JSCR Journal of Surgical Case Reports

Journal of Surgical Case Reports, 2015;3, 1–2

doi: 10.1093/jscr/rjv018
Case Report

CASE REPORT

Perforated diverticulitis sigmoidei after laparoscopic cholecystectomy

Salameh Eljaja, Sabah Hadi, and Alaa El-Hussuna*

Department of Gastrointestinal Surgery, Slagelse Hospital, Slagelse, Denmark

*Correspondence address. Skovbyvej 18, Skovlunde 2740, Denmark. Tel: +45 21 51 72 35; Fax: +45 58 55 98 60; E-mail: alaanewemail@gmail.com

Abstract

We present a case of 47-year-old healthy man who underwent an uneventful elective laparoscopic cholecystectomy. Despite the postoperative analgesia with non-steroidal anti-inflammatory drugs (NSAIDs), the patient developed diffuse abdominal pain culminating on the second postoperative day when the patient also had rebound tenderness. A diagnostic laparoscopy showed diverticular perforation, which was treated with laparoscopic lavage and drain. The patient's condition continued to deteriorate and the drain output resembled faecal material necessitating an emergency sigmoidium resection. The histopathological examination confirmed inflammation and perforation in the diverticulosis-bearing segment. The use of NSAID can be a reason for perforation, and may be for diverticulitis. NSAID should be used with caution in patients with a previous history or endoscopic-verified diverticulosis.

INTRODUCTION

Non-steroidal anti-inflammatory drugs (NSAIDs), used on both short and long terms, can induce ulceration in the upper gastrointestinal (GI) tract. However, cases where NSAIDs induce perforation of the lower GI tract are rare [1]. NSAIDs act as a non-selective (for example, diclofenac and Ibuprofen) or selective cyclooxygenase (COX) inhibitor (for example, Celecoxib). The COX-1 enzyme contributes to the physiological functions of the body including the protection of gastric mucosa. Therefore, COX-1 inhibitors can induce gastric ulceration [2]. NSAIDs can damage the small bowel and the colon [3]. Enteropathy is frequent and may be present in >60% of patients taking these drugs long term. In most cases, damage is subclinical, including increased mucosal permeability, inflammation, erosions and ulceration, but other more serious clinical outcomes such as anaemia, overall bleeding, perforation, obstruction, diverticulitis and deaths have been described [3].

We report a case of a middle-aged man, who underwent an outpatient laparoscopic cholecystectomy, and was pre- and postoperatively treated with NSAIDs. One week after the operation, the patient developed diverticulitis sigmoidei, complicated by perforation.

CASE REPORT

A 47-year-old heavy smoker, otherwise healthy male, presented with 3 weeks of gallstone pain. After a couple of admissions where the patient was treated with a non-selective COX inhibitor (Todolac 600 mg daily) for ~2 weeks, he was scheduled to outpatient laparoscopic cholecystectomy.

Dissection of gallbladder was complicated by blood oozing from the liver. It was treated successfully, and haemostasis established. A drain was placed to monitor further bleeding episodes. The drain was removed on the second postoperative day as the output was negligible. Treatment for postoperative pain included 10 mg morphine tablet on need with a maximum dose of six times, Paracetamol tablet 1000 mg four times per day and Ibuprofen 400 mg three times per day.

On the evening of the second postoperative day, the patient complained of increasing diffuse pain. Vital signs were normal

Received: November 9, 2014. **Accepted:** February 9, 2015

and stable. Blood tests were normal. He was observed overnight, and the following morning the situation worsened with rebound tenderness and rigid abdomen. The situation was rapidly deteriorating, thus an immediate surgical intervention was indicated without diagnostic imaging. An emergency diagnostic laparoscopy revealed a diffuse peritonitis due to perforation of a diverticulum in colon sigmoidium. The abdomen was irrigated, and two drains were placed in both the right and left sides according to the department's guidelines. The following day, a leakage around the drains was noted, and a brilliant blue was given. A blue-coloured output in the drains confirmed the suspicion of perforation. The patient's condition was deteriorating, necessitating a new diagnostic laparoscopy that showed massive intraperitoneal fibrin plagues and terminal ileum adherent to the pelvis most probably due to the inflammation of the re-perforated colon sigmoideum. Resection of the diverticulosis-bearing segment in colon sigmoidium ad modum Hartmann was performed, and the patient was treated with broad-spectrum antibiotics. The postoperative course was uneventful and the patient was discharged on fifth postoperative day, after the third operation. The stoma was closed after 3 months. Histopathological examination of the specimen showed a spontaneous colonic perforation, inflammation in the sigmoidium colon wall, peri-diverticular fatty tissue in the sigmoidium mesentery and peritoneum.

