ACETAMINOPHEN INDUCED ANALGESIC NEPHROPATHY: A CASE REPORT

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ABSTRACT

Analgesics are the medications act in various ways on the peripheral and central nervous system used to achieve analgesia (relief from pain). It can cause two different forms of kidney damage acute renal failure and analgesic nephropathy. Analgesic nephropathy is a chronic condition can result from use of painkillers every day for several years that gradually leads to irreversible kidney failure and the permanent need for dialysis or a kidney transplant to restore kidney function.

KEYWORDS: Analgesics, Analgesic nephropathy, Analgesia.

INTRODUCTION

Analgesic nephropathy is a chronic disease leads to irreversible damage to kidney and which results in frequent dialysis or a renal transplantation to restore kidney function.[1] It is caused by prolonged use of analgesics, especially over the counter (OTC) medications like nonsteroidal anti-inflammatory drugs (NSAIDs). About 6 or more pills per day for three years increases the risk of this problem.[2] Researchers estimate that 4 out of 100,000 people will develop analgesic nephropathy. It is most common in women over 30 years. Daily users of analgesics had significantly more renal disease than infrequent users.[3]

An analgesic is any medicine intended to relieve pain. Over-the-counter analgesics are the painkillers available without a prescription which include aspirin, acetaminophen, ibuprofen and Naproxen sodium etc.[4] These drugs present no danger for most people when taken in the recommended dosage. But some conditions make taking even these common painkillers dangerous for the kidneys. Also taking one of these drugs regularly over a long period of time may increase the risk for kidney problems.[4,5] Most drugs that can cause kidney damage are excreted only through the kidneys.[1]
Analgesic nephropathy involves damage to one or both kidneys caused by over-exposure to mixtures of medications, especially over-the-counter pain remedies (analgesics). This frequently occurs as a result of self-medicating, often for some type of chronic pain.[6]

**Risk factors include**

- Use of OTC analgesics containing more than one active ingredient.
- Chronic headaches, painful menstrual periods, backache, or musculoskeletal pain.
- Emotional or behavioral changes.
- History of dependent behaviors including smoking, alcoholism, and excessive use of tranquilizers.[7]

A physical examination may show signs of interstitial nephritis or kidney failure, blood pressure may be high and abnormal heart or lung sounds when listening to the chest with a stethoscope. There may be signs of premature skin aging, swelling, especially in the lower legs. Lab tests may show red or white blood cells in the urine, with or without signs of infection. There may be small amounts of protein in the urine.[8]

**CASE REPORT**

A 43 years female patient was admitted in the female Medicine department of Basaveshwara Medical College Hospital & research centre, Chitradurga on 4th September 2015. The chief complaints were swelling of both lower limbs, puffiness of the face, swelling in hands since 20 days. The patient was a chronic user of painkillers since 20 years for even a minute pain. On general examination the patient was conscious and coherent and the physical appearance was looking weak and her vitals were as follows BP-90/70 mm of Hg, PR-56bpm, CVS-S1,S2+,RS-30cpm, CNS- no abnormality present, P/A- distension+. The Laboratory examination shows BUN-30mg/dl, S.Cr-1.8mg/dl, Na+-136mmol/l, CL-106mmol/l, K+-4.4mmol/l, T3-0.91ng/ml, T.bilirubin-0.60mg/dl, D.bilirubin-0.30mg/dl, SGOT-11 IU/L, SGPT-22IU/L, T.Pro-6.5 g/dl, Alkaline phosphatase- 45IU/L, Sr.albumin-4.1mg/dl, Hb-12.2g/dl, RBC-4.56million cells/cu.mm,WBC-8300cells /cu.mm, N-48%, L-44%, E-05%, M-03%, Platelet-3.36lacs/ml, ESR-15mm/hr, sugar albumin-3-4, WBC- 1-2. Based on the subjective and objective evaluation patient have experienced analgesic nephropathy due to the prolonged usage of Acetaminophen (NSAIDs).Clinical evaluation was done and patient was treated symptomatically with parenteral diuretics (lasix 40mg iv bid), parenteral Proton pump inhibitor (pantaprazole 40mg iv bid), parenteral ceftriaxone 1gram IV bid, parenteral dobutamine 2amp IV OD. On day 2, BP-90/60mmHg, PR-70bpm and swelling of lower
limbs, puffiness of face and swelling of hands did not subsided but on day 3, BP-80/60mmHg, PR-72bpm, and the patient condition got improved. On day 4, BP-90/70 mmHg, PR-74 bpm & there is no fresh complaints. On day 5 the patient got discharge. After collecting the past and current history from the patient, causality assessment of ADR was done by using Naranjo’s scale and severity assessment using Hartwig’s & Seigels scale.

Causality assessment of suspected drug to cause ADR- ACETAMINOPHEN

Naranjo Causality Assessment Scale.

<table>
<thead>
<tr>
<th>Question</th>
<th>Yes</th>
<th>No</th>
<th>Don’t know</th>
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<tbody>
<tr>
<td>1 Are there previous conclusion reports on this reaction?</td>
<td>+1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2 Did the adverse event appear after the suspect drug was administered?</td>
<td>+2</td>
<td>-1</td>
<td>0</td>
</tr>
<tr>
<td>3 Did the AR improve when the drug was discontinued or a specific antagonist was administered?</td>
<td>+1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4 Did the AR reappear when drug was readministered?</td>
<td>+2</td>
<td>-1</td>
<td>0</td>
</tr>
<tr>
<td>5 Are there alternate causes [other than the drug] that could solely have caused the reaction?</td>
<td>-1</td>
<td>+2</td>
<td>0</td>
</tr>
<tr>
<td>6 Did the reaction reappear when a placebo was given?</td>
<td>-1</td>
<td>+1</td>
<td>0</td>
</tr>
<tr>
<td>7 Was the drug detected in the blood [or other fluids] in a concentration known to be toxic?</td>
<td>+1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>8 Was the reaction more severe when the dose was increased, or less severe when the dose was decreased?</td>
<td>+1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>9 Did the patient have a similar reaction to the same or similar drugs in any previous exposure?</td>
<td>+1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>10 Was the adverse event confirmed by objective evidence?</td>
<td>+1</td>
<td>0</td>
<td>0</td>
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Scoring • > 9 = definite ADR • 5-8 = probable ADR • 1-4 = possible ADR • 0 = doubtful ADR

CONCLUSION

Analgesic nephropathy (AN) is chronic a tubule-interstitial nephritis caused by cumulative lifetime use of large amounts (eg, ≥ 2 kg) of certain analgesics typically with aspirin, acetaminophen, codeine, or caffeine. Analgesic nephropathy is a slowly progressive renal disease, characterized by renal papillary necrosis.[8] Clinical evidence linking high consumption of analgesic preparations with analgesic nephropathy is overwhelming. Most patients who admit to over-consuming analgesics have taken preparation containing more than one compound.[7] Effective prevention of analgesic nephropathy consists of the prohibition of over-the-counter sales of preparation containing at least 2 analgesics associated with caffeine and/or codeine.[4]
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REFERENCE