ORIGINAL RESEARCH PAPER
Volume-9 | Issue-1 | January-2020 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

NSAID INDUCED NEPHROPATHY WITH PERIPHERAL NEUROPATHY AND ENCEPHALOPATHY IN AN HEPATITIS C PATIENT : A CASE REPORT

Nephrology

Monika Kapoor*
Department Of Pharmacy Practice, I.S.F College Of Pharmacy, Moga, Punjab, India.
*Corresponding Author

ABSTRACT

The long term usage of selective and non-selective cyclooxygenase-2 (COX-2) specific Non-Steroidal anti-inflammatory drugs also known as NSAID can lead to the progress of complications like chronic kidney disease and peripheral neuropathy that is unquestionable. A lot of cases have been observed to date that occurred due to long term use of analgesics/NSAID. However, the key role of a single analgesic drug used for a particular duration of time that lead to nephropathy was challenging. This report is to spotlight the case of NSAID induced nephropathy in an 80 years old male patient who was Hepatitis C positive and was admitted to the medicine department due to acute febrile illness and breathlessness.

KEYWORDS

Nsaid Induced Nephrotoxicity, Encephalopathy, Peripheral Neuropathy, HCV, Ultracet(tramadol Hcl/acetaminophen)

INTRODUCTION

NSAIDs are one of the most well-known wrongdoers in the development of nephrotoxicity in high-risk patients. Both COX-1 and COX-2 pathways are inhibited by the Non-selective NSAIDs. COX-1 is known to maintain the blood flow towards renal tubules. When COX-1 gets activated, it synthesizes the prostaglandins which cause vasodilation of afferent arterioles and improves renal perfusion. When these non-selective NSAIDs are administered for a long period of time they inhibit the COX-1 pathway and reduces renal perfusion and eventually cause renal arrest. [1-2] This may further lead to tubular necrosis and acute renal failure. NSAIDs also lead to interstitial nephritis and papillary necrosis, eventually resulting in chronic renal failure. [3] However, nephropathy is non-specific, slowly progressive chronic renal disease, usually asymptomatic in many patients and is later diagnosed. Generally, patients with NSAID induced nephropathy are presented with flank pain, hypertension, anemia, hematuria, atherosclerotic changes. Urine shows no nephrotic proteinuria, hematuria, and pyuria which is usually sterile. Kidneys are usually bilaterally shrunken with irregular contours and show papillary calcification. The diagnostic criteria for analgesic nephropathy are as follows: [4-8]

- Previous history of daily usage of the analgesic drug for more than five years.
- Renal imaging which shows small kidneys/bumpy kidneys/ papillary calcifications.
- Proteinuria less than 3 g/day.
- Sterile pyuria.

Any three parameters from the above-mentioned criteria and history of long term use of NSAID makes a strong diagnosis. In this case study, all of the above four parameters were observed which assures nephrotoxicity due to the use of NSAID. [9]

CASE REPORT

An 80-year-old male patient presented to the medicine department with chief complaints of breathlessness, increased frequency of urine and high-grade fever since the past 15 days and numbness in his feet and hands. The patient complained that his problem of breathlessness starts abruptly which restricts his day to day activity and fever is also progressive in nature and not associated with chills. He usually forgets things very quickly and felt lethargic. The patient has a previous history of anorexia, pyuria, and peripheral edema. There was no past history of kidney disease. The patient was suffering from knee pain for the past 5-6 years for which he consulted a local physician near his home. The prognosis was done to check for arthritis but it came out to be normal joint pain only. The physician advised him to take tablet Ultracet whenever pain occurs. Tablet Ultracet is an analgesic combination of Acetaminophen and tramadol. The patient used to get relief after taking the tablet. However, he developed a habit of taking the tablet whenever pain occurred. According to the patient, he got addicted to this and without being getting noticed he took the tablet for a continuous 5 years till now.

He was non diabetic and non-hypertensive and never took any herbal concoction. There was no family history of kidney disease, diabetes or hypertension. The patient was vegetarian by diet and did not ingest either alcohol or tobacco nor did he smoke.