DISCUSSION

In this case report, a young patient treated with Todolac before, and Ibuprofen after an outpatient laparoscopic cholecystectomy developed diverticulitis and perforation in colon sigmoidium. After two emergency diagnostic laparoscopies, the patient was treated by resection of the diverticulosis-bearing segment in colon sigmoidium ad modum Hartmann.

This patient had no previous history of diverticulosis and no previous endoscopic investigation of large bowel. It is interesting to note that this patient developed ulceration, perforation and inflammation only 1 day after having his gall bladder removed. A possible explanation is that the patient had diverticulosis coli. A large majority (80–85%) of patients with diverticular disease will remain entirely asymptomatic throughout their life. Only 1–2% will require hospitalization and 0.5% will require surgery [4]. Thus, asymptomatic diverticular disease, which ulcerated and later perforated due to the use of NSAIDs [5, 6], can be anticipated. Another possibility is NSAID-induced diverticulitis [3]. NSAID-induced diverticulitis has earlier been described in the literature, which makes it another possible explanation for the course of events in this case [3]. COX-1 inhibition seems to be the reason, pathophysiology was earlier described, but further studies are needed [4].

Numerous cases of ulceration and perforation of ventricle and intestines, both small and large, due to use of NSAIDs, are reported but perforation in sigmoidium colon is a rare finding [5]. COX-1 inhibitors have recently been pointed out as a major risk factor for developing diverticular bleeding [7] and colonic ulcers in patients with long-term use of NSAIDs [8].

Two earlier case reports showed that colonic perforation occurred due to short-term use of diclofenac alone [9]. Both cases involved young patients, who had been taking diclofenac for different reasons, over a short period. NSAIDs may exert their deleterious effects on the lower GI tract through both local and systemic actions. Systemic effects are caused by the inhibition of COX-1 and reduction of gastric epithelial cytoprotection by prostaglandins, thus exposing the gut for ulceration. The local damage of the intestinal mucosa in the distal bowel segment seems to be caused by sustained release formulation with a high enterohepatic circulation. The latter may act time and again on the intestinal mucosa through metabolites secreted in the gallbladder.

Recent studies on rats have shown that the selective COX-2 inhibitor Celecoxib has a less detrimental effect on the mucosa of the intestines [10]. This might be an alternative to be used in patients susceptible to gut ulceration.

CONFLICT OF INTEREST STATEMENT

None declared.

REFERENCES

1. Klein M, Linnemann D, Rosenberg J. Non-steroidal anti-inflammatory drug-induced colopathy. *BMJ Case Rep* 2011 pii:bcr.10.2010.3436; doi:10.1136/bcr.10.2010.3436.
2. Sostres C, Gargallo CJ, Arroyo MT, Lanas A. Adverse effects of non-steroidal anti-inflammatory drugs (NSAIDs, aspirin and coxibs) on upper gastrointestinal tract. *Best Pract Res Clin Gastroenterol* 2010;**24**:121–32.
3. Lanas A, Sopeña F. Nonsteroidal anti-inflammatory drugs and lower gastrointestinal complications. *Gastroenterol Clin North Am* 2009;**38**:333–52.
4. Sopeña F, Lanas A. Management of colonic diverticular disease with poorly absorbed antibiotics and other therapies. *Therap Adv Gastroenterol* 2011;**4**:365–74.
5. El Hajj I, Hawchar M, Sharara A. NSAID-induced colopathy: case report and review of the literature. *J Med Liban* 2009;**57**:274–6.
6. Klein M, Gögenur I, Rosenberg J . Risk of anastomotic leakage in postoperative treatment with non-steroidal anti-inflammatory drugs. *Ugeskr Laeger* 2010;**172**:1660–2.
7. Yuhara H, Corley DA, Nakahara F, Nakajima Y, Koike J, Igarashi M, et al. Aspirin and non-aspirin NSAIDs increase risk of colonic diverticular bleeding: a systematic review and meta-analysis. *J Gastroenterol* 2014;**49**:992–1000.
8. Gut A, Halter F, Ruchti C. Nonsteroidal antirheumatic drugs and acetylsalicylic acid: adverse effects distal to the duodenum. *Schweiz Med Wochenschr* 1996;**126**:616–25.
9. Shahzad N, Maqbool A, Gauhar TM, Azim KM. Colonic perforation induced by short term use of slow release diclofenac. *J Coll Physicians Surg Pak* 2012;**22**:328–9.
10. Fornai M, Antonioli L, Colucci R, Pellegrini C, Giustarini G, Testai L, et al. NSAID-induced enteropathy: are the currently available selective COX-2 inhibitors all the same? *J Pharmacol Exp Ther* 2014;**348**:86–95.