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증 례

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대구가톨릭대학교 의과대학 응급의학교실[1], 경상대학교 의과대학 응급의학교실[2]

이숙희[1] · 이경우[1] · 정진희[2]

Pneumatosis Cystoides Intestinales and Portomesenteric Venous Gas following Anticholinesterase Pesticide Poisoning

Suk Hee Lee, M.D.[1], Kyung-Woo Lee, M.D., Ph.D.[1], Jin Hee Jung, M.D.[2]

Department of Emergency Medicine, College of Medicine, Catholic University of Daegu, Daegu[1], Department of Emergency Medicine, School of Medicine, Gyeongsang National University, Jinju[2], Korea

Pneumatosis cystoides intestinalis and portomesenteric venous gas are uncommon radiological findings, but are found commonly in cases of bowel ischemia, or as a result of various non-ischemic conditions. A 72-year-old man visited an emergency center with altered mental status 2 hours after ingestion of an unknown pesticide. On physical examination, he showed the characteristic hydrocarbon or garlic-like odor, miotic pupils with no response to light, rhinorrhea, shallow respiration, bronchorrhea, and sweating over his face, chest and abdomen. Laboratory results revealed decreased serum cholinesterase, as well as elevated amylase and lipase level. We made the clinical diagnosis of organophosphate poisoning in this patient based on the clinical features, duration of symptoms and signs, and level of serum cholinesterase. Activated charcoal, fluid, and antidotes were administered after gastric lavage. A computerized tomography scan of the abdomen with intravenous contrast showed acute pancreatitis, poor enhancement of the small bowel, pneumatosis cystoides intestinalis, portomesenteric venous gas and ascites. Emergent laparotomy could not be performed because of his poor physical condition and refusal of treatment by his family. The possible mechanisms were believed to be direct intestinal mucosal damage by pancreatic enzymes and secondary mucosal disruption due to bowel ischemia caused by shock and the use of inotropics. Physicians should be warned about the possibility of pneumatosis cystoides intestinalis and portomesenteric venous gas as a complication of pancreatitis following anticholinesterase poisoning.

Key Words: Anticholinesterase, Pancreatitis, Pneumatosis cystoides intestinalis, Poisoning, Portomesenteric venous gas

Introduction

Anticholinesterase pesticides including organophosphate, carbamate and organophosphate compounds are widely used insecticides in agriculture, which inhibit the enzyme cholinesterase. The signs and symptoms of anticholinesterase poisoning are miosis, muscle weakness, bradycardia, diarrhea, salivation,

책임저자: 이 경 우
대구광역시 남구 두류공원로 17길 33
대구가톨릭대학교 의과대학 응급의학교실
Tel: 053) 650-4197, Fax: 053) 650-4930
E-mail: turtle072@hanmail.net

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lacrimation, sweating, and secretions in the respiratory and gastrointestinal tract[1]. Complications such as respiratory failure, intermediate syndromes, and rarely acute pancreatitis can occur[1]. Anticholinesterase poisoning can be diagnosed by clinical suspicion such as typical clinical symptoms, unique smell of pesticides or solvents, and serum cholinesterase activity[2].

Pneumatosis cystoides intestinalis and portomesenteric venous gas are not specific diseases, but mere diagnostic findings in patients with acute abdominal pathology[3]. Pneumatosis cystoides intestinalis and portomesenteric venous gas are uncommon radiological findings, but can be found commonly in cases of bowel ischemia, or as a result of various non-ischemic conditions less frequently[4]. Even though pneumatosis cystoides intestinalis and portomesenteric venous gas can be found in any type of diseases regardless of severity, the exact pathogenesis is not fully understood[5]. This is an unusual case of a patient with pneumatosis cystoides intestinalis and portomesenteric venous gas as a complication of unknown pesticide poisoning.