On examination the patient was conscious, he had dyspnoea, was febrile, mildly dehydrated and pale. He was not able to walk properly on his own. The pulse rate was 82 beats per min, full volume and regular. There was no thickening of the arterial wall. The blood pressure was 110/70 mmHg. First and second heart sound was heard without any murmur. Random blood sugar was observed to be 129 mg/dl. The patient was showing asterixis – like movements due to encephalopathy.

The diagnosis revealed that the patient had nephrotoxicity with end-stage renal disease, peripheral neuropathy with encephalopathy and high-grade fever. Based on the diagnosis patient was admitted to the medicine male ward and the investigations were then carried out.

According to the Complete blood count (CBC) report the hemoglobin of the patient was 8.7 gm/dl and Total Leucocyte Count (TLC) came out to be 21400/cmm. The patient was also examined for 25 Hydroxy Vitamin D which was 9.1 ng/ml. Lipid profile was also examined which was pretty normal. He was investigated for viral markers in which the Anti-HCV Ab Rapid Test was reactive and the patient was diagnosed with hepatitis C. Arterial blood gas analysis (ABG) report showed partial pressure of oxygen to be 300mmHg. Three days later he was again examined for CBC and the TLC count was further reduced to 19800/cmm.

Renal function test analysis was made and urea 250 mg/dl, Creatinine 7.4 mg/dl, Uric acid 9.1 mg/dl were observed. The serum electrolytes were Calcium 4.5 mg/dl, phosphorus 6.3 mg/dl, sodium 133 mEq/L, potassium 3.4 mEq/L and chloride 100 mEq/L. Blood culture and sensitivity tests were done and no growth of aerobic pyogenic organisms was obtained after 48 hours of aerobic incubation at 37ºC. Ultrasound abdomen was done in which the renal scan (figure 1) showed right kidney normal in size, outline with increased echogenicity. Corticomedullary differentiation was maintained. A cortical cyst of 21x21mm was seen at the upper pole. The left kidney was also normal in size, outlined with increased echogenicity. A cortical cyst of 20x22mm was seen at the upper pole. No free fluid was observed in the peritoneal cavity. No upper retroperitoneal lymphadenopathy was seen. Drug therapy was then initiated which is given in table 1.

Table 1: Drug therapy administered to the patient on the day of admission

Drug	Dose	Frequency	Route
Inj. Pentocid(Pantoprazole)	40mg	BD	IV
Inj. Gluci(elemental calcium)	NS 100ml	OD	IV
Inj. Tazomac(Piperacillin+Tazobactom)	4.5 gm	BD	IV
Inj. Meconerv forte(Vitamin)	10mg	OD	P/O
Tab. Tryptomer(Amitriptyline)		TDS	

Cap. Pregator(Alpha lipoic acid + Folic Acid + Methylcobalamin + Pregabalin + Pyridoxine)	1 tablet	HS	P/o
Tab. Dytor(Torsemide)	20 mg	OD	P/o
Disogel syrup	2 tsp	TDS	P/o
Inj. Arachitol Nano	60000 IU	once weekly	IM

BD- Twice a day; **OD-** Once a day; **TID-** Thrice a day; **HS-** at bedtime
Three days later RFT was examined and urea 156 mg/dl, creatinine 5.0 mg/dl was observed. But, the patient was in a dyspnoeic state and saturation was low (<80%) and in bradycardia. Tablet tryptomer was stopped and paracip infusion 100 ml was advised when needed. Nebulization with asthalin and budecort was started. No significant changes were observed and the patient was then shifted to ICU and oxygen therapy was initiated.

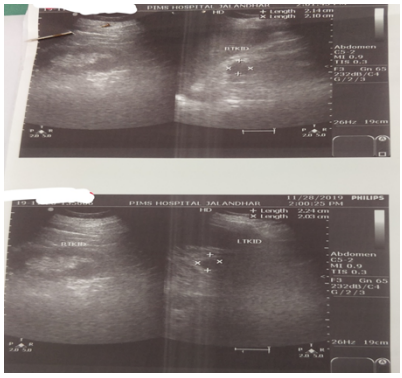

Figure 1: Ultrasound scan of the abdomen

Blood pressure was dropped to 80/50 mmHg and hence injection Norad (Noradrenaline) 2 amp in 100 ml (10microdrops/min) was started and Inj. Sodabicarb I/V 10 ml stat was given.

