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Predictors of prognosis in children with portal venous gas detected by ultrasound

Takahiro Hosokawa[1], Yoshitake Yamada[2], Yutaka Tanami[1], Yumiko Sato[1], Mayumi Hosokawa[3], Eiji Oguma[1]

[1]Department of Radiology, Saitama Children's Medical Center, Saitama, [2]Department of Radiology, Keio University School of Medicine, Tokyo, [3]Department of Pediatrics, Saitama City Hospital, Saitama, Japan

Abstract

Aims: To evaluate ultrasound findings in order to determine potential predictors of prognosis in pediatric patients with portal venous gas (PVG) detected by ultrasound. **Materials and methods:** Thirty-nine children were included and divided into two groups: benign PVG (n=24) and life-threatening PVG (n=15; 6 surgical interventions and 9 deaths). Possible predictors, i.e., the location of PVG in the liver, the distribution of intestinal pneumatosis, ascites and free air were compared between the two groups. **Results:** A significant difference was noted between the two groups in terms of the distribution of intestinal pneumatosis (limited to the large bowel, benign vs life-threatening = 60.9% (14/23):21.4% (3/14), p=0.040), the absence of ascites (benign vs life-threatening = 79.1% (19/24):40.0% (6/15), p=0.019) and patient age (benign vs life-threatening = 52.5±65.3 months vs 19.7±44.0 months, p=0.019). No significant difference was observed in the location of PVG in the liver, the presence of free air, and sex between the two groups. Conclusions: In pediatric patients with PVG, including various ages and underlying diseases, intestinal pneumatosis limited to the large bowel and absence of ascites were predictors of a benign prognosis. However, despite the presence of these predictors, some patients with PVG required surgical intervention, thereby suggesting that the cause of PVG, such as necrotizing enterocolitis, volvulus, or pancreatitis, must be also carefully evaluated.

Keywords: portal venous gas; intestinal pneumatosis; ascites; ultrasound

Introduction

Portal venous gas (PVG) is defined as the presence of gas in the portal vein [1-4]. This finding has been linked to certain intestinal disorders and studies have associated the presence of this sign with a patient mortality rate of 30-75% [4-7]. However, recently, PVG was detected in not only pediatric patients with life-threatening conditions, such as ischemia or necrotizing enterocolitis (NEC) [1,8,9], but also in those without life-threatening conditions such as patients who received post-hematologic stem cell transplantation, immunosuppressive therapy, chemotherapy and those with bowel inflammation, food allergies and disease or trauma of the hepatobiliary system [9-15].

PVG can be identified in patients using abdominal radiography, computed tomography (CT) and ultrasound. A recent study reported that the detection of PVG by ultrasound was superior to that by CT [16]. In addition, some case reports described that PVG was only detected by ultrasound and not by CT [17-20]. In pediatric patients, CT imaging requires exposure to radiation and possible sedation. In contrast to CT, ultrasound imaging can be performed in real time without the need for radiation

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Corresponding author: Yoshitake Yamada, MD, PhD
Department of Radiology, Keio University School of Medicine, 35 Shinanomachi, Shinjuku-ku, Tokyo 160-8582, Japan
Phone: +81-3-3353-1977
Fax: +81-3-3353-1977
E-mail: yamada@rad.med.keio.ac.jp

exposure or sedation; additionally, it can be readily and repeatedly performed at the patient's bedside [9,21,22].

Until now, few studies have evaluated ultrasound findings that may be effective predictors in children with PVG and these few reports included only cases of neonates with NEC [23,24]. To the best of our knowledge, no clinical studies have evaluated ultrasound findings to determine predictors of prognosis in pediatric patients with various diseases in whom PVG was detected by ultrasound. Therefore, in this study, we evaluated the ultrasound findings to determine potential predictors of prognosis in pediatric patients with PVG detected by ultrasound.