Case

A 72-year-old man with diabetes mellitus, hypertension and dementia was admitted to hospital with altered mental status 2 hours after ingestion of unknown pesticide which had been kept in a soda bottle. There was no way to check which content he took, because the patient or his family did not bring the container with a tag. His blood pressure was 110/70 mmHg, heart rate 80 beat per minute, respiratory rate 24 per minute and body temperature 36°C. His mental status was unconscious and there was no response to pain. It turned out that his Glasgow Coma Scale score was 3. He showed the characteristic hydrocarbon or garlic-like odor, miotic pupils with no response to light, rhinorrhea, shallow respiration, bronchorrhea, and sweating over his face,

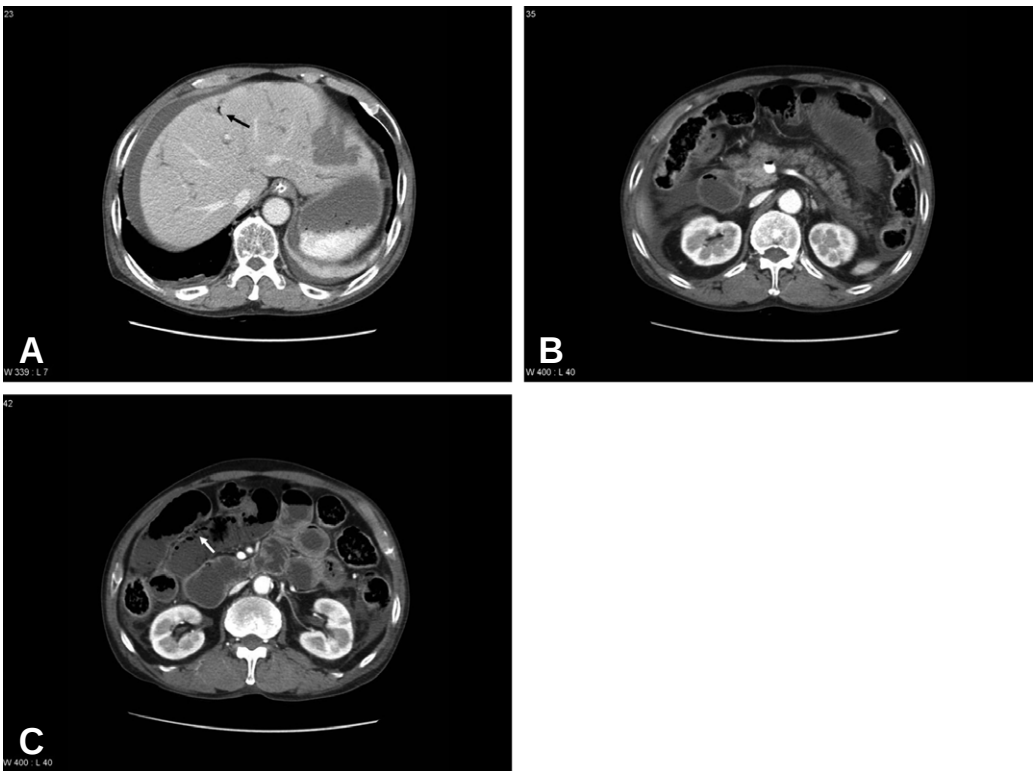

Fig. 1. (**A**) Portal vein gas (black arrow) and perihepatic fluid collection. (**B**) Acute pancreatitis. (**C**) Pneumatosis cystoides intestinalis (white arrow). CT scan of the abdomen with intravenous contrast shows (**A**) gas in the portal vein (black arrow) and perihepatic fluid collection, (**B**) acute pancreatitis with peripancreatic fluid collection and gas within the superior mesenteric vein, and (**C**) pneumatosis cystoides intestinalis (white arrow), poor enhancement and fluid-filled dilatation of the small bowel loops.