On the fourth day, vitals were stable Inj. Tazomac was stopped and Inj. Supime twice a day was initiated. Inj. Moxiflox (moxifloxacin) OD, Inj. Levipill (Levetiracetam) 100mg BD, Duphalac enema BD, Inj. Vitamin K OD, tab. Rifaximin 400mg BD and inj. Glucci IV TDS was further started. RT feed 200ml 4hrly and Normal saline 10mk with 2KCl was given. Urine output was observed daily and was 1200 ml/day. The rest of the treatment was being continued as per the initial treatment chart. Physiotherapy was being done daily.

On the fifth day of admission, his general condition was sick, B.P was 110/60 mmHg and pulse 84/min and dialysis was started. After 3 hours of dialysis, pulse was 92/min and B.P was 150/120 mmHg. On the next day, the patient was not recovering and still had the same condition. His prognosis was well explained to his attendants that the chances of recovery were rare. Tablet Dytor (torsemide) was stopped and Inj. Lasix 40mg twice daily was started. The attendants asked for discharge on request for shifting the patient to PGI, Chandigarh. I took a follow up of the case and called the attendants to enquire about the further health. Unfortunately, due to multiple organ failure the patient expired over there.

CONCLUSION

Early detection and cessation of NSAIDs stabilizes or ameliorates the renal function and in some cases countermand renal function impairment. Unfortunately, diagnosis in such cases is delayed due to elusive presentation. The patient was presented very late during his end-stage renal disease and was still using the drug before that. High-risk patients must be educated properly regarding the use of NSAIDs. In the case of nephrotoxicity, the infection must be treated and fluid and electrolyte balance must be maintained. Anemia should be corrected and renal replacement therapy must be initiated when indicated. OTC (over the counter) painkillers must be banned in order to reduce the prevalence of drug-induced nephropathy. For elderly patients, these drugs must be used with caution and therapy must be planned accordingly in order to avoid overusage.

ETHICAL DECLARATION

Informed consent was taken from the patient before the data collection stating that no identity of the patient will be disclosed.

CONFLICT OF INTEREST

Author declares that they have no conflicts of interest.

ABBREVIATION-

HCV- Hepatitis C positive NSAID- Non Steroidal anti-inflammatory Drugs COX- cyclo-oxygenase pathway

REFERENCES

1. Perazella MA, Eras J. Are selective COX-2 inhibitors nephrotoxic?. American Journal of Kidney Diseases. 2000 May 1;35(5):937-40.
2. Vanam R, Reddy PM. A Case Study of Drug Induced Acute Interstitial Nephritis with combined use of Beta-Lactams, NSAID's and Amikacin.
3. Ejaz P, Bhojani K, Joshi V. NSAIDS and kidney. J Assoc Physicians India. 2004;52:632–640
4. Okafor UH, Unuigbe EI, Onwuchekwa AC, Emem Chioma P. Case Report-Analgesic nephropathy as a cause of end stage renal disease in a 55 year old Nigerian. Nigerian journal of clinical practice. 2012;15(2):231-4.
5. Drukker W, Schwarz A, Vanherweghem JL. Analgesic nephropathy: an underestimated cause of end-stage renal disease.
6. Elseeviers MM, Bosteels V, Cambier P, De Paepe M, Godon JP, Lins R, et al. Diagnostic Criteria of analgesi nephrotoxicity in patients with end stage renal failure, results in Belgian study. Nephrol Dial Transplant 1992;7:479 86
7. Pintér I, Mátyus J, Czégány Z, Harsányi J, Homoki M, Kassai M, Kiss É, Kiss I, Ladányi E, Lőcsey L, Major L. Analgesic nephropathy in Hungary: the HANS study. Nephrology Dialysis Transplantation. 2004 Apr 1;19(4):840-3.
8. Waddington F, Naunton M, Thomas J. paracetamol and analgesic nephropathy: are you kidneying me?. International medical case reports journal. 2015;8:1.
9. Gooch K, Culleton BF, Manns BJ, Zhang J, Alfonso H, Tonelli M, Frank C, Klarenbach S, Hemmelgarn BR. NSAID use and progression of chronic kidney disease. The American journal of medicine. 2007 Mar 1;120(3):280-e1.