

Propofol-Induced Severe Necrotizing Pancreatitis

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ABSTRACT

Propofol is a widely used sedative for gastrointestinal endoscopic procedures. Drug-induced pancreatitis is a relatively rare disease possibly because of poor recognition. Propofol-induced pancreatitis is an extremely rare phenomenon. We present a 22-year-old healthy man who underwent esophagogastroduodenoscopy with propofol as a sedative. Soon after, he developed acute upper gastrointestinal symptoms and was diagnosed with pancreatitis. His prolonged hospital course was complicated with necrotizing pancreatitis, acute respiratory distress syndrome, septic shock, and other end-organ damages. We hope to increase awareness of a life-threatening adverse event of a commonly used anesthetic such as propofol.

INTRODUCTION

Propofol, 2,6-diisopropylphenol, is a commonly used agent for induction and maintenance of anesthesia. It has also gained widespread popularity for gastrointestinal (GI) endoscopic procedures because it is short-acting and has fewer side effects.^{1,2} Drug-induced pancreatitis is a relatively rare disease possibly because of poor recognition.³ However, propofol-induced pancreatitis has been reported, although a seemingly rare phenomenon.³ We present a case of severe necrotizing pancreatitis with multiorgan failure, attributed to delayed reaction after receiving propofol for an elective esophagogastroduodenoscopy (EGD) procedure.

CASE REPORT

A 22-year-old white man with a medical history of gastroesophageal reflux disease and past cholecystectomy underwent EGD evaluation for intermittent esophageal dysphagia of many years. His only home medication was omeprazole. He had no EGD, previous exposure to propofol, or known drug and environmental allergies. He did not use tobacco, drink alcohol, or use any illicit drugs. He had no family history of significant medical conditions. EGD was accomplished without any immediate complications. For procedural sedation, he received a total of 150 mg of intravenous propofol. EGD revealed mild esophageal stricture at the esophagogastric junction, which was dilated to 20 mm using a balloon dilator. Esophageal mucosal changes, including ringed esophagus, feline appearance, and longitudinal furrows, were also found in the middle and lower third of the esophagus. Biopsies were taken from the esophagus which showed eosinophilic esophagitis. The remainder examined upper GI tract was otherwise unremarkable.

He tolerated the procedure and anesthesia well. However, about 90 minutes after his discharge, he called and reported a sudden onset of severe epigastric pain and several episodes of emesis that started after a meal. He was instructed to go to the emergency department immediately for further evaluation. In the emergency department, he was found to have marked elevation of his lipase at about 14,000 U/L and a total leukocyte count of 30,000 cells per liter with no eosinophilia, high neutrophils, and normal lymphocyte counts. His triglycerides level was 79 mg/dL. Initial abdominal and pelvic computed tomography showed peripancreatic fluid without any evidence of cyst, abscess, or necrosis. He was initially admitted to the general medical floor for intravenous fluid hydration and pain control but was soon transferred to the intensive care unit (ICU) because of deterioration and development of hypoxia and hemodynamic instability.

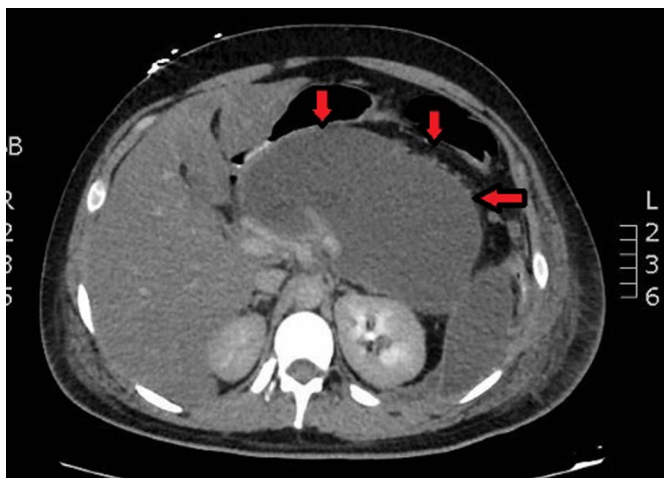


Figure 1. Abdominal displaying pancreatitis with peripancreatic fluid collection (red arrows).

In the ICU, he developed acute hypoxic respiratory failure due to acute respiratory distress syndrome requiring intubation and mechanical ventilation. Subsequently, he developed septic shock, requiring vasopressor support and broad-spectrum antibacterial and antifungal agents; significant pleural effusion, requiring thoracentesis and chest tube placement; and acute kidney injury that improved gradually without the need for dialysis. Repeat abdominal and pelvic computed tomography 10 days later demonstrated pancreatic necrosis with extensive peripancreatic fluid extending to post-pararenal space along with retroperitoneal fluid collection, requiring percutaneous drain placement and ultimately pancreatic necrosectomy (Figure 1). To complicate matters further, the patient also developed severe ischemic colitis because of shock, requiring a subtotal colectomy. The patient had had a prolonged course of hospitalization, mostly in the ICU setting at 2 tertiary care centers for over 6 months, receiving cares from multiple medical and surgical specialties. He was then transferred to rehab facility and is currently doing well.