chest and abdomen on the physical examination. Laboratory results revealed decreased serum cholinesterase level (1 IU/L; reference value: 203-406 IU/L), elevated amylase level (762 U/L; reference value: 29-110 U/L), elevated lipase level (over 400 U/L; reference value: 14-60 U/L), and mild azotemia (blood urea nitrogen 25.1 mg/dL; reference value: 6-20 mg/dL and creatinine 1.24 mg/dL; reference value: 0.6-1.2 mg/dL). The laboratory testing for specific compound of anticholinesterase was not available. He was intubated and supported by mechanical ventilation on admission after detecting his comatous mentality and poor respiratory function. Activated charcoal, fluid, and antidotes (atropine, pralidoxime) were administered after gastric lavage. A total 306mg of atropine was administered. It was given as bolus (1-5 mg every 5 minutes) until the patient was fully atropinized. Subsequently, atropinization was maintained with either multiple boluses of atropine or infusion. Pralidoxime was administered as loading dose of 1 g with normal saline followed by maintenance dose of 500 mg per hour until he expired. After 7 hours, systolic blood pressure dropped to 70 mmHg. Dopamine and norepinephrine infusion were started because of refractory shock despite adequate fluid resuscitation. On the second day of admission, laboratory results revealed decreased serum cholinesterase level (3 IU/L; at 37 hours after admission), elevated amylase (over 2,400 U/L) and lipase level (over 2,400 U/L). It was impossible to quantify the exact amount of amylase and lipase, which was greater than 2,400 U/L. Computerized tomography (CT) scan of the abdomen with intravenous contrast showed acute pancreatitis, poor enhancement of the small bowel, pneumatosis cystoides intestinalis, portomesenteric venous gas and ascites (Fig. 1). The emergent laparotomy, as a mandatory next step, could not be performed because his physical condition was getting worse despite the adequate administration of fluid, inotropics, antidotes and antibiotics. Furthermore, his family refused to have more operative interventions for him. He finally expired on the third day after admission.

Discussion

This case was a patient who had acute pancreatitis and shock with cholinergic symptoms after ingestion of an unknown pesticide. We made the clinical diagnosis of organophosphate poisoning in this patient based on the characteristic clinical features, long duration of symptoms and signs, strong smell of pesticides or solvents, and reduced cholinesterase level checked on admission and 37 hours after admission. We observed elevated amylase, lipase level and findings suggesting acute pancreatitis, pneumatosis cystoides intestinalis and portomesenteric venous gas on his CT scan.

Acute pancreatitis following anticholinesterase poisoning is caused by increased cholinergic stimulation, spasm of the sphincter of oddi and pancreatic ductal hypertension[1]. It is mild and rarely has fatal complications[6]. There is no previous report about acute pancreatitis with pneumatosis intestinalis or portomesenteric venous gas following anticholinesterase poisoning. There are some cases that patients with necrotizing pancreatitis had pneumatosis cystoides intestinalis or portomesenteric venous gas but some necrotizing pancreatitis following anticholinesterase poisoning cases have been reported without pneumatosis cystoides intestinalis and portomesenteric venous gas[7-10]. But in this case, one of the causes of leading him to death is bowel involvement as a complication of pancreatitis and refractory shock.

Pneumatosis cystoides intestinalis and portomesenteric venous gas have been known to be most commonly caused by bowel ischemia. Other factors can be non-ischemic conditions, including inflammatory, infectious, obstructive, iatrogenic, traumatic, neoplastic, and idiopathic causes[3,4]. In acute pancreatitis, direct damage to the intestinal mucosa by pancreatic enzymes or the secondary mucosal disruption due to bowel ischemia were suggested as possible mechanisms of gas translocation from the lumen through the bowel wall[11,12]. Gas in the intestinal lumen can pass through the intestinal wall and travel via the superior or inferior mesenteric vein and its branches into the portal vein[11,13].

In this case, we can also consider that hypotensive episodes and the use of inotropic drugs could presumably result in splanchnic vasoconstriction and bowel ischemia. Although the pathogenesis of pneumatosis cystoides intestinalis and portomesenteric venous gas in acute bowel ischemia has not been established yet, a study supposed that it may be related with ulcerations and mucosal damage of the bowel wall in ischemic mucosa. When mucosal discontinuity occurs, it will permit the passage of gas from the intestinal lumen into the bowel wall[3].

