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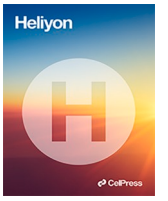

Case report

Hepatic portal venous gas after ingesting glyphosate: A case report and literature review

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ABSTRACT

Background: Glyphosate is a widely used herbicide. Clinical presentations of glyphosate intoxication show variation, but hepatic portal venous gas(HPVG) caused by glyphosate poisoning is rarely reported. Herein, we report a rare case of ominous HPVG after ingesting glyphosate. HPVG, which used to be an ominous abdominal radiologic sign, is associated with numerous underlying abdominal pathologies, ranging from benign conditions that require no invasive treatment to potentially lethal diseases that necessitate prompt surgical intervention.

Case summary: A young woman who ingested 100 mL glyphosate 6-h prior was admitted to the emergency intensive care unit. Before admission to our hospital, the patient was administered gastric lavage treatment with 10000 mL of normal saline in the local hospital. After 14 h, her laboratory examinations showed systemic inflammatory response syndrome and multiple organ dysfunction syndrome, while the condition deteriorated. Computed tomography of the abdomen showed multilinear air densities in the portal vein, hepatic branches, and mesenteric vessels, intestinal obstruction, and intestinal necrosis. Septic shock and a severe abdominal infection were diagnosed. The patient was treated conservatively as they could not tolerate surgery and, after 20 h died of septic shock.

Conclusion: We reviewed 289 cases of "hepatic portal venous gas" in PUBMED and analyzed the etiology and treatment of HPVG accompanied by the underlying pathology. We concluded that HPVG is a radiological sign associated with various diseases, and the prognosis mainly depends on the underlying cause and clinical condition. As glyphosate may erode the digestive tract, attention should be paid to the volume, pressure, and speed of gastric lavage in treating glyphosate poisoning to avoid fatal complications such as HPVG. Abdominal symptoms need to be closely observed, and changes in the early onset of the condition in clinical practice need to be responded to promptly.

1. Core tip

This was a rarely case of the patient who ingested glyphosate, which led to HPVG. After reviewing the published literature using the PubMed MEDLINE database, we found that >40 types of diseases that can cause HPVG. Among all of 289 cases identified, a total number of 90 cases of HPVG were due to gastrointestinal ischaemia with a mortality rate of 31.8 %. The prognosis is related to the

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pathology itself. Distinguishing between benign and potentially lethal diseases is important to prompt surgical intervention. We infer that the HPVG with oral glyphosate poisoning is related to the toxic effects of the herbicide, as well as inappropriate gastric lavage pressure and speed. This is a reminder to pay attention to the aggravation of medical treatment in such patients, where early detection and treatment can improve mortality.

2. Introduction

The ease of access to glyphosate as an alternative to paraquat allows for intentional ingestion of glyphosphate and commonly occurs in suicide attempts throughout Asia. The mechanisms of glyphosate poisoning are not the same as those of paraquat and organophosphate poisoning [1]. The clinical features of glyphosate surfactant poisoning are varied and include renal and hepatic impairment, respiratory distress, pulmonary oedema, shock, and disturbance of consciousness [2]. Nevertheless, an antidote is unavailable for glyphosate surfactant poisoning, and its management is principally symptomatic and supportive [2], including ingested gastric lavage, early renal replacement therapy, and using intravenous lipid/fat emulsion. Glyphosate surfactant poisoning can also cause corrosive injuries to the gastrointestinal tract.

Herein, we describe a case where the patient purposely ingested glyphosate for suicide. The computed tomography (CT) images showed hepatic portal venous gas (HPVG), mesenteric vein gas, and pneumatosis intestinalis. The patient finally died of intestinal ischaemia due to glyphosate and inappropriate gastric lavage.

Timeline:

Time	Event
−6 h	Intentionally ingested approximately 100 mL glyphosate
−1 h	Administered gastric lavage treatment using 10000 mL normal saline
0 h	Admitted to our hospital
14 h	Patient complained of severe abdominal pain
17 h	Patient's condition deteriorated
17:25 h	CT showed multilinear air density in the portal vein
18 h	Patient's condition meant she could not tolerate surgery
20 h	Patient was pronounced dead

Table 1
The laboratory tests of the patient.

