

## Acute Kidney Injury Sequel to Aspirin Induced Intestinal Obstruction

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**Abstract:** Self medication is common, though the harmful effects are usually not overt. In some cases the effects are many, serious and could be quite fatal. NSAIDs are drugs that are usually abused and their use, if contrary to instructions results in conditions that if care is not taken would not only result in hospital attendance but also require some invasive or surgical resolution. A classical example is the indiscriminate use of aspirin which would not only inflame the bowels but will also bring about bowel obstruction. With the adoption of a careful clinical appraisal and a choice of conservative treatment, an intestinal obstruction could be resolved thereby minimizing the possibility of re-admission as cases of abdominal surgical intervention are associated with increased risk of relapse.

**Keywords:** Self medication, aspirin, bowel obstruction.

### INTRODUCTION

A 34 year old African male presented to clinic with a distended and painful abdomen. Proper examination and history taking showed that he had had episodes of malaise within the penultimate week. He had to resort to over the counter self medication, taking aspirin in abnormal doses. Five days to presentation he had experienced a severe abdominal discomfort, constipation but no nausea or vomiting. The outcome was a cessation of bowel movement. Two days to presentation at clinic, he was given a concoction of herbal laxatives at home, this proved ineffective.

### PRELIMINARY EXAMINATIONS

On admission, blood was collected for widely agglutination test and a full blood count. All FBC parameter were well within the reference range. Ultrasound scan of the abdominal cavity revealed an intestinal obstruction but no perforation. It was decided that pending further abdominal occurrence, no invasive procedures were to be performed on the patient, the option being that the obstruction classified as being simple needs a conservative treatment.

### INITIAL TREATMENT AND COMPLICATIONS

The patient was placed on total oral fluid and diet restrictions. While intravenous dextrose saline drip with standard amount of supplements were administered. The patient was closely monitored for possible bowel movement and urine output was measured. It was thereafter noted that approximately

180mls of urine was passed through a period when 1500mls of intravenous fluid was given. Oliguria had set in; acute kidney injury was indicted.

### RESOLUTION, FURTHER LABORATORY EXAMINATIONS AND TREATMENT

On the fifth day of admission an abdominal sensation overwhelmed the patient; Bowel movement had definitely resumed. : feaces was unformed and foul smelling. Blood was collected for the determination of Creatinine, Urea and, HCO<sub>3</sub>, Sodium and Potassium were 173µmol/L (50-132 µmol/L), 6.9mmol/L(2.2-5.8mmol/L), 18mmol/L (20-30mmol/L), 129mmol/L (120-140mmol/L), 4.4mmol/L(3-5mmol/L); Acute kidney injury was confirmed. Intravenous infusion was withdrawn while glucose in warm water was administered orally at 200mls/24hours until the 20<sup>th</sup> day when the patient started taking light cereals, vegetables and fruits, a period during which urine output was noted to be increasing progressively. Blood collected on the 25<sup>th</sup> day for determination of plasma creatinine and electrolytes showed improvement: creatinine-148 µmol/L, bicarbonate-21mmol/L, Potassium-3.8mmol/L, Sodium-134mmol/L. The patient, though emaciated was discharged on the 28<sup>th</sup> day with the physicians charge that he returns for clinical appraisal 5day intervals.

### DISCUSSION

Intestinal obstruction is an obstruction of the intestines, preventing the normal movement of chyme and the products of digestion. It can occur at any level

distal to the duodenum of the small intestine and is a medical emergency. Surgical procedures are performed on occasion however, in life-threatening cases of intestinal obstruction, such as when the root cause is a fully lodged foreign object or malignant tumor. Though, in the management of small bowel obstructions it was once said, "never let the sun rise or set on small-bowel obstruction"[1] because a significant portion (Approx. 5.5%) [1] of small bowel obstructions could have fatal outcomes if treatment is delayed, It is the discretion of the physician to determine whether a case is simple or will require a surgical emergency. This is made possible by improvements in patient care and state of the art imaging techniques. This fact is definitely the rationale behind the supervising physician's reluctance to indicate that the patient should undergo more invasive

procedures.

Acute kidney injury this could be defined as a kidney dysfunction of acute onset but sufficient enough to bring about a disruption of renal homeostasis [2]. It is diagnosed using the estimation of plasma creatinine levels but according to the Kidney Disease: Improving Global Outcomes (KDIGO) work group. The diagnosis and grading of acute kidney injury is based on either plasma creatinine level or the estimation of urine output. In this present research, the use of plasma creatinine level is the assessment method of choice, the assessment based on urine output in these patients being somehow severe and cumbersome[3]. The tabular presentation below shows the peculiarities of the KDIGO RIFLE classification method [4].

**Tabular presentation of the KDIGO based method in the staging of AKI, using the RIFLE criteria of Bellomo et al.; [4]**

Stage	Plasma creatinine	urine output
Risk	1.5-fold increase in serum creatinine level or >25% decrease in GFR	<0.5 ml/kg per h for 6 h
Injury	Twofold increase in serum creatinine level or >50% decrease in GFR	<0.5 ml/kg per h for 12 h
Failure	Threefold increase in serum creatinine level or 75% decrease in GFR or increase in serum creatinine level to $\geq 4$ mg/dl ( $\geq 354$ $\mu$ mol/l) with an acute increase $\geq 0.5$ mg/dl ( $\geq 44$ $\mu$ mol/l)	<0.3 mg/kg per h for 24 h Anuria for 12 h
Loss	Persistent AKI with complete loss of renal function (>4 weeks)	N/A
End-stage renal disease	Persistent AKI with complete loss of renal function (>3 months)	N/A

The first plasma creatinine level of the patient in this study was found to be well above the upper limit of the reference range, all other parameters being consistent with acidosis- an important hallmark of AKI. If the stage of AKI in this case was to be graded, stage 2(failure) would be the most appropriate, using the KDIGO based criteria as set in the work of Odewusi and Oyedeji (2015) [5] as a standard. The causes of AKI are numerous but the most possible cause in this study is an hypovolaemia sequel to bowel obstruction. The rationale could not be farfetched, the patient had resorted to herbal remedies in the five days prior to admission, but there was no medium of rehydration since no drip was given and the site of obstruction was distal to the colon where absorption of water occurs, hence hypovolaemia. In fact this is the reason why hydration is highly indicated in patients presenting with intestinal obstruction. It is noted that this case of obstruction as most patients [6], improved with conservative care within the week, usually staying in the hospital a few more days until they are able to eat and walk [6]. However, the observed renal complication warranted further stay within the precincts of the hospital.

**Self medication and resort to herbal remedies**

Humans are exposed to a variety of potential nephrotoxic substances on a rather frequent basis [7]. Several therapeutic agents have known nephrotoxic potential; classic examples include anti-microbial agents, chemotherapeutic agents, analgesics, and immunosuppressive agents [8-15]. Some of these drugs are prescription drugs while the majority is over-the-counter. A similar point is that Nigerians believe in the efficacy of alternative/ complimentary therapy, herbal concoctions, infusions and mixes, maybe because it is most often relatively cheap, and more accessible. Moreover, Africans are faced by the menace of exorbitant cost of "western drugs". Though it is not the duty of the investigators in this research to doubt the efficacy of alternative therapy, we stress that more harm than good may be done if wrong diagnoses are made, or if the treatment is ineffective. A worst case scenario would be a situation in which one or more ingredients of the therapy are potential nephrotoxic phytochemicals. Similar circumstances are cases of product preservation and adulteration or substitution of a key ingredient [16, 17] most probably to maximize profits. An occurrence that cannot be forgotten so soon is the case where Infant formula was being adulterated

with melamine, a substance highly capable of causing injury to the kidney [18].

## CONCLUSION

With the adoption of a careful clinical appraisal and a choice of conservative treatment, an intestinal obstruction resolved thereby minimizing the possibility of re-admission as cases of abdominal surgical intervention are associated with increased risk of relapse. Statistics from U.S. healthcare reporting a 18.1% re-admittance rate within 30 days for patients who undergo SBO surgery [19]. Majority of patients also form adhesions after major abdominal surgery [19]. Common consequences of these adhesions include small-bowel obstruction, chronic abdominal pain, pelvic pain, and infertility [19]. An episode of AKI was also conservatively resolved without the use of diuretics the adverse effect of which on the electrolytes and acid base balance [20, 21] would have disrupted the physicians grasp on the patient's condition.