Materials and methods

Patients

The Ethics Committee of our institution approved this retrospective study and the requirement for informed consent was waived. We reviewed medical records spanning a 9-year period from April 2010 to September 2018 and identified 42 children who were suspected to have PVG based on ultrasound findings. Of the 42 children with PVG detected by ultrasound, two were excluded from the study, as the radiologists could not arrive at a consensus on the presence of PVG during the review process. Another patient with an umbilical vein catheter in the portal vein and in whom an air bubble was introduced by this catheter, was excluded as well. Finally, 39 children were included in current study. Our cohort was divided into two groups: benign PVG and life-threatening PVG. The benign PVG group included patients who did not undergo surgical interventions such as laparoscopic or open surgery for intestinal ischemia or obstruction, and the life-threatening PVG group included patients who underwent surgical interventions or those who died.

Evaluation of predictors of disease outcome using ultrasound

All sonograms were obtained using a scanner with a high-resolution (9-15MHz) linear transducer (LOGIQ 7, E9 and S8, GE Healthcare, Waukesha, WI, USA). In addition, all sonograms were performed by four radiologists with experience ranging from 10-20 years.

According to previous studies [4,7-13,23,24], we evaluated the following four factors: 1) location of PVG in the liver (segmental or diffuse; the former represents PVG that is located only in the left/right lobe of the liver; 2) distribution of intestinal pneumatosis (limited to the large bowel or not; the former represents intestinal pneumatosis located only in the large bowel or PVG originating from the inferior mesenteric vein alone; 3) ascites (absence or presence); and 4) free air (absence

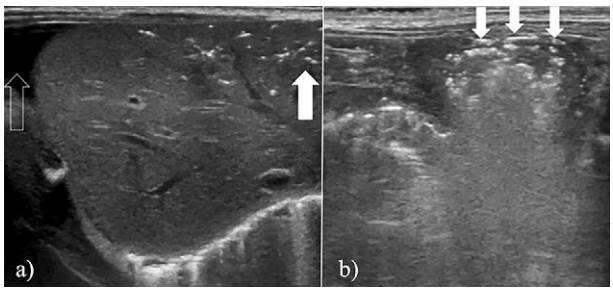

Fig 1. A 1-month-old male patient with portal venous gas (PVG) with a confirmed diagnosis of necrotizing enterocolitis after patent ductus arteriosus closure: a) Gray-scale sonogram shows hyperechoic foci in the liver parenchyma due to the segmental accumulation of gas bubbles in the left lobe (white arrow). Ascites is detected around the liver (frame arrow); b) Gray-scale sonogram reveals hyperechoic foci in the wall of the small intestine (white arrow). The distribution of pneumatosis intestinalis was categorized as not limited to the large bowel.

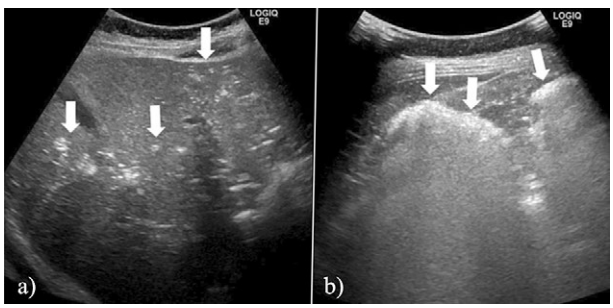

Fig 2. An 9-year-old female patient with portal venous gas (PVG) with a confirmed diagnosis of enteritis: a) Gray-scale sonogram shows hyperechoic foci in the liver parenchyma due to the accumulation of gas bubbles. PVG is located diffusely in the liver (white arrow); b) Gray-scale sonogram reveals hyperechoic foci in the wall of the sigmoid colon. Intestinal pneumatosis is limited to the large bowel (white arrow).

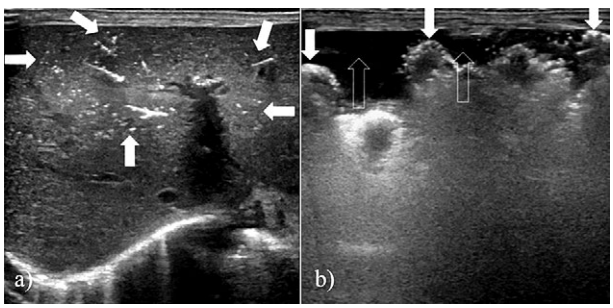

Fig 3. A 1-month-old male patient with portal venous gas (PVG) with a confirmed diagnosis of necrotizing enterocolitis following aortic coarctation and pulmonary artery banding: a) Gray-scale sonogram reveals hyperechoic foci in the liver parenchyma due to the accumulation of gas bubbles. PVG is diffuse in the liver (white arrow); b) Gray-scale sonogram reveals hyperechoic foci in the wall of the small intestine. Intestinal pneumatosis is not limited to the large bowel (white arrow). Ascites is detected around the small intestine (frame arrow).

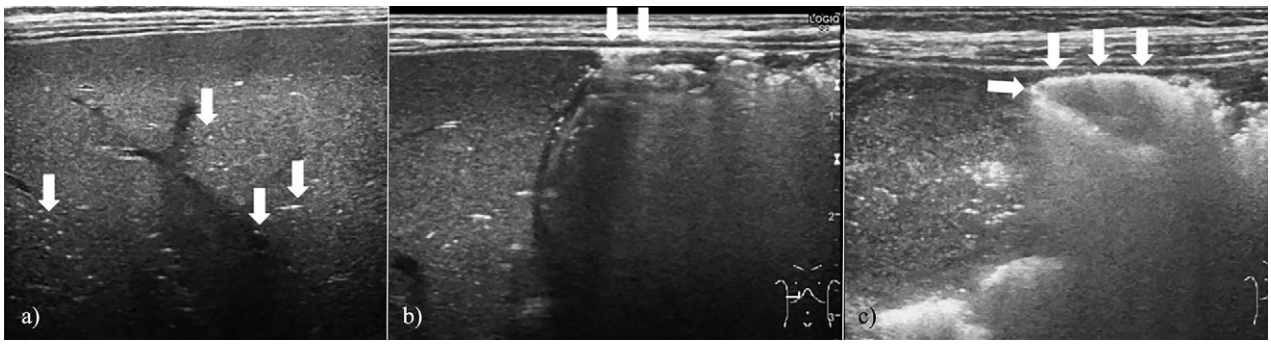

Fig 4. A 9-month-old male patient with portal venous gas (PVG) with a confirmed diagnosis of intestinal necrosis: a) Gray-scale sonogram shows hyperechoic foci in the liver parenchyma due to the accumulation of gas bubbles. PVG is located diffusely in the liver (white arrow); b) A hyperechoic line, revealed near the small intestine (white arrow), is diagnosed as extraluminal air; c) Gray-scale sonogram reveals hyperechoic foci in the wall of the small intestine; intestinal pneumatosis is not limited to the large bowel (white arrow).

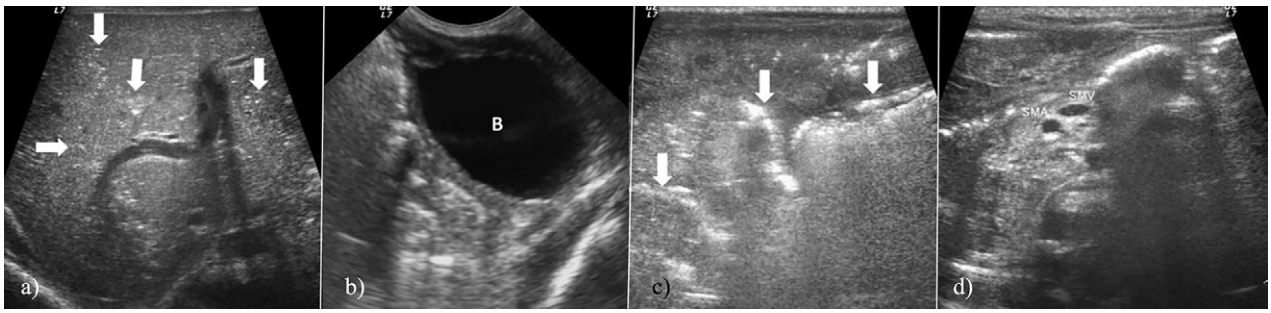

Fig 5. A 10-day-old male patient with portal venous gas with a confirmed diagnosis of volvulus with midgut malrotation: a) Gray-scale sonogram shows hyperechoic foci in the liver parenchyma due to the diffuse accumulation of gas bubbles in the liver (white arrow); b) Ascites is not detected by gray-scale sonography (B; bladder); c) Gray-scale sonogram reveals hyperechoic foci in the wall of the small intestine; intestinal pneumatosis is not limited to the large bowel (white arrow); d) A transverse sonogram showing reversal of the positions of the mesenteric vessels. The superior mesenteric vein (SMV) is located to the left of the superior mesenteric artery (SMA).

or presence). We exemplified in figures 1-5 some of the encountered situations.

Review process

We reviewed the four previously described factors detectable by ultrasound. Two radiologists, with 15 and 10 years of clinical experience, respectively, reviewed all the images on a 1600×1200 picture archiving and communication systems monitor (PACS, GE Healthcare, Waukesha, WI, USA) and the observations were noted by consensus. During the review process, the radiologists were blinded to the patient's surgical, physical, and other imaging findings.

Statistical analysis

Data are presented as mean±standard deviation. Fisher's exact test was used to compare the location of PVG in the liver, distribution of intestinal pneumatosis, and the presence of ascites and free air between the two groups. Additionally, the Mann-Whitney U test was used to compare patient age, and Fisher's exact test was used to compare patient sex between the two groups. The significance level for all tests was set at $p<0.05$ (two-sided). All data were analyzed using a commercially available software program (JMP version 12; SAS Institute Inc, Cary, NC, USA).

Results

Patient characteristics are summarized in Table I (27 males and 12 females). The mean age was 39.9±59.6 months (range: 0-171 months).

Of the 39 patients, 24 were included in the benign PVG group and 15 were included in the life-threatening PVG group (6 surgical interventions and 9 deaths). A summary of the two groups, including observations pertaining to the four predictors, is presented in Table II. Two cases that lacked ultrasound findings to determine the distribution of intestinal pneumatosis were excluded from the analysis of intestinal pneumatosis distribution.