	Admission of 3 hours	Admission of 12 hours	Admission of 20 hours
Arterial blood gases			
PH	7.35	7.202	6.850
PCO_2 (mmHg)	16.8	20.6	
PO_2 (mmHg)	70	83.1	37.8
HCO_3^- (mmol/l)	9.0	7.9	22.9
lactate	8.0	5.2	15.2
Complete blood count			
WBC ($\times\ 10^9$/L)	33.09	29.59	3.02
neutrophil ratio (%)	92.5 %	95.3 %	70.9 %
HGB (g/L)	152	159	112
PLT($\times\ 10^9$/L)	266	359	167
Biochemical data			
D-D ($\mu g/\ nL$)	12.41	1.56	5.10
APTT (s)	33.5	>180s	>180s
PT (s)	12.7	13.9	59.2
PCT(ng/ml)	15.8	>100	>100
IL-6 (pg/ml)	>5000	4848	>5000
K (mmol/l)	3.12	3.54	6.102
Na (mmol/l)	142.5	138.45	165.28
Mg (mmol/l)	0.733	1.010	1.318
creatinine (μ mol/L)	159.44	171.84	294.92
ALT (U/L)	71.6	528	2983.7
AST (U/L)	214	714	7028.9
lactate dehydrogenase (U/L)	789	2463	6439
myoglobin (ng/ml)	1259	>3000	>3000
creatine kinase (U/L)	144.43	1121	3387
BNP (pg/ml)	361.5	2677	22655
Troponin(ng/ml)	0.01	0.108	0.213
albumin (g/L)	49.5	50.10	16.2
globulin (g/L)	26.31	28.67	12.77
Blood glucose (mmol/l)	7.69	11.64	0.54

Patient Information: A 37-year-old female was admitted to the emergency ICU after intentionally ingesting approximately 100 mL of glyphosate 6-h prior with intentions to commit suicide due to family emotional conflicts. One hour before she arrived at our hospital, she went to the local hospital and was given gastric lavage treatment using 10000 mL of normal saline in the emergency department. **Physical Examination:** When the patient went to the emergency room at our hospital, they had no complaint of abdominal pain or heavy breathing. Her consciousness was clear. The vital signs were body temperature 37 °C, a blood pressure of 144/99 mmHg, a pulse rate of 110 beats/min, respiratory rate of 20 times/minute, blood oxygen saturation 98 % and physical examination showed that the lips were slightly swollen and that the oral mucosa was intact with no erosions; the cardiopulmonary examination was normal. After 5 h, the patient complained of nausea, vomiting, abdominal pain, heavy breathing, and delirium, and her vital signs were unstable (body temperature 39 °C, blood pressure 110/70 mmHg, pulse rate 150 beats/min). The abdomen was soft, with no rebound tenderness or abdominal guarding. **Assessment:** Laboratory examination and arterial blood gas analysis showed the following: white blood cells (WBC) 33.09 $\times$ 10^9/L, neutrophil ratio 92.5 %, Hemoglobin (HGB) 15.2 g/L, glutamic alanine transaminase 214 U/L, creatinine 159.4 μ mol/L, troponin 0.037 ng/mL, and myoglobin 1259 ng/mL, pH 7.35, $PaCO_2$ 16.8 mmHg, PaO_2 70 mmHg, HCO_3^- 9.0 mmol/L, and Lac 8.0 mmol/L, BE −13.5. We treated the patient with fluid resuscitation and the administration of IV cefoperazone sulbactam sodium. The patient remained anuretic after admission. To promote toxic metabolism, correct internal environmental disorders, and maintain hemodynamic stability, bedside blood purification therapy (lasting 11 h, ultrafiltrate 1100 mL) and organ function support therapy were administered. Fourteen hours after admission, the patient complained of severe abdominal pain, and a physical examination revealed rebound tenderness with tense abdominal walls. After 3 h, the condition of the patient deteriorated, leading to unconsciousness. The patient had whole body convulsions, foaming at the mouth, incontinence, and hypotension (43/24 mmHg). Blood examinations showed a WBC 3.02 $\times$ 10^9/L, neutrophil ratio 70.9 %, PT 59.2 s, blood glucose 0.54 mmol/L, osmotic pressure 322.89 Osm, alanine aminotransferase 2983.7 u/L, aspartate aminotransferase 7028.9 u/L, albumin 16.2 g/L, total immunoglobulin 12.77 g/L, creatine kinase 3387 μ mol/L, lactate dehydrogenase 6439 U/L, myoglobin >3000 ng/mL, troponin 0.21 ng/mL, and human brain natriuretic peptide 22655 pg/mL, and the lactate level had increased to 15.0 mmol/L. The changes in the laboratory examinations of this patient are shown in Table 1. **Interventions:** The patient developed respiratory distress and required invasive mechanical ventilation. Continuous renal replacement therapy (CRRT) was discontinued because of extreme instability in vital signs. Large doses of vasoactive drugs were needed to maintain blood pressure. Computer tomography(CT)of the chest and abdomen was performed because of the patient's persistent abdominal pain. On abdominal CT (shown in Fig. 1), multilinear air densities in the portal vein, hepatic branches, and mesenteric vessels, intestinal obstruction, intestinal necrosis, and left perirenal exudation were seen. Right middle lung infection and pulmonary oedema were also seen on her chest CT. **Diagnostic:** Septic shock and severe abdominal infection were diagnosed after discussion with multiple departments. **Follow-up and outcomes:** As the patient could not tolerate surgery due to her condition, the family members were unwilling to take risks for surgical treatment. Despite fluid resuscitation (23 h rehydration 9000 mL), high-dose vasopressors, and intravenous antibiotic therapy, the patient was pronounced dead 20h after admission to our hospital.

