An 80-year-old male patient presented to the medicine department with chief complaints of breathlessness, increased frequency of urination and high-grade fever since the past 15 days and numbness in his feet. The patient complained that his problem of breathlessness started 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 anemia, 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:

- 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.

**CASE REPORT**

An 80-year-old male patient who was Hepatitis C positive and was admitted to the medicine department due to acute febrile illness and breathlessness. 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 with no murmur. Random blood sugar was observed to be 129 mg/dl. The patient was showing astrexis – 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 37°C. 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 cortic 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>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Frequency</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inj. Pentocid(Pantoprazole)</td>
<td>40mg</td>
<td>BD</td>
<td>IV</td>
</tr>
<tr>
<td>Inj. Gluc(elemental calcium)</td>
<td>NS 100m</td>
<td>OD</td>
<td>IV</td>
</tr>
<tr>
<td>Inj. Tazomac(Piperacillin+Tazobactom)</td>
<td>4.5 gm</td>
<td>BD</td>
<td>IV</td>
</tr>
<tr>
<td>Inj. Meconev forte(Vitamin)</td>
<td>10mg</td>
<td>OD</td>
<td>P/O</td>
</tr>
<tr>
<td>Tab. Tryptomer(Amitriptyline)</td>
<td>TDS</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
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.

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 oversusage.

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

Figure 1: Ultrasound scan of the abdomen