

PROPOFOL AND POSTOPERATIVE PANCREATITIS- A CASE REPORT

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Introduction

Propofol is a commonly used agent to induce and maintain general anesthesia. Since its introduction in 1989, there have been reports of acute pancreatitis after single administration doses of propofol¹ in otherwise healthy patients, and as such, a suggestive link has been made between the two. Drug-induced acute pancreatitis has been reported to be 1.4% of acute pancreatitis cases, yet most patients that developed acute pancreatitis after propofol exposure were chronically ill with multiple co-existing medical conditions². We present a case of postoperative pancreatitis that developed in a healthy woman who received a single induction dose of propofol on two separate occasions.

Methods

In order to review the possible correlation between propofol and acute pancreatitis, all acute pancreatitis cases within one year were reviewed retrospectively with the approval of the institutional review board. Out of 103 postoperative pancreatitis cases, we excluded all cases that had upper abdominal surgery or the lack of use of propofol. There was only one case of pancreatitis in a non-upper abdominal surgery with exposure to propofol.

Results

Case Report: A healthy 42 year old female, G0P0, without any past medical history, had been experiencing intermenstrual bleeding for one year. Pelvic ultrasound revealed two leiomyomas within the uterus. She denied a history of gallstones, pancreatitis, hepatitis, hyperlipidemia, drug or alcohol use, medication use and drug allergies. She was admitted for elective total abdominal hysterectomy. Induction of anesthesia was accomplished with propofol 150 mg, fentanyl 100 ug, and vecuronium 6 mg. Sevoflurane+O₂+air was used for maintenance. The procedure and extubation were uneventful.

On POD #1, she had a fever of 100.2° F and received one dose of Tylenol. On POD #2 she complained of abdominal pain as her diet was slowly advanced. By POD #3, she had nausea followed by tenderness and guarding. An abdominal CT revealed thickened bowel wall and small bilateral pleural effusions. On POD #5 she underwent exploratory laparotomy and again received propofol 150 mg bolus for induction. The intra-operative findings consisted of 500 cc of red tinged fluid and an edematous small bowel, but no injury to the small bowel or large bowel, and no abscess. Over the next two days she showed significant clinical improvement from pre-laparotomy indices. On POD #3 (from the ex-lap) she was started on TPN and over the next couple of days became progressively nauseous as her abdominal pain returned. By POD #8 she had a tender, distended abdomen, low grade fever of 100.9° F and a first recorded value for amylase of 189 and lipase of 421. Her TPN was stopped. An abdominal ultrasound ruled out gallstone or biliary abnormality. Pancreatitis was diagnosed with an unknown cause. Over the next 5 days the enzymes trended towards normal, her symptoms resolved, and she was discharged without further incident. Her only triglyceride value was taken POD #12 and was 73 mg/DL.

Conclusion

This case of acute postoperative pancreatitis developed in a patient without any risk factors. She received 150 mg of propofol twice, five days apart, for induction of both surgical procedures. In reviewing the presentation of symptoms within the timeline of exposure to agents, propofol appears most likely, as she developed the symptomatology of pancreatitis prior to the second dose of propofol and the start of TPN. Most reported cases involve propofol infusions for prolonged periods. Leisure et al³ reasoned that it would be unlikely that intraoperative bolus doses of propofol would either raise blood lipids or cause pancreatitis. However, as in this case, if an otherwise healthy patient undergoes an uncomplicated surgical procedure and develops postoperative abdominal pain, one should see if the patient received propofol and may thus be experiencing an acute pancreatitis.

References

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