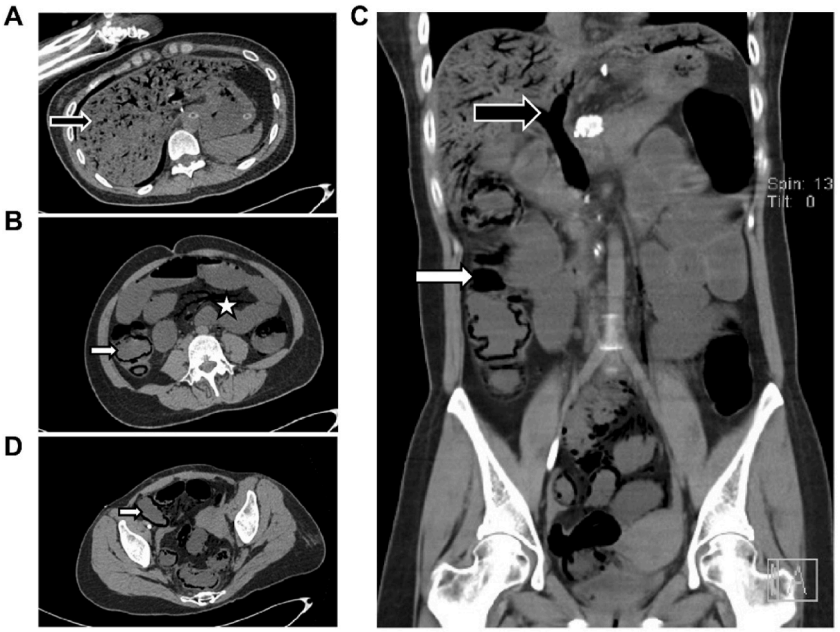

Fig. 1. CT image of HPVG, mesenteric venous gas, pneumatosis intestinalis: The black arrow shows HPVG, the right arrow shows pneumatosis intestinalis, and the star shows mesenteric vein gas.

3. Methods

A review of the published literature on HPVG was conducted using the PubMed MEDLINE database, and a search strategy with the prespecified keyword "hepatic portal venous gas" and "glyphosate poisoning" was adopted. According to the keyword search, we identified rare cases where glyphosate poisoning caused HPVG. In the PubMed MEDLINE database, we adopted "hepatic portal venous gas," "adult, " and "case report" as the search strings, which identified 289 items.

