

Case Report

Acute interstitial nephritis related to lansoprazole administration

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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. Medical 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 wide spread 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

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