Correspondence

Perforative Peritonitis Probably Caused by Short-Term Use of Diclofenac Sodium

Sir,

Non-steroidal anti inflammatory drugs (NSAIDs), frequently prescribed for the management of musculoskeletal pain, have well documented gastrointestinal (GI) adverse effects. We present here a case of perforative peritonitis probably related to the use of therapeutic dose of diclofenac sodium for only nine days.

A 70-year old woman presented with complaints of abdominal pain and fever since five days. She did not give history of haematemesis, melaena or haematochezia. She reported use of diclofenac sodium, 50 mg, twice a day for nine consecutive days for relief from backache. There was no history of consumption of any other medication. The presentation was acute and there was no history of dyspepsia, peptic ulcer or acid reflux disease. Past surgical history was not significant. On examination, she was febrile, had tachycardia and was hypotensive. On abdominal palpation, tenderness, guarding and rigidity were present indicating perforative peritonitis. This was confirmed by a plain chest radiograph which showed free air under the right dome of the diaphragm.

An exploratory laparotomy was performed. A 5 mm perforation was seen in the anterior wall of the first part of the duodenum which was repaired using an omental patch. Generalised peritoneal contamination with presence of bilious fluid was noted. Repeated peritoneal washes were given and bilateral drains were placed. Postoperatively, the output was monitored and she was treated with antimicrobials, proton pump inhibitors (PPIs) and analgesics. The patient was discharged on day eight after presentation. There were no post-operative complications.

The causality assessment of the adverse drug reaction (ADR) was carried out using the Naranjo Scale. The assessment revealed the ADR to be ‘probably’ associated with diclofenac sodium.

NSAIDs with low risk of GI damage include ibuprofen, diclofenac and etodolac. Factors such as age, gender, history of previous GI ulceration, smoking, alcohol use, concomitant medication and Helicobacter pylori infection are associated with a greater risk of GI damage. Higher incidence of gastric perforation as compared to duodenal perforation has been observed with increase in age. Also, pyloric perforation is more frequent compared to duodenal in women. These findings are contrary to what was seen in the reported patient.

Secondary ulcers have acute presentations more frequently compared to primary ulcers. According to a survey, 81% of patients hospitalized with serious NSAID-induced complications had no previous gastrointestinal symptoms. They tend to present with complications such as haemorrhage or perforation as seen in this case. Gastrointestinal perforation on short term usage of NSAIDs is rare and this patient had documented use for only nine days.

Prevention of gastrointestinal adverse events related to NSAID use can be achieved by advising patients to take the drug after meals and the concomitant prescription of anti ulcer medication. Data suggests that the fixed combination of misoprostol and diclofenac is one of the safest formulations. The availability of some NSAIDs as over-the-counter medication, its widespread use and self medication has resulted in frequent adverse drug effects of relatively safe medication. This can be prevented by patient education and following the dictum of prescribing the correct drug in the minimum effective dose.

A patient who has sustained a GI ulcer secondary to NSAID use should avoid further use of them. A follow-up upper gastrointestinal endoscopy is desirable post-surgery to rule out concomitant gastric mucosa pathology.

This adverse drug reaction was reported to the South-West Zonal Centre of the National Pharmacovigilance Programme of India.

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