4. Results

Among these case reports, >40 types of diseases have been reported to cause HPVG (as shown in Table 2), including intestinal ischaemia, mesenteric artery thrombosis, mesenteric venous thrombosis, mesenteric artery occlusion/embolism, mesenteric ischaemia, mesenteric infarction, diffuse arterial intestinal ischaemia, nonocclusive mesenteric ischaemia, bowel infarction, bowel necrosis, necrotizing enteritis, stomach necrosis, gastric ischaemia, perforation/fistula, enteritis, gastric intubation, gastric/intestinal dilatation, intestinal obstruction, abdominal infection, thrombophlebitis, digestive ulcer, pancreatitis, Crohn's disease, trauma, diverticulitis, intestinal infection, complications of endoscopic procedures, ulcerative colitis, splenic flexure carcinoma, gastric emphysema, dives, idiopathic, ischemic enteritis, dialysis, drug and poisoning, chemotherapy, graft-versus-host disease, and cholangitis. In total 40 cases of HPVG caused by gastrointestinal ischaemia were identified (Table 3). Among these 90 patients, 46.7 % were surgically treated. The mortality rate was 31.8 %, percentage of surviving patients who received surgery was 41.8 %, and rate of death among those who were treated by surgery was 25.7 % (Table 4). Nelson found that the mortality rate of HPVG was 29%–33 % [3]. Drugs that can cause HPVG include α-glucosidase inhibitors [4] and aspirin [5]. The underlying diseases associated with HPVG are

Table 2
Review of HPVG with the different disease types.

The types of diseases	Total (%)	Survived (%)	Surgical (%)
Gastrointestinal ischaemia	$n_1 = 90$		
Intestinal ischaemia	15 (5.19)	4 (26.7)	5 (33.3)
Mesenteric ischaemia	15 (5.19)	8 (53.3)	2 (13.3)
Nonocclusive mesenteric ischaemia	14 (4.08)	13 (92.9)	7 (50.0)
Bowel necrosis	9 (3.11)	9 (100.0)	8 (88.9)
Mesenteric artery occlusion/embolism	8 (2.77)	3 (37.5)	4 (50.0)
Ischaemic enteritis	7(2.42)	6 (85.7)	4 (57.1)
Bowel infarction	4 (1.38)	2 (50.0)	2 (50.0)
Necrotizing enteritis	4 (1.38)	2/2 (100.0)	2/2 (100.0)
Diffuse arterial intestinal ischaemia	3 (1.04)	0 (0.0)	3 (100.0)
Thrombophlebitis	3 (1.04)	3 (100.0)	1 (33.3)
Stomach necrosis	3 (1.04)	1 (33.3)	1 (33.3)
Mesenteric artery thrombosis	2 (0.69)	1 (50.0)	1 (50.0)
Mesenteric infarction	2 (0.69)	1 (50.0)	1 (50.0)
Mesenteric venous thrombosis	1 (0.35)	1 (100.0)	1 (100.0)
Non-gastrointestinal ischaemia	$n_2 = 199$		
Complications of endoscopic procedures	20 (6.92)	15/18 (83.3)	2/18 (11.1)
obstruction Intestinal	16 (5.54)	11/14 (78.6)	9/13 (69.2)
Crohn's disease	15 (5.19)	14/14 (100.0)	5/14 (35.7)
Abdominal infection	14 (4.48)	13 (92.9)	5 (35.7)
Gastric/intestinal dilatation	14 (4.84)	12/13 (92.3)	3/12 (25.0)
Perforation/fistula	12 (4.15)	9 (75.0)	8 (66.7)
Trauma	9 (3.11)	8/8 (100.0)	4/8 (50.0)
Diverticulitis	9 (3.11)	9 (100.0)	7 (77.8)
Pancreatitis	8 (2.77)	4 (50.0)	0 (0)
Chemotherapy	8 (2.77)	5 (62.5)	1 (12.5)
Tumour	8 (2.77)	5/6 (83.8)	0/6 (0.0)
Intestinal infection	6 (2.08)	4 (66.7)	1 (16.7)
Drug and poisoning	6 (2.08)	5/5 (100.0)	1/5 (20.0)
Enteritis	4 (1.38)	4 (100.0)	0 (0.0)
Gastric ischaemia	4 (1.38)	4 (100.0)	1 (25.0)
Ulcerative colitis	4 (1.38)	1/1 (100)	0/1 (0.0)
Splenic flexure carcinoma	4 (1.38)	2/2 (100.0)	2/2 (100.0)
Gastric emphysema	4 (1.38)	3 (75.0)	4 (100.0)
Gastric intubation	4 (1.38)	3 (75.0)	1 (25.0)
Digestives ulcer	3 (1.04)	2/2 (100.0)	1/2 (50.0)
Dives	3 (1.04)	3 (100.0)	0 (0.0)
Idiopathic	3 (1.04)	3 (100.0)	0 (0.0)
Graft-versus-host disease	3 (1.04)	3 (100.0)	1 (33.3)
Cholangitis	3 (1.04)	3 (100.0)	1 (33.3)
Dialysis	2 (0.69)	1/1 (100.0)	0/1 (0.0)
Others	13 (4.50)	7 (53.8)	5 (38.5)

Table 3
Different outcomes and treatments according to the gastrointestinal ischaemia status.

	Total (N)	Surgical (%)	Survived (%)
Gastrointestinal ischaemia	90	42 (46.7)	54 (60.0)
Non-gastrointestinal ischaemia	199	62 (31.2)	153 (76.9)

Table 4
HPVG with different outcomes after different treatments and with different underlying diseases.

	Total (N)	Survived (N)	Death (N)
Surgical treatment	102	85 (41.8 %)	17 (25.3 %)
Conservative treatment	168	118 (58.1 %)	50 (74.6 %)
Age			
60 y ≥ Age >12 y	96	83	13
70 y ≥ Age >60 y	58	47	11
Age >70 y	90	62	28
Gender			
Female	90	76	14
Male	155	116	39
Hypertension	45	26	19
Diabetes	44	30	14
Tumour	40	32	8
Abdominal surgery history	33	27	6
Coronary heart disease	28	17	11
End-end kidney disease	22	13	9
Hyperlipidaemia	14	8	6
COPD	13	8	5
Stroke	13	8	5
Atrial fibrillation	11	4	7
History of thrombosis	9	6	3
Immunosuppression	9	9	0
Chronically bedbound	3	2	1

hypertension, diabetes mellitus, tumor, history of abdominal surgery, coronary heart disease, end stage renal disease, hyperlipidemia, COPD, stroke, atrial fibrillation, history of embolism, immunosuppression, and prolonged bed rest. Some patients can have concurrent multiple underlying diseases. Hypertension, diabetes, and tumors are the most common underlying diseases (Table 4).

5. Discussion

5.1. The characteristics and mechanisms of HPVG

HPVG was first described by Wolfe and Evens in infants with necrotizing enterocolitis (NEC) in 1955 [6]. HPVG has been gradually recognized as a radiologic sign and cannot be used as a predictor of death alone. The centrifugal flow of the portal venous blood causes peripheral transport, and the characteristic of HPVG findings on abdominal plain radiography is a branching radiolucency extending to within 2 cm beneath the liver capsule. Regarding the pathogenesis of HPVG, two mechanisms have been proposed. One is that an elevated intramural pressure by bowel distension enables the entrance of intraluminal gas into the venous circulation in the damaged intestinal mucosa. Mucosal damage and bowel distention are important facts in this theory [7]. Mechanical factor-induced mucosal injury is not always accompanied by infection and is consequently associated with a low probability of intestinal perforation, and therefore intervention by surgery is not the preferred method [8]. Another mechanism is that gas-forming bacteria invade the submucosa and then enter the portal venous system [8]. Bacteria such as *Enterobacter* and *Klebsiella* species, *Clostridium perfringens*, and *Klebsiella oxytoca* produce gas can cause HPVG [9]. Even when evidence of bowel obstruction or ischaemia is not present, translocation of bacteria into the portal circulation is considered a primary mechanism.

5.2. Ominous HPVG

The occurrence of HPVG with intestinal ischaemia usually indicates an ominous intra-abdominal pathology [10]. Both the superior mesenteric artery occlusion and nonocclusive mesenteric ischaemia (NOMI) can cause bowel necrosis with a mortality rate reportedly as high as 75%–90 %. In addition to looking for HPVG, the presence of intramural gas and gas in the mesenteric arcades should also be looked for. The CT features include bowel distention, gas in the superior mesenteric vein, and pneumatosis intestinalis. Pneumatosis cystoides intestinalis coexisting with HPVG is highly suggestive of transmural bowel infarction [11]. NOMI includes all forms of mesenteric ischaemia without occlusion of the mesenteric arteries and accounts for 20%–30 % of all cases of acute mesenteric ischaemia [10]. In our review, the rate of HPVG caused by NOMI was 4.08 %. NOMI is caused by insufficient blood flow to meet the metabolic demands of visceral organs [12]. Predisposing conditions such as peripheral vascular disease, congestive heart failure,

obstructive pulmonary disease, atrial dysrhythmia, and insulin-dependent diabetes mellitus are reportedly associated with splanchnic hypoperfusion when ultrafiltration is used. Five of fourteen patients with NOMI were undergoing dialysis, one patient had bleeding, and several had severe atherosclerosis of the inferior mesenteric artery. The gold standard in diagnosing peripheral splanchnic vasospasm remains angiography [10]. Since the delayed treatment of NOMI can cause intestinal necrosis with poor prognosis, early surgical intervention is usually considered [13]. Among the 14 patients who were diagnosed with NOMI, only one patient died, and half of the patients were surgically treated.

