INTRODUCTION
Non-steroidal anti-inflammatory drugs (NSAIDs) are widely prescribed drugs because of their strong analgesic, anti-inflammatory and antipyretic properties. Their main limitation is gastrointestinal (GI) tract toxicity.\(^1\) The adverse effects upon upper GI tract are well characterized. There are also studies that show toxicity of NSAIDs on large bowel for example they can lead to formation of colonic strictures, colitis resembling inflammatory bowel disease (IBD), or complicated diverticular disease (i.e. perforation or abscess).\(^2-4\) To the best of our knowledge, there are only two published case reports to-date that show large bowel perforation attributable to NSAIDs use only.\(^5,6\) In one of these case reports, formulation (plain, slow release or enteric coated) of NSAIDs used is not mentioned.\(^5\)

We present here a case report of colonic perforation after short term use of slow release diclofenac.

CASE REPORT
A 22 years old male presented in Emergency Room with abdominal pain, constipation and vomiting for 2 days. He had history of mumps parotitis 10 days back for which he had taken slow release diclofenac (2 x 100 mg), esomeprazol (2 x 40 mg) and cefixime (3 x 400 mg) for 6 days, after which he started having lower abdominal pain, increasing in intensity with time and became generalized abdominal pain in one day. He changed his doctor and was advised oral ciprofloxacin (2 x 500 mg), paracetamol (3 x 500 mg), and iron + B complex preparations. He took these medications for 2 days but his symptoms worsened over time. He was referred to a tertiary care hospital. On presentation, he had symptoms and signs of peritonitis with temperature of 101°F, blood pressure of 110/80 mmHg, pulse rate of 118/minute and respiratory rate of 22/minute. He had no other co-existing disease. There was no history of any allergy and interventional procedure. Digital rectal examination (DRE) findings were normal. Laboratory investigations revealed normal haemoglobin and WBC count. Erect abdominal radiograph showed free air under right hemi-diaphragm. Emergency exploratory laparotomy was done with diagnosis of gut perforation.

There were about 2 liters of gut contents in the abdominal cavity with whole gut stuck in soft tissue adhesions. Stomach and duodenum were normal on exploration. There was a 1 x 1.5 cm perforation at lower end of sigmoid colon. There was no granulomatous tissue present, no evidence of diverticulosis. Biopsy was taken and primary repair of perforation done with proximal diversion loop colostomy.

Patient received intravenous ceftriaxone (2 x 1000 mg) and metronidazole (3 x 500 mg) postoperatively. Histopathological examination was reported as non-specific acute and chronic inflammation with no evidence of granuloma formation or malignancy.

On 4th postoperative day, patient again developed abdominal pain and signs of peritonitis with gut contents seeping through previous laparotomy incision. At this time he was running fever of 102°F, blood pressure 120/80 mmHg, pulse 122/minute and respiratory rate of 22/minute. Re-exploration of the abdomen was done. There was about one liter of faecal matter upon opening the abdomen, flimsy adhesions of small gut and there was a 1 x 1 cm perforation in the mid of transverse colon. Left hemicolectomy was done with end transverse colostomy. Operative specimen was sent for histopathology. This time patient received intravenous

**ABSTRACT**
Upper gastrointestinal (GI) toxicity of non-steroidal anti-inflammatory drugs (NSAIDs) is well characterized. There is also documented data regarding their adverse effects on lower GI tract, like colonic strictures, inflammatory bowel disease and complications of diverticular disease in the form of abscess or perforation. But there are only two case reports published previously that show colonic perforation due to use of NSAIDs solely. We present here a case of colonic perforation induced by short-term use of slow release diclofenac in a young man. Colonic perforation should be considered as the possible diagnosis in patients with acute abdomen and NSAIDs to be one of the differentials if other possibilities are ruled out.

**Key words:** Non-steroidal anti-inflammatory agents (NSAIDs). Diclofenac. Colon perforation.

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### Colonic Perforation Induced by Short Term Use of Slow Release Diclofenac

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**CASE REPORT**

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Imipenem (3 x 500 mg) and metronidazole (3 x 500 mg). Postoperative recovery was smooth. Histopathological examination report did not show any granulomatous process or evidence of malignancy. In an attempt to establish the aetiology of perforation, request was sent to histopathologist to look specifically for any findings consistent with inflammatory bowel disease and the report again did not show any pathology. Serological test for HBV and HCV by ELISA showed negative results. Patient was discharged on 13th postoperative day.

**DISCUSSION**

Non-steroidal anti-inflammatory drugs are widely used for decades because of their strong analgesic, anti-pyretic and anti-inflammatory properties. The main factor that limits their use is gastrointestinal (GI) toxicity for example mucosal inflammation, erosions, ulcers, bleeding, anaemia and strictures. Incidence and risk of NSAIDs associated upper GI tract injury and complications are well characterized. There are a number of studies that suggest that NSAIDs may cause lower GI tract injury and complications as well. One of these case reports did not show large bowel perforation that is attributable to NSAIDs associated upper GI tract injury and complications. These studies show that lower GI complications are more common in patients taking enteric coated (EC) or slow release (SR) formulations of the drug and less in those using selective cyclo-oxygenase (COX-2) inhibitors. Amongst COX-2 selective inhibitors lumiracoxib is known to be novel drug that has better cardiovascular safety profile as well.

Common adverse effects on lower GI tract that are characterized by these studies are mucosal inflammation, erosions, ulcers, bleeding, strictures and aggravation of pre-existing inflammatory bowel disease and diverticular disease of the colon resulting in perforation. But to-date, there are only two case reports that show large bowel perforation that is attributable to NSAIDs use only. One of these case reports did not specify the formulation of the drug that was used.

NSAIDs are known to cause GI toxicity by both local action on gut mucosa and by systemic route. These drugs reduce the synthesis of inflammatory mediators like prostaglandins by inhibiting the cyclo-oxygenase (COX) enzyme. Decreased levels of prostaglandins in gut mucosa predispose it to adverse effects. Slow-release and enteric coated tablets by pass the stomach and release their active drug in distal GI tract slowly over a period of time thus increasing the time of contact with distal gut mucosa so they are associated more with distal GI tract complications.

In this case, patient had taken the slow release formulation of diclofenac 100 mg in twice-a-day dosage. Per-operatively in both the operations there was no granulation tissue present and no sign of malignancy or diverticular disease was present. Postoperative histopathology of operative specimen ruled out any other pathological cause of perforation with no evidence of malignancy or granulation tissue. Both HCV and HBV were negative by ELISA. So perforation was attributable only to the use of slow release diclofenac.

In pre-operative radiological investigation, abdominal ultrasonography and erect X-ray abdomen were performed in this case. In suspicious cases, there should always be CT scan to confirm the diagnosis pre-operatively, if time allows.

In patients with history of NSAIDs use, who present with symptoms and signs of peritonitis, distal GI perforation should always be considered as one of the possible diagnosis if other causes of his symptoms are ruled out. Physicians should be cautious to advise NSAIDs and these drugs should be given for as short period of time and in as low a dose as possible. If only anti-pyretic or analgesic effect is required, paracetamol should be the first choice. Use of selective COX-2 blockers in not free of cardiovascular complications, but lumiracoxib with minimal cardiovascular complications can be used in patients prone to develop NSAIDs induced complications.

**REFERENCES**