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A case study on over the counter use of antibiotics and NSAIDS leading to acute kidney injury

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Abstract
OTC medicines are easily accessible to common people. Many people prefer this medicine in order to relieve themselves from any kind of pain or injury they suffer. Excess use of these medications without consulting the doctor may lead to serious complications. Long term use of antibiotics and analgesics can degrade the renal functioning and other life threatening situation. We report a case of 48year old man suffered from nail injury and he has taken Amikacin and Analgesics for the injury which leads to kidney injury. This case will help people for making awareness regarding the use of OTC drugs.

Keywords: Nail injury, Acute kidney injury, Amikacin and Analgesic.

1. Introduction
Acute kidney injury previously called as acute renal failure is the sudden reduction in kidney function resulting in retention of nitrogenous(creatinine and urea) and other waste products as well as the failure to maintain fluid, electrolyte and acid-base homeostasis. The incidence of drug-induced nephrotoxicity has been increasing with the ever increasing number of drugs and with ease availability of the over the counter medication like Non-steroidal anti-inflammatory drugs (NSAIDs), antibiotics, angiotensin converting enzyme inhibitors (ACEI) and contrast agents are the major culprit drug contributory to kidney damage [3].

2. Case Presentation
A 48 year male was presented in emergency ward with a complain of open wound nail injury of his left foot which occurred due to trauma. On examination it was seen swelling was present on left foot. At the admission patient was conscious and had no complains except pain at the site of injury. The social history shows that the patient is mild alcoholic and no smoking habits. The patient was not having the history of diabetes and hypertension. In the causality ward, Analgesics and antibiotics are commonly administered intravenously to help patient to get relief from pain and also to prevent spread of infection as the wound is open.
The wound was aseptically cleaned and pus was drained and surgical dressing was done. The vitals were stable but there was increased in swelling of the leg the doctor advised liver function test and renal function test. On first day in hospital the creatinine was 6.8 and there was increase in WBC count. The creatinine level increased on the next day. After counselling patient about his previous medication, patient informed the doctor he was taking OTC medications for the injury from medical shop. The nephrologists advised immediately to stop analgesic and antibiotic. Other class of antibiotics were started. As there was increase in creatinine level to 9.7 doctor advised for hemodialysis, the patient underwent dialysis for two times. Complete monitoring of input and output was maintained. The creatinine level and WBC Counts was progressively coming to normal.
Along with the above drugs other medications were also prescribed to treat the patient. They are not mentioned as this article is mainly concentrated with antibiotics and analgesics. After 15 days of pharmacotherapy patient recovered from his injury and swelling was subsided. The patient was able to stand and balance on both the legs. The patient was clinically stable Laboratory reports were normal. The patient was counselled about OTC drugs can causes harm if taken without doctors advised. Always avoid non-prescription drug.

Table 1: Laboratory Finding

Lab investigation	Test Value	Normal Range
1st Day S. Creatinine	6.8	0.6-1.5
2nd Day S. Creatinine	7.2	0.6-1.5
3rd Day S. Creatinine	9.7	0.6-1.5
4th Day S. Creatinine	7.0	0.6-1.5
5th Day S. Creatinine	4.7	0.6-1.5
6th Day S. Creatinine	4.0	0.6-1.5
7th Day S. Creatinine	2.4	0.6-1.5
8th Day S. Creatinine	2.2	0.6-1.5
11th Day S. Creatinine	1.7	0.6-1.5
14th Day S. Creatinine	1.4	0.6-1.5

Table 2: WBC's count of the patient

WBC's Count of the patient at the admission	WBC's count of the patient at the discharge
27.300 cells/cumm	11,300cells/cumm

Table 3: antibiotics and analgesics given to patient not knowing of drug induced aki

S. No	Name of the drug	Dose, route and frequency	Duration of administration
1	Magnova	1.125 g I.V bd	D1-D2(stop)
2	Amikacin	500 mg I.V bd	D1-D3(stop)
3	Dolo	650 mg oral bd	D2-D3(stop)

Table 4: Other class of antibiotics and analgesic given to patient are

S. No	Name of the drug	Dose, route and frequency	Duration of administration
1	Clindamycin	600mg I.V bd	D3-D15
2	Meropenem	500mg I.V bd	D7-D9
3	Tramadol	50mg Oral (sos)	D7-D15
4	Dalacin	600mg Oral tid	D10-D15
5	Augmentin	625mg Oral tid	D10-D15
6	Dolonex	20mg IM bd (sos)	D7-D9

3. Discussion

3.1 NSAIDs

Nonsteroidal anti-inflammatory drugs (NSAIDs) are a class of medications used for analgesic and anti-inflammatory benefits. NSAIDs can induce several different forms of kidney injury including hemodynamically mediated acute kidney injury (AKI); electrolyte and acid-base disorders; acute interstitial nephritis (AIN), which may be accompanied by the nephrotic syndrome; and papillary necrosis. Non-selective NSAIDs are inhibitors of both COX-1 and COX-2 pathways. COX-1 is responsible for maintenance of renal blood flow. Activation of COX-1 synthesizes the prostaglandins which cause vasodilation of afferent arterioles and it improves renal perfusion. Due to continuous administration of non-selective NSAIDs, COX-1 pathway gets inhibited which reduces renal perfusion and ultimately it leads to interstitial nephritis and renal shutdown. Because of the large number of patients that take NSAIDs (estimates of more than 70 million prescriptions and 30 billion over-the-counter doses annually), this translates to upwards of 2.5 million patients experiencing a nephrotoxic event annually. AKI can occur with any class of traditional, nonselective NSAID or cyclooxygenase-2 (COX-2) specific NSAIDs [4].

3.2 Aminoglycosides (AMG)

AMG are prototype drugs having nephrotoxicity as major side effect. Number of patients developing nephrotoxicity increases with duration of therapy reaching 50% with 14 days 2 or more of therapy.

Aminoglycosides preferentially affect the proximal tubular cells. These agents are freely filtered by the glomeruli and quickly taken up by the proximal tubular epithelial cells, where they are incorporated into lysosomes after first interacting with phospholipids on the brush border membranes. They exert their main toxic effect within the tubular cell by altering phospholipid metabolism. In addition to their direct effect on cells, aminoglycosides cause renal vasoconstriction.

The 2 critical factors in the development of acute kidney injury (AKI) secondary to aminoglycoside nephrotoxicity are dosing and duration of therapy. Aminoglycoside uptake by the tubules is a saturable phenomenon, so uptake is limited after a single dose. Thus, a single daily large dose is preferable to 3 doses per day. One dose per day presumably causes less accumulation in the tubular cells once the saturation point is reached [5].

4. Acknowledgment

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5. Conflict of Interests

There is no conflicts of interest.

6. Conclusion

As there is large number of drugs available today judicious use of such drugs is required to prevent untoward side effects especially on such vital organ as kidney. Identifying high-risk patients and quick recognition of drug-induced injury-related syndrome with prompt cessation of the offending drug are the key to managing such a case before the injury causes permanent damage to the renal tissue.

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