

International Journal of Current Research in Medical Sciences

ISSN: 2454-5716 (A Peer Reviewed, Indexed and Open Access Journal) www.ijcrims.com



Case Report

Volume 8, Issue 1 -2022

DOI: http://dx.doi.org/10.22192/ijcrms.2022.08.01.004

Case Report of NSAIDs induce Peripheral Edema

T. Sasi Kumar*, B. Sathya Narayana Reddy*, S. Surya Kiran, K. Tejaswini, S. Mohammed Althaf.

Annamacharya College of Pharmacy, Thallapaka, Rajampet, Andhra Pradesh - 516126 E-mail: *tsasikumar.tiruttani369@gmail.com*

Abstract

NSAIDs are the drugs that block the production of certain body chemicals which causes inflammation, these drugs reduce pain, fever, blood clots and also causes side effects like edema, cardiac problem, respiratory, GI conditions. Inhibition of PGE2 causes sodium and fluid retention in the patients causes peripheral edema.

Keywords: NSAIDs, Peripheral Edema, Side Effects, and Adverse Reactions.

Introduction

Non-steroidal anti-inflammatory drugs (NSAIDs), were prescribed by physicians as analgesic, antipyretic and anti-inflammatory agents which is the most used drug of class around worldwide. Most of the side effects are due to COX-1 inhibition, because of its action in several systems including cell cleansing. In kidney mostly act on glomerular filtration rate maintenances along with renal problems some other complications like gastrointestinal, cirrhosis, cardiovascular, and platelet (thrombotic events) changes requires prescription.¹ Effect of renal caution in the inhibition homeostasis caused by of cyclooxygenase-dependent generation of prostaglandins (PG) is the primary mechanism. Each nephron component COX isoform expresses at different levels. More PG synthesis (including PGE2 and PGI2) involves NSAIDs associated with edema. 2-5% of NSAIDs users were uncommon in the occurrence of Peripheral edema.² Most of the NSAIDs and COX-2 inhibitors are apparently causing fluid retention and effects on blood pressure. These drugs show the impact of musculoskeletal conditions on functional impairment outstrips that result from cardiovascular, respiratory, renal, gastrointestinal, psychiatric, and neurologic conditions in the United States. The most common side effects of NSAIDs were fluid retention and edema results of prostaglandin E2 production inhibition.³

Hayashi et al. showed that NSAID users have a high risk of kidney injury, renal risks from NSAIDs could be reversible. NSAIDs are considered safe, the therapeutic dose also causes the risk of loss of renal function, high dose of NSAIDs shows the greater risk of AKI in patients.^{4,5} Abnormal accumulation of fluid in body interstitial space is known as Edema. nephrotic and nephritic syndromes are the leading cause of renal edema.⁶

Case Report

A male patient was admitted to the male medicine ward in a government general hospital with the chief complaints of shortness of breath for 1 week and altered sensorium for 2 days. History of NSAIDs drug abuse present and known case of CKD. The patient was a known smoker and not a known case of Diabetes, Hypertension, Asthma. The patient was diagnosed with Altered sensorium with AKI with Sepsis with



Figure :1 Shows Edematous in leg

Outcomes and follow up:

After suspecting the conditions, patients were advised Antibiotics to control infection rate (sepsis, cellulitis) and provide symptomatic treatment. After 2 weeks of treatment, the patient showed significant improvement in his condition and get discharged.

Discussion

NSAIDs promote sodium and water retention is explained by a reduction in which prostaglandin-induced, both renal chloride reabsorption and the action of the antidiuretic hormone are inhibited.⁷ NSAIDs inhibits the cyclooxygenase culminates in a decrease in total perfusion and medullary influx deviation (decrease GFR) and water and sodium retention results in renal vasoconstriction and medullary ischemic causes acute kidney injury, chronic effects can result in chronic kidney injury which leads to fluid retention causes edematic conditions.^{1,8} Antirheumatic drug, calcineurin inhibitors, an antitumor drug, and NSAIDs are the hypoglycemia with right lower limb cellulitis. Lab reports for that patient shows Hemoglobin-11.6g/dl, total count- 8000 cell/cum, Neutrophils 88%, Lymphocytes 10%, Eosinophils 1%, Monocytes 1%, Platelets- 1.1 lacks/cum. B. Urea – 39mg/dl, S. Creatinine – 0.8mg/dl.CT Scan Brain – Mild bilateral periventricular ischemic changes. The patient got the treatment with Inj. Cefperazone Salbactum (1.5g), Inj. Pantop (40mg), Inj. Lasix (40mg), 5% dextrose infusion and 25% dextrose infusion. Inj. Optineuron.





drugs induced by renal failure. High incidence of acute kidney injury in the nephrotoxic potential of dual or triple combinations of (NSAIDs + reninangiotensin system inhibitors or NSAIDs + diuretics). Heart attack and stroke have the adverse effects of NSAIDs. Other adverse effects include stomach pain, constipation, diarrhea, gas, heartburn, nausea, vomiting, and dizziness.⁹ PGE2 inhibition causes sodium retention, which causes weight gain, peripheral edema, Blood pressure, or rarely, congestive heart failure.¹⁰ The treatment includes Antibiotics for the control of infection to that patient.

Conclusion

Edema is a condition in which fluid accumulation in body space is developed by the infection or drugs like NSAIDs. The proper usage of NSAIDs with caution and the proper indication of prescription helps in prevention of edema in patients. This knowledge will help in identify and reporting these types of conditions.

References

- 1. Lucas GNC, Leitão ACC, Alencar RL, Xavier RMF, Daher EF, Silva Junior GBD. Pathophysiological aspects of nephropathy caused by non-steroidal anti-inflammatory Bras Nefrol. 2019 drugs. J Jan-Mar;41(1):124-130. doi: 10.1590/2175-8239-JBN-2018-0107. Epub 2018 Sep 21. PMID: 30281062; PMCID: PMC6534025.
- Largeau B, Cracowski JL, Lengellé C, Sautenet B, Jonville-Béra AP. Drug-induced peripheral oedema: An aetiology-based review. Br J Clin Pharmacol. 2021 Aug;87(8):3043-3055. doi: 10.1111/bcp.14752. Epub 2021 Feb 20. PMID: 33506982.
- 3. Aneja and Michael Adverse cardiovascular effects of NSAIDs: driven by blood pressure, or edema?: Therapeutic Advances in Cardiovascular Disease (2008) 2(1) 53– 66 DOI: 10.1177/ 1753944707088184.
- 4. Dixit , Thuy, Kirschner et al. Significant Acute Kidney Injury Due to Non-steroidal Antiinflammatory Drugs: Inpatient Settin: Pharmaceuticals 2010, 3, 1279-1285;doi:10.3390/ph3041279.
- 5. Zhang, Peter, Bell et al. Non-steroidal antiinflammatory drug induced acute kidney

injury in the community dwelling general population and people with chronic kidney disease: systematic review and metaanalysis: BMC Nephrology (2017) 18:256 DOI 10.1186/s12882-017-0673-8

- Bobkova, Chebotareva, Lydiya et al. Edema in renal diseases – current view on pathogenesis: Nephrology @ Point of Care 2016; 2(1): e47-e55 DOI: 10.5301/pocj.5000204.
- Knights, Arduino, Miners et al. Nonselective nonsteroidal anti-inflammatory drugs and cardiovascular events: is aldosterone the silent partner in crime? Br J Clin Pharmacol 61:6 738–740
- 8. Vaughan Keeley and Neil Piller, Edemacausing medications Take note of your medications and talk with your doctor: ask the expert: 2015.
- Hayashi K, Miki K, Kajiyama H, Ikemoto T and Yukioka M (2021) Impact of Nonsteroidal Anti-inflammatory Drug Administration for 12 Months on Renal Function. Front. Pain Res. 2:644391. doi: 10.3389/fpain.2021.644391.
- Craig Brater. Renal Effects of Cyclooxygyenase-2-Selective Inhibitors: Journal of Pain and Symptom Management: Vol. 23 No. 4S April 2002.

Access this Article in Online	
	Website:
	www.ijcrims.com
	Subject:
Quick Response Code	

How to cite this article:

T. Sasi Kumar, B. Sathya Narayana Reddy, S. Surya Kiran, K. Tejaswini, S. Mohammed Althaf. (2022). Case Report of NSAIDs induce Peripheral Edema. Int. J. Curr. Res. Med. Sci. 8(1): 35-37. DOI: http://dx.doi.org/10.22192/ijcrms.2022.08.01.004