Diagnosis of bowel involvement following acute pancreatitis is difficult. Bowel complications are generally manifested 4-6 weeks after the onset of acute pancreatitis. Physicians can diagnose when the patients' clinical signs and symptoms get worse and they suspect bowel complications with CT scan of abdomen because it has proven useful for diagnosing the early stage of bowel ischemia[3,14]. We doubt his rapid progression including his poor condition, elevation of pancreatic enzyme level, and refractory shock. We suggest that CT scan of abdomen is performed to patients who don' t have only clinical manifestations of acute pancreatitis but also have unexplainable courses.

The exact mechanism of pneumatosis cystoides intestinalis, and portomesenteric venous gas in this patient is not well known, but possible mechanisms were believed to be direct intestinal mucosal damage by pancreatic enzymes and secondary mucosal disruption due to bowel ischemia from shock and the use of inotropics.

Conclusion

This case illustrated a rare complication; acute pancreatitis with pneumatosis cystoides intestinalis and portomesenteric venous gas following anticholinesterase poisoning. Physicians should be warned about some possibilities of pneumatosis cystoides intestinalis, portomesenteric venous gas as a complication of pancreatitis following anticholinesterase poisoning, and they need to be educated to how to treat it accordingly.

REFERENCES

1. Hsiao CT, Yang CC, Deng JF, Bullard MJ, Liaw SJ. Acute pancreatitis following organophosphate intoxication. J Toxicol Clin Toxicol 1996;34:343-7.
2. Hmouda H, Ben Salem C, Bouraoui K. Management of acute organophosphorus pesticide poisoning. Lancet 2008; 371:2169-70.
3. Lai WH, Hwang TL, Chen HW. Portomesenteric venous gas in acute bowel ischemia: report of a case. Surg Today 2008;38:656-60.
4. Wiesner W, Mortelé KJ, Glickman JN, Ji H, Ros PR. Pneumatosis intestinalis and portomesenteric venous gas in intestinal ischemia: correlation of CT findings with severity of ischemia and clinical outcome. AJR Am J Roentgenol 2001;177:1319-23.
5. St Peter SD, Abbas MA, Kelly KA. The spectrum of pneumatosis intestinalis. Arch Surg 2003;138:68-75.
6. Yoshida S, Okada H, Nakano S, Shirai K, Yuhara T, Kojima H et al. Much caution does no harm! Organophosphate poisoning often causes pancreatitis. J Intensive Care 2015; 3:21.
7. Chan AC, Poon RT. Portal venous gas and pneumatosis intestinalis in necrotizing pancreatitis. Clin Gastroenterol Hepatol 2014;12:e57-8.
8. Roeyen G, Chapelle T, Jorens P, de Beeck BO, Ysebaert D. Necrotizing pancreatitis due to poisoning with organophosphate pesticides. Acta Gastroenterol Belg 2008;71:27-9.
9. Rizos E, Liberopoulos E, Kosta P, Efremidis S, Elisaf M. Carbofuran-induced acute pancreatitis. JOP 2004;5:44-7.
10. Moritz F, Droy JM, Dutheil G, Melki J, Bonmarchand G, Leroy J. Acute pancreatitis after carbamate insecticide intoxication. Intensive Care Med 1994;20:49-50.
11. Faberman RS, Mayo-Smith WW. Outcome of 17 patients with portal venous gas detected by CT. AJR Am J Roentgenol 1997;169:1535-8.
12. Paran H, Epstein T, Gutman M, Shapiro Feinberg M, Zissin R. Mesenteric and portal vein gas: computerized tomography findings and clinical significance. Dig Surg 2003;20:127-32.
13. Sebastià C, Quiroga S, Espin E, Boyé R, Alvarez-Castells A, Armengol M. Portomesenteric vein gas: pathologic mechanisms, CT findings, and prognosis. Radiographics 2000;20:1213-24.
14. Chai JY, Yun SI, Bae SS, Chae HB, Park SM, Youn SJ et al. A case of jejunal infarction and perforation due to acute pancreatitis. Korean J Gastroenterol. 2004;43:120-4.