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Non-inflammatory or non-ischemic vascular gas on emergent multi-detector computed tomography: Eight years' experience

Kazuya Sugimori, Izumi Torimoto, Kyota Nakamura, Masaaki Kondo, Kazushi Numata, Shigeo Takebayashi

ABSTRACT

The study aimed to characterize the etiology and clinical significance of non-inflammatory or non-ischemic vascular gas on multi-detector computed tomography (MDCT). We reviewed MDCT images and clinical charts of patients with vascular gas excluding inflammatory or ischemic entities in our hospital between 2008 and 2015. The local cases and the case report papers, which were extracted from English literature in PubMed were summarized according to iatrogenic or non-iatrogenic causes to analyze etiology for the entry of air into the circulation. Our local series demonstrated single or multiple collection of vascular gas in 15 patients including one with systemic arterial gas; the most frequent was cerebral vascular gas (CVG, n = 11, 0.8–12 mL) followed by hepatic vascular gas (n = 10, 0.4–256 mL). The accumulative 144 cases including the 15 local cases included 62 (43.1%) with iatrogenic vascular gas; the most frequent was central venous catheter-related CVG (48 cases) with 39.5% mortality followed by hepatic portal venous gas (20 cases) with 15% mortality. A careful search for clues on MDCT images was useful in discussing the etiology of vascular gas entry points and increased awareness of the emergent clinical settings where the vascular gas occurred.

Keywords: Brain, Iatrogenic complication, Portal vein, Vascular gas

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INTRODUCTION

Multi-detector computed tomography (MDCT), which allows a thin-slice scan and has been used emergently in patients in various clinical settings, is useful in the detection of small amounts of vascular gas. A post-processing algorithm using MDCT data that can generate multiplanar images allows assessment of anatomical location of vascular gas [1]. Unlike arterial gas, small venous gas produces no clinical signs and symptoms. However, the embolism can easily occur when gas enters the cerebral vein because the air blood interaction causes the development of a network comprising air bubbles and fibrin strands that intersperses with aggregates of the platelets, red blood cells, and fat globules [2]. We encountered vascular gas in various clinical situations, including iatrogenic complications. The morbidity

and mortality associated with vascular gas resulting in embolism can be prevented through a better understanding of its pathophysiology and increased awareness of various clinical settings where they occur. Emergency departments are especially high-risk locations for venous air embolism because of frequent use of venous access (central and peripheral) and intravenous drug and fluid infusions [3].

We present this article analyzing the etiology of air entry into the veins on emergent MDCT scanning in our experienced cases as well as extracted published cases by systematic literature reviews.

MATERIALS AND METHOD

Approval for this retrospective study was obtained from our institutional review board, and a waiver for informed consent was obtained.

Local cases

One of the authors reviewed our MDCT reports database between 2008 and 2015 to select patients with vascular gas excluding contaminated air at intravenous injection of contrast material. Clinical characteristics, imaging findings, treatment and outcome were collected. MDCT scan of the trunk or the head were performed using a 16-row MDCT unit (Aquilion16; Toshiba Medical Systems, Ohtawara, Japan). The trunk from the neck to the pelvis was scanned with 16×1 mm collimation (16 detectors with 1-mm section thickness), a beam pitch of 0.9375, a rotation speed of 0.5 second, a table speed of 15.0 mm per rotation and a tube current determined by automated tube modulation. The head images were acquired with 16x0.5 mm collimation, a beam pitch of 0.6875, a rotation speed, of 1 second, a table speed of 5.5 mm per rotation and 120-kVp tube current. Images were reconstructed at the MDCT console to a section thickness of 5 mm. Each image was sent to a picture archiving and communication system (PACS, Synapse ver. 3.1, Fuji-Film Medical Co., Tokyo, Japan). The area of each axial scan of venous gas was determined on the monitor by tracing the margin of the gas with a built-in cursor controlled by a track ball. Section volumes were then calculated by multiplying by section thickness (1 mm in the truncal scan and 0.5 mm in head scan) and were added to determine the total gas volume. Coronal reconstructed images were obtained with a 1.0 mm thickness and a reconstruction interval of 0.8 mm. Areas of air-containing pixels of axial images were multiplied by the reconstruction interval of 0.8 mm between contiguous images and all of these subvolumes were added.

Literature search

Cases were extracted from English literature in PubMed (NCBI) for the following search term: “venous gas”, “venous gas embolism”, “arterial gas”, and “arterial gas embolism” in combination with specific organs in our local cases. The case report papers and our local cases were summarized according to causes. In addition, we analyzed the etiology of air entry into the vessel, hypothesis of traveling gas to the vessels and its clinical significance to increase awareness of the clinical settings where the vascular gas occurred.

RESULTS

Our local series demonstrated single or multiple collections of vascular gas in 15 patients who were three with iatrogenic vascular gas (Table 1) and 12 with non-iatrogenic gas (Table 2); the most frequent was cerebral vascular gas (CVG, n = 11, 0.8–12 mL) followed by hepatic vascular gas (n = 10, 0.4–256 mL). A total of 20 case reports, including one review article with 165 included cases, were extracted by PubMed search terms related to vascular gas in combination with “cerebral” or “hepatic”. The majority of literature reported on single patients. MDCT or computed tomography scan was performed in 129 of the 165 patients. We selected the 129 patients, including 59 with iatrogenic-caused vascular gas (Table 3) and 70 with non-iatrogenic-caused vascular gas (Table 4).

Of the accumulative 144 cases which were 129 published reporting cases and 15 local cases, 62 (43.1%) including 3 local cases were iatrogenic and 82 (56.9%) including 12 local cases were non-iatrogenic. The most frequent entity of iatrogenic vascