and diagnosis could only be confirmed with a renal biopsy.

Considering the 2 cases of lansoprazole induced AIN reported in literature1, the drug was withdrawn in both the cases and patient was treated with steroids. Subsequent follow up showed an improvement. Concomitant disease in one case included diabetes mellitus similar to our case. Our case has a similar presentation with regard to age of presentation, duration of treatment with PPI as in an analysis of series of PPI (omeprazole) induced AIN reported by Myers et al7 in which the average age at diagnosis was 65.8 years (range 36- 86) and average duration of PPI administration was 2.7 months (range 1 week to seven months).

The main direction of therapy is withdrawal of the offending drug and supportive management for acute renal impairment, with or without steroid treatment. Even though the evidence for use of steroids in drug induced AIN has
come from small, uncontrolled studies and case reports, they may have a role and are frequently used. Available data demonstrates a higher number and a shorter time interval for recovery of renal function. Use of prednisolone has probably contributed to the early and complete recovery of renal function in our case.

With more widespread use of PPIs including the relatively newer agents like lansoprazole, the potential for this rare but serious adverse reaction needs to be given due importance. Prompt evaluation of renal function is warranted in patients who develop non-specific symptoms while taking PPIs. Early recognition will assist in preventing irreversible renal injury.

References

Case Report

Colloid milium: a rare cutaneous deposition disease
Simeen Ber Rahman1, Arfan Ul Bari2, Nadeem Mumtaz3
Department of Dermatology1,3, Military Hospital, Rawalpindi, Department of Dermatology2, Combined Military Hospital, Muzaffarabad, AJK, Pakistan.

Abstract
Colloid milium is a rare degenerative skin disorder known by the development of small translucent, yellowish brown papular nodules or plaques, generally located in sun exposed areas. Clinically they are of two types, adult and juvenile type. We present a case of adult type Colloid milium in a 60 years old female patient with clinical and histological findings unmistakable of the condition. She was treated with IPL (Intense Pulsed Light) laser following unsatisfactory response with dermabrasion.

Introduction
Colloid milium is a rare cutaneous deposit disease characterized by the presence of multiple, dome-shaped, translucent yellowish brown papules and plaques developing on sun-exposed areas of skin showing colloid in dermal papillae on histology. There are 2 broad variants namely an adult-onset type (nodular colloid degeneration), and a juvenile form. The origin of the colloid deposition in the dermis is thought to be due to degeneration of elastic fibers in the adult form and due to degeneration of UV-transformed keratinocytes in the juvenile form. No known figures exist on prevalence, but more than 100 case reports are present in the world literature. The condition is more frequent in fair-skinned individuals and adult form is more common in elderly males. The rare juvenile form occurs before puberty and is often familial. Patients are usually asymptomatic, but they may have transient itching in affected areas. Skin lesions are waxy, partially translucent, firm papules that occur in crops, ranging from 1-5 mm in diameter. Lesions reach their peak within 3 years, after which they are more or less static. Gelatinous material can be expressed on pressing the lesions. In the nodular form, larger nodules or plaques develop. Most common sites of involvement are cheeks, periocular area, nose, ears, and neck. Lesions may also occur on the back of the hands and forearms. The classic adult and nodular forms are believed to be due to excessive sun exposure as the lesions mostly occurring on skin exposed sites in individuals with fair complexions and outdoor occupations. The juvenile form is inherited, perhaps suggesting an inherited susceptibility to UV light. Trauma, gas oils, phenols and long term use of hydroquinone bleaching creams may be contributory factors in addition to light and petroleum constituents. On histology, typical fissured eosinophilic colloid masses are seen in the dermis. Electron microscopy may sometimes be necessary to distinguish colloid from amyloid as under light microscopy both can show same staining pattern. Upon electron microscopy, wavy bundles of filaments are seen of colloid, in contrast to the straight, nonbranching, filaments of amyloid. Dermabrasion, cryotherapy, and diathermy treatments have been tried with limited success. The Er:YAG laser may be more successful than dermabrasion. Genetic counseling is advisable for the rare juvenile form. Sun avoidance seems sensible. We tried intense pulse light (IPL) with good response and to the best of our knowledge it was not used before.

Case Report
A 60 years old female presented with history of...