Exhaustive diagnostic workup had been performed to exclude other causes of pancreatitis. This patient had a cholecystectomy 2 years ago. His initial abdominal imaging and laboratory work ruled out gallstone pancreatitis and pancreatic divisum. He had no history of alcohol use. Extensive and repeated laboratory tests and microbiology excluded common infectious pathogens. A nasopharyngeal respiratory viral polymerase chain reaction testing was positive for human rhinovirus; however, serum testing was negative, which rules out a viral cause for the patient's pancreatitis. Autoimmune etiology was ruled out because immunoglobulin levels were normal. He had initial unremarkable blood work, including normal serum triglyceride level, except for elevated lipase level and total white count at presentation. His only home medication was omeprazole that he had been taking for multiple years. He had no previous trauma or exposure to drugs or

toxins. Testing for rare causes such as cystic fibrosis and myotonic dystrophy was also negative. He had no history of vascular disease or vasculitis. He had no known history of drug or environmental allergies. Given the chronological association of propofol administration and immediate development of acute pancreatitis without any other causes, it was concluded that the patient had propofol-induced severe necrotizing pancreatitis.

DISCUSSION

Propofol is lipophilic and a global central nervous depressant with a rapid onset of sedation, dose-related hypnotic effect, and a quick recovery profile.⁴ Although extensive use of propofol has increased patient satisfaction and acceptability in GI procedures, risks of over-sedation and complications are not negligible.^{5,6} Deeper sedation levels can increase the risk of procedural hypotension, colonic perforation, and aspiration pneumonia.^{5,7,8} Moreover, propofol may lead to increase in mortality and morbidity from cardiopulmonary complications in elderly compared with younger patients probably because of reduced renal clearance.⁹

Case reports have been published over the years that showed propofol as a rare causative agent for pancreatitis.^{1,4,10-13} The mechanism of this drug-induced pancreatitis is largely unknown, although propofol-induced hypertriglyceridemia, hypersensitivity, or direct pancreatic toxicity had been suggested as possible culprit.^{3,14} We present a case of severe necrotizing pancreatitis within hours of after single dosage of propofol exposure that ultimately led to multiorgan failure. Initial triglyceride levels were normal, and all other causes of pancreatitis were systematically ruled out. Hence, the mechanism of propofol-induced pancreatitis in our case remains unidentified, suggesting an idiosyncratic reaction.

Although propofol-induced pancreatitis is a rare phenomenon, the exact incidence is unclear and potentially much higher due in part to possible poor recognition, especially for mild cases. We hope that this case report will help increase pancreatitis awareness as a potentially life-threatening adverse event associated with propofol sedation and not assume postprocedural abdominal pain to be secondary to abdominal dilation from periprocedural air insufflation. The debate is likely to continue as to the cost, risks, and benefits of ever-popular propofol-induced deep sedation vs conventional benzodiazepines and opioid-induced conscious sedation for GI endoscopic procedures. We would like to make the clinicians aware of the rare but potentially fatal complication of propofol, and they should consider other alternative sedatives.

DISCLOSURES

Author contributions: All authors contributed equally to this manuscript. H. Zhang is the article guarantor.

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Informed consent was obtained for this case report.

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REFERENCES

- Jawaid Q, Presti ME, Neuschwander-Tetri BA, Burton FR. Case report: Acute pancreatitis after single-dose exposure to propofol: A case report and review of literature. *Dig Dis Sci* 2002;47(3):614–8.
- Muniraj T, Aslanian H. Hypertriglyceridemia independent propofol-induced pancreatitis. *JOP* 2012;13(4):451–3.
- Csomor J, Murinová I, Broulíková K, et al. Propofol-induced acute pancreatitis. *J Clin Pharm Ther* 2017;42(4):495–8.
- Gottschling S, Meyer S, Krenn T, et al. Effects of short-term propofol administration on pancreatic enzymes and triglyceride levels in children. *Anaesthesia* 2005;60(7):660–3.
- Goudra B, Singh PM, Gouda G, Borle A, Carlin A, Yadwad A. Propofol and non-propofol based sedation for outpatient colonoscopy-prospective comparison of depth of sedation using an EEG based SEDLine monitor. *J Clin Monit Comput* 2016;30(5):551–7.
- Lovett P, Gómez V, Hodge DO, Ladlie B. Propofol versus midazolam/fentanyl sedation for colonoscopy in the elderly patient population. *J Perianesth Nurs* 2017;32(3):210–4.
- Adeyemo A, Bannazadeh M, Riggs T, Shellnut J, Barkel D, Wasvary H. Does sedation type affect colonoscopy perforation rates? *Dis Colon Rectum* 2014; 57(1):110–4.
- Basavana Goudra AN, Nuzat A, Singh PM, Borle A, Carlin A, Gouda G. Association between type of sedation and the adverse events associated with gastrointestinal endoscopy: An analysis of 5 years' data from a tertiary center in the USA. *Clin Endosc* 2017;50(2):161.
- Horiuchi A, Nakayama Y, Tanaka N, Ichise Y, Katsuyama Y, Ohmori S. Propofol sedation for endoscopic procedures in patients 90 years of age and older. *Digestion* 2008;78(1):20–3.
- Donmez A, Arslan G, Pirat A, Demirhan B. Is pancreatitis a complication of propofol infusion? *Eur J Anaesthesiol* 1999;16(6):367–70.
- Gottschling S, Larsen R, Meyer S, Graf N, Reinhard H. Acute pancreatitis induced by short-term propofol administration. *Paediatr Anaesth* 2005; 15(11):1006–8.
- Ting TW, Lee JH. Acute pancreatitis after propofol infusion in a teenage patient. *Anaesth Intensive Care* 2012;40(3):561–2.
- Donmez A, Sener M, Candan S, Arslan G. Can we blame Propofol for pancreatitis? *Pharmacotherapy* 1999;19(10):1181–2.
- Asghar MU, Cheema HA, Tanveer K, Leinwand J. Propofol infusion and acute pancreatitis: A review. *Am J Ther* 2020;27(4):e3711–4.

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