## REFERENCES

1. Maglinte DD, FM Kelvin, MG Rowe MG, GN Bender GN, DM Rouch; "Small-bowel obstruction: optimizing radiologic investigation and nonsurgical management". *Radiology*, 2001; 218(1): 39–46.
2. Whitby L G Smith AF, Beckett CJ; Renal disease in: Lecture notes on clinical chemistry, 4th edition, and Blackwill scientific publication, 1989; 150-174.
3. Leung KC, Tonelli M, James MT; Chronic kidney disease following acute kidney injury—risk and outcomes *Nature Reviews Nephrology* 2013; 9:77-85.
4. Bellomo R, Ronco C, Kellum JA, Mehta RL, Alevsky P; "Acute renal failure - definition outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group" *.Crit.Care*.2004;8 (4): R204–12.
5. Odewusi OO, Oyedeji SO; A study on the stage of presentation of acute kidney injuries to Clinics in western Nigeria. *African Journal of Cellular Pathology* 2015; 4:29-33.
6. The Eastern Association for the Surgery of Trauma (2010). Small Bowel Obstruction.
7. American Society of Nephrology; Renal Vulnerability to Drug Toxicity 2009; 4(7): 1275-1283.
8. Schetz M, Dasta J, Goldstein S, Golper T; Drug-induced acute kidney injury. *Curr.Opin Crit Care* 2005; 11:555– 565.
9. Elseviers MM, DeBroe ME; Analgesic nephropathy, Is it caused by multi-analgesic abuse or single substance use? *Drug Saf*. 1999; 20:15– 24.
10. Izzedine H, Launay-Vacher V, Deray G; Antiviral drug-induced nephrotoxicity. *AmJ Kidney Dis* 2005; 45:804– 817.
11. Kintzel PE; Anticancer drug-induced kidney disorders. *Drug Saf* 2001; 24:19–38.
12. Perazella MA; Drug-induced nephropathy: An update *Expert Opin. Drug Saf*. 2005; 4: 689–706.
13. Gambaro G, Perazella MA; Adverse renal effects of anti-inflammatory agents: Evaluation of selective and nonselective cyclooxygenase inhibitors. *J InternMed* 2003; 253: 643– 652.
14. Rougier F, Ducher M, Maurin M, Corvaisier S, Claude D, Jeliffe R, *et al.*; minoglycoside dosages and nephrotoxicity. *Clin Pharmacokinetics*. 2003; 42:493–500.
15. Lamieire NH, Flombaum CD, Moreau D, Ronco C; Acute renal failure in cancer patients. *Ann of Med*. 2005; 37: 13– 25.
16. Isnard B C, Deray G, Baumelou A, Le Quintrec M, Vanherweghem JL; Herbs and the kidney. *Am J Kidney Diss*. 2004; 44 (1):1– 11.
17. Blowey DL; Nephrotoxicity of over-the-counter analgesics, natural medicines, and illicit drugs. *Adolesc Med Clin*.2005; 16: 31–43.
18. Wang JJ, Chen PC, Hwang KC; Melamine and nephrolithiasis in children in Taiwan. *N Engl J Med* 2009; 360 (11):1157– 1158.
19. Agency for Healthcare Research and Quality "Readmissions to U.S. Hospitals by Procedure" (PDF). April 2013. Assessed August 4, 2015.
20. Liakakos TN, Thomakos PM Fine, Dervenis C, RL Young; "Peritoneal Adhesions: Etiology, Pathophysiology, and Clinical Significance". *Dig Surgery*, 2001.
21. Heusel JW, Siggard-Anderson O, Scott MG; ;Physiology and disorders of water,electrolyte, and acid-base metabolism ;In Tietz textbook of clinical chemistry.WB saunders,Philadelphia,Pennsylvania 1999;1204-1270
22. Whitby LG, Smith AF, Beckett CJ; Disturbances of Water, sodium and Potassium balance in: Lecture notes on clinical chemistry, 4<sup>th</sup> edition, Blackwill scientific publication. 1989; 38-62.