Gastric ischaemia can also cause HPVG. Gastric ischaemia can be caused by diffuse or localized vascular insufficiency, which can be seen in sepsis, thromboembolism or acute thrombosis, local vasculitis, gastric volvulus or gastric dilatation; however, many cases are idiopathic with no clear etiology [14]. Endoscopy is the gold standard for diagnosis and can detect early ischemic changes [14]. Gastric ischaemia impairs gastric motility, causing stomach dilatation, which exacerbates ischaemia [14]. When no evidence of peritonism or hemodynamic compromise is present, Iain Rankin suggests that a "watch and wait" approach may be useful [15]. A low threshold for surgical intervention should be adopted if the condition of the patient does not improve or if clinical deterioration occurs [15]. The conservative management includes gastric acid suppression, decompression with a nasogastric tube, and antibiotics.

In some cases, HPVG was accompanied by gastric wall gas. Gastric emphysema and emphysematous gastritis are different. Patients with gastric emphysema are nontoxic and usually asymptomatic. Conversely, emphysematous gastritis is a rare form of gastritis due to the invasion of the gastric wall by gas-forming organisms [16] and always rapidly progresses to a state of septic shock [17]. A CT scan is the best imaging modality for the differentiation between both entities, as air within the stomach wall in cases of emphysematous gastritis causes a streaky and linear consistency compared with the round air bubbles seen in gastric emphysema [18].

When necrotizing pancreatitis is accompanied by HPVG, this usually indicates concurrent mesenteric ischaemia [19]. Faberman and Mayo-Smith [20] proposed that HPVG was caused by the degradation of the intestinal mucosa by pancreatic enzymes, and the presence of intestinal ischaemia allows air to translocate from the bowel lumen into the portal venous system in cases of sepsis. This requires early detection combined with aggressive surgical management.

HPVG combined with thrombophlebitis of the portal or mesenteric veins can be regarded as an indicator of poor prognosis [21]. Pylethrombophlebitis is defined as the combination of infection and thrombosis within the portal veins [22]. The management is administration of appropriate antibiotics and resection (or drainage) of the primary septic source [23].

The sources of HPVG in diverticulitis are either direct communication between the intestinal lumen and the portomesenteric vein system because of necrotizing vasculitis in the wall of an intramesocolic abscess or septic thrombophlebitis of the inferior mesenteric vein complicated by gas-forming microorganisms [24]. The two underlying mechanisms seem to have different effects on the symptoms and mortality of the condition [24]. The prognosis is favorable if an underlying intramesocolic abscess is present or is perforation occurs in complicated diverticulitis; however, the prognosis of HPVG due to septic thrombophlebitis and gas-forming organisms is poor [24].

A reported 6 % of HPVG cases are associated with intra-abdominal abscesses [7], including those in the pelvis, retroperitoneum, tubo-ovarian [25], pyelonephritis, and subphrenic areas. When intra-abdominal abscesses are accompanied by diffuse peritonitis, this may cause bacteria to colonize the mesenteric portal circulation [26].

Two cases of prolonged cardiopulmonary resuscitation (CPR) associated with PI and HPVG were identified in the literature review. The pathogenic mechanism was probably bowel infarction caused by poor mesenteric perfusion during and after CPR [27], especially in elderly patients with severe atherosclerosis. Reuter et al. [28] proposed a different pathogenic mechanism. During CPR, in addition to mechanical compression, bag-mask ventilation with improper neck positioning before tracheal intubation could cause gastric inflation and bowel distension [28].

5.3. Benign HPVG

With the development of radiological techniques, benign HPVG has been observed in numerous diseases, including inflammatory bowel disease, acute gastric dilatation, blunt abdominal trauma, jejunostomy, catheter insertion, and other isolated cases [29]. In several cases, this has been described because of diagnostic or therapeutic invasive procedures such as surgery, hepatic artery embolization, colonoscopy [30], and operative endoscopic procedures [31]. When this is isolated or asymptomatic, the evolution of portal venous gas may be a typically benign process [32]. Close observation in intensive care units is currently a priority over immediate hyperbaric oxygen therapy [33].

HPVG in patients with inflammatory bowel disease, such as ulcerative colitis and Crohn's disease (CD), can be caused by mucosal damage alone or can occur in combination with bowel distension, sepsis, or invasion by gas-producing bacteria [21]. Approximately 58 % of cases of HPVG associated with CD are iatrogenic due to colonoscopy, barium enema, or blunt abdominal trauma [34]. Enterovenous fistula, which is an extremely rare complication of CD, can directly transfer bowel gas to the portal venous system. Lim et al. [21] concluded that HPVG caused by an enterovenous fistula is an indication for urgent surgery. Based on our research, the mortality rate among patients with CD and HPVG appears to be relatively high. Surgical intervention is not always conducted, especially in the absence of peritoneal signs or free intraperitoneal gas [21].

HPVG can also be caused by gastric dilatation, especially when the patient has ileus, barium enema, or blunt abdominal trauma. If a sudden increase in gastric pressure occurs, gastric ischaemia, necrosis, and perforation can occur [35]. In patients with acute gastric dilatation without vascular or obstructive pathology, the presence of HPVG and even gastric mucosal ischaemia should not be considered as a particular indication for emergency surgery [36]. If gastric dilatation is present due to endoscopy, inserting a Levin tube for gastric decompression plays an essential role in the rapid removal of portal vein gas [37].

HPVG due to blunt abdominal trauma was initially reported to signal vascular injury and bowel necrosis [38]. However, cases of

benign HPVG with blunt abdominal trauma have been reported [39]. The most likely explanation is that a sudden increase in intra-abdominal pressure, accompanied by concomitant mucosal disruption, and this may force the intraluminal gas into the bowel wall, where it is absorbed into the portal circulation [40].

Associations have also been reported between HPVG and several chemotherapeutic agents and molecular-targeted agents, such as cyclophosphamide, doxorubicin, paclitaxel, docetaxel, bevacizumab, cisplatin, sunitinib, sorafenib, bevacizumab, and gefitinib [41]. HPVG is triggered by chemotherapy-induced enterocolitis, causing small bowel dilatation and oedema with resultant intestinal mucosal damage [42].

HPVG after endoscopic retrograde cholangiopancreatography is a rare complication [43]. This can occur because of a vascular laceration during precut sphincterotomy or because of a porto-biliary fistula, which is possibly associated with tumor infiltration or inflammatory-related conditions [44].

Percutaneous liver biopsy can cause benign HPVG. Transgression of the portal vein with the entry of ambient air during biopsy is the likely cause of portal venous gas [45]. Portal venous gas from a diving injury is an infrequent finding [32]. Gas embolism is one of the main causes of diving injury and can occur after decompression sickness. HPVG condition could also be associated with anastomotic leakage and the management of this surgical complication should also solve the portal venous gas problem [46].

5.4. The prognosis and treatment of HPVG

The prognosis of HPVG is related to the pathology itself and is not influenced by the presence of HPVG [47]. While patients without a lethal condition can be conservatively treated, distinguishing between patients with nonlethal cases of HPVG and those who require surgery is difficult [48]. Iannitti et al. [49] suggested that surgery should be recommended with frank peritonitis, with additional CT findings (complete small bowel obstruction, mesenteric ischaemia), with certain recent interventions (e.g., vascular surgery procedures), or with a complicated medical diagnosis (complicated infectious or inflammatory process). Koami et al. [50] found that using criteria of low blood pressure (<systolic BP 108 mm Hg), high lactate dehydrogenase (>387 U/L), and the presence of pneumatosis intestinalis led to 100 % sensitivity and 78 % specificity for necrotic bowel. Hani et al. [51] found that older age, peritonitis signs, and elevated BUN are most highly associated with ischaemia and necrotic bowel. A study emphasized the importance of the extent of HPVG and the existence of intestinal pneumatosis [52]. Ikegame [53] recommended that follow-up CT after a few days seems to be the most useful. When the physical findings associated with the need for emergent surgery are not observed, HPVG or the mesenteric vein gas quickly disappears in a few days. In patients with HPVG, another important factor contributing to survival and prognosis is the existence of long-term chronic diseases, such as chronic renal failure, diabetes mellitus, and hypertension [54]. Long-term chronic diseases can reportedly decrease immune function and alter the intestinal microbial flora and tolerance ability of patients with HPVG, which may lead to fatality [53,55].

5.5. HPVG caused by herbicide

As a nonselective herbicide, glyphosate is widely used in agriculture. Commercial glyphosate-based formulations most commonly range from concentrations of 1 %–41 % and generally comprise an aqueous mixture of the isopropylamine (IPA) salt of glyphosate, a surfactant, and various minor components. The mechanisms of toxicity of glyphosate formulations are complicated and unknown; both glyphosate and its surfactants can cause human poisoning, and IPA may be involved in the uncoupling of oxidative phosphorylation [56]. The clinical features of this herbicide poisoning are varied. Ingestion of >85 mL of a concentrated formulation may cause significant toxicity in adults, and the symptoms can include gastrointestinal corrosive effects on the mouth and throat, epigastric pain, dysphagia, renal and hepatic impairment, respiratory distress, impaired consciousness, shock, arrythmia, metabolic acidosis, and hyperkaliemia. Patients who orally ingest glyphosate should be treated with gastric lavage and catharsis in the early stages, as well as blood purification when necessary [56]. Gastrointestinal corrosion is common in patients who ingest glyphosate. Talbot et al. [57] reported that the ingestion of glyphosate surfactant caused gastrointestinal erosion and hemorrhage in 66 % and 8 % of patients, respectively. Another study [58] revealed that corrosive esophageal, gastric, and duodenal injuries were present in 68 %, 72 %, and 16 % of patients, respectively. Herein, we described a rare case in which accidental ingestion of glyphosate was shown to lead to HPVG. After reviewing the causes of HPVG, we hypothesize that the main mechanisms are corrosive intestinal mucosal damage caused by the herbicide. In this case, X-ray–opaque substances were not visible in the intestinal tract on CT images as compared with a case of an elderly man who ingested 1 L of glyphosate-based herbicide, where CT images revealed a radiopaque fluid accumulation from the stomach to the jejunum [59]. In our case, although most of the glyphosate may have been removed by gastric lavage in our patient, the gastrointestinal mucosa is more susceptible to glyphosate damage. Furthermore, the sudden increase in intramural pressure due to the gastric lavage exacerbated the intestinal insufficiency. Finally, intestinal ischaemia and gas-forming bacteria invading the submucosa caused septic shock. Thus, even when glyphosate is removed by gastric lavage, this can still cause intestinal complications and a poor prognosis.

This report has some limitations. First, we did not perform blood or urine tests to detect the blood glyphosate concentration of the patient, and a postmortem examination was not performed. Another limitation is that an abdominal CT was not performed on the patient on admittance to our hospital. If patients show HPVG in the early stages of admission, intervention should be performed as early as possible.

6. Conclusion

HPVG itself cannot be an independent disease and is a radiology sign that is associated with a disease that can range from ominous intestinal ischaemia to an iatrogenic situation. The treatment of HPVG does not depend on the extent of HPVG but depends on the underlying primary etiology and the existence of underlying clinical conditions. When the condition of a patient worsens or the etiology is intestinal ischaemia, it is strongly recommended to conduct laparotomy exploration. The herbicide glyphosate can cause erosion of the digestive tract, and attention should be paid to the pressure of gastric lavage to avoid the rapid expansion of the digestive tract and the translocation of intestinal bacteria resulting in HPVG, septic shock, and even death.

Ethical Statement

The study has been approved by the Ethics Committee of Affiliated Hospital of Guizhou Medical University in accordance with the Helsinki Declaration.

Ethical approval statement

We completed the guideline of case report standards provided by the Ethics and Editorial Policies of Heliyon. We have obtained written informed consent from the authorized proxy for the publication of this case report, any accompanying data and images. We confirm that all content presented in this case report, including associated data and images, have been anonymized to the best possible extent.

Data availability statement

Data supporting this study are included within the article.

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Yingxia Wu: Supervision. **Yijie Zhang:** Methodology. **Jiangquan Fu:** Writing – original draft. **Feng Shen:** Validation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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