Table I. Patient characteristics

No.	Sex	Age (months)	Location of PVG in the liver*	Distribution of PI**	Ascites***	Free air***	Prognosis	Diagnosis	Pre-history
1	M	0	Segmental (LL)	NL to the large colon	Absent	Absent	Benign	Paralytic ileus	Small intestinal atresia
2	M	5	Diffuse	NL to the large colon	Absent	Absent	Benign	Food allergy	CBD dilatation
3	M	13	Diffuse	NL to the large colon	Absent	Absent	Benign	Food allergy	TGA, BAS
4	M	170	Diffuse	NL to the large colon	Absent	Absent	Benign	Enteritis	None
5	M	171	Diffuse	NL to the large colon	Absent	Absent	Benign	Enteritis	Cholecystectomy
6	M	0	Segmental (LL)	NL to the large colon	Present	Absent	Benign	NEC	PDA closure
7	M	8	Diffuse	NL to the large colon	Present	Absent	Benign	Enteritis	Strangulated ileus, SBS
8	M	23	Diffuse	NL to the large colon	Present	Absent	Benign	Enteritis	VP shunt
9	M	149	Diffuse	NL to the large colon	Present	Absent	Benign	Intestinal ischemia	Heatstroke
10	F	0	Diffuse	Large colon only	Absent	Absent	Benign	Food allergy	None
11	M	0	Diffuse	Large colon only	Absent	Absent	Benign	Food allergy	None
12	M	1	Diffuse	Large colon only	Absent	Absent	Benign	Food allergy	None
13	M	2	Diffuse	Large colon only	Absent	Absent	Benign	Food allergy	None
14	F	3	Diffuse	Large colon only	Absent	Absent	Benign	Food allergy	None
15	F	22	Segmental (RL)	Large colon only	Absent	Absent	Benign	Food allergy	NEC, SBS
16	M	24	Segmental (RL)	Large colon only	Absent	Absent	Benign	Enteritis	NEC
17	F	25	Segmental (RL)	Large colon only	Absent	Absent	Benign	Food allergy	NEC, SBS
18	F	49	Diffuse	Large colon only	Absent	Absent	Benign	Enteritis	BMT
19	M	61	Diffuse	Large colon only	Absent	Absent	Benign	Enteritis	CHARGE
20	M	72	Segmental (LL)	Large colon only	Absent	Absent	Benign	Intestinal necrosis	Strangulated ileus
21	F	119	Diffuse	Large colon only	Absent	Absent	Benign	Enteritis	BMT
22	F	165	Segmental (LL)	Large colon only	Absent	Absent	Benign	Enteritis	Strangulated ileus, SBS
23	F	168	Diffuse	Large colon only	Absent	Absent	Benign	SLE enteritis	SLE
24	F	1	Segmental (LL)	N/A	Present	Absent	Benign	Paralytic ileus	Duodenal atresia
25	M	0	Diffuse	NL to the large colon	Absent	Absent	LT (surgery)	Volvulus	None
26	M	0	Diffuse	NL to the large colon	Absent	Absent	LT (surgery)	Volvulus	None
27	M	0	Diffuse	NL to the large colon	Absent	Absent	LT (death)	NEC	PAA, VSD
28	M	9	Diffuse	NL to the large colon	Absent	Present	LT (death)	Intestinal necrosis	Septic shock
29	M	0	Diffuse	NL to the large colon	Present	Absent	LT (death)	Intestinal necrosis	Septic shock
30	M	0	Diffuse	NL to the large colon	Present	Absent	LT (death)	Intestinal necrosis	OTCD
31	M	0	Diffuse	NL to the large colon	Present	Absent	LT (surgery)	NEC, Intestinal stenosis	Small intestinal atresia
32	M	1	Diffuse	NL to the large colon	Present	Absent	LT (death)	NEC	CoA complex, PA banding
33	F	6	Diffuse	NL to the large colon	Present	Absent	LT (surgery)	Adhesion ileus	MMIHS
34	F	25	Diffuse	NL to the large colon	Present	Absent	LT (death)	DIC	DIC
35	M	73	Diffuse	NL to the large colon	Present	Absent	LT (death)	Pancreatitis	Ebstein's anomaly
36	M	163	Segmental (RL)	Large colon only	Absent	Absent	LT (death)	Pancreatitis	Esophageal atresia
37	M	0	Segmental (LL)	Large colon only	Present	Absent	LT (surgery)	NEC	TGA, BAS
38	F	3	Diffuse	Large colon only	Present	Absent	LT (surgery)	Intestinal necrosis	Volvulus, NEC
39	M	15	Diffuse	N/A	Absent	Absent	LT (death)	Intestinal necrosis	Small intestinal atresia, SBS

*, "Location of PVG in the liver" is classified as segmental or diffuse, in which the former represents PVG that is located in the left/right lobe (L/RL) of the liver segment alone.
**, "Distribution of intestinal pneumatosis" is classified as being limited to the large bowel alone or not (NL), in which the former represents intestinal pneumatosis located only in the large bowel or PVG originating from the inferior mesenteric vein alone.
***, "ascites" and "free air" are classified as the absence or presence.
Abbreviations: PVG, portal venous gas; LT, Life-threatening; PI, intestinal pneumatosis; N/A, not applicable; NEC, necrotizing enterocolitis; CBD, common bile duct; VP shunt, ventricular peritoneal shunt; SBS, short bowel syndrome; CHARGE, Coloboma of iris, Heart disease, Atresia choanae, Retarded growth and mental development, Genital hypoplasia and Ear anomalies and deafness; SLE, systemic lupus erythematosus; OTCD, ornithine transcarbamylase deficiency; CoA, coarctation of the aorta; PAA, pulmonary artery atresia; VSD, ventricular septum defect; PA banding, pulmonary artery banding; PDA, patent ductus arteriosus; MMIHS, megacystis microcolon intestinal hypoperistalsis syndrome; DIC, disseminated intravascular coagulation; TGA, transposition of the great arteries; BAS, balloon atrial septostomy; BMT, bone marrow transplantation.

Overall, a significant difference was observed with regard to the distribution of intestinal pneumatosis (benign vs life-threatening = 60.9% vs 21.4%), the absence of ascites (benign vs life-threatening = 79.1% vs 40.0%) and age (benign vs life-threatening = 52.5±65.3 months vs 19.7±44.0 months) between the two groups. No significant difference was observed in the location of PVG in the liver (p=0.263), presence of free air (p=0.385) or patient sex (p=0.305) between the two groups.

Discussions

Through the use of ultrasound in this study, we found that the presence of intestinal pneumatosis limited to the large bowel and the absence of ascites were predictive factors for a benign prognosis in pediatric PVG patients of various ages and with different underlying diseases. The location of PVG in the liver, presence of free air or patient sex did not appear to be predictive of the patient's disease outcome. On the other hand, in the current study, some patients with ascites and intestinal pneumatosis not limited to the large colon did not require surgical intervention and they recovered after only receiving medical treatment. Additionally, despite the absence of ascites, two patients, who presented with intestinal pneumatosis not limited to the large colon due to volvulus with midgut malrotation, were determined to have a life-threatening prognosis and needed surgical intervention. In consideration of these cases, when examining children with PVG, the sonographer should carefully seek not only intestinal pneumatosis location and ascites, but also the cause of PVG; additionally, the physician may need to consider each patient's condition and select appropriate treatment on a case-by-case basis.

Previous studies that focused on intestinal pneumatosis using CT, identified the following predictors of poor prognosis: disease not limited to the large colon and the presence of ascites [11,12]. Additionally, in a previous study that focused on neonates with NEC, ascites was identified to be a predictor of poor prognosis [24-26]. The results of the current study, which used ultrasound and included children of various ages, are similar to those previously reported. In children with this disease, ultrasound is generally the first form of examination to be performed; therefore, these study findings may facilitate appropriate and rapid treatment management.

Although the presence of free air was statistically not associated with patient outcome in our limited study cohort, one patient who presented with this finding underwent surgical intervention (life-threatening). Previous studies have described how this finding was important when deciding to perform a surgical intervention [26],

Table II. Predictors of prognosis in the benign PVG and life-threatening PVG groups

	Benign PVG	Life-threatening PVG	p value
Total	n = 24	n = 15	-
Location of PVG in the liver (n = segmental/diffuse)	n = 8/16	n = 2/13	0.263
Distribution of intestinal pneumatosis* (n = limited to the large bowel/not limited)	n = 14/9	n = 3/11	0.040
Ascites (n = present/absent)	n = 5/19	n = 9/6	0.019
Free air (n = present/absent)	n = 0/24	n = 1/14	0.385
Age (Mean±SD)(range, months)	52.5 ± 65.3 (0–171)	19.7 ± 44.0 (0–161)	0.019
Sex (n=male/female)	n = 15/9	n = 9/6	0.305

* Two cases were excluded due to the lack of information available regarding the location of intestinal pneumatosis when detected by ultrasound.
PVG, portal venous gas

and our result with regard to free air was consistent with that of previous reports.

The mean age of the life-threatening PVG group was less than that of the benign PVG group (p=0.019). This difference may have been attributed to the underlying causes of PVG, such as midgut volvulus and NEC, which occurred during the neonatal period [27,28].

The current study had some limitations. First, the number of patients included in the study was low; thus, additional studies including a larger number of patients are required to confirm our preliminary findings. Second, the quality of ultrasound imaging obtained is dependent on the skill of the operator and the patient's condition. In our study, operators who were comparatively experienced in ultrasound performed the examination; despite this, the distribution of intestinal pneumatosis could not be evaluated in some cases.

Conclusion

In pediatric PVG patients of various ages and with different underlying diseases, the presence of intestinal pneumatosis limited to the large bowel and the absence of ascites were predictors of a benign prognosis. We did find that ultrasound, which can be easily performed at the bedside, may be helpful in predicting benign disease outcomes. However, PVG occurred in various diseases that required specific treatment, such as surgical intervention. Therefore, the cause of PVG, such as necrotizing enterocolitis, volvulus, or pancreatitis, should be also carefully evaluated.

Conflict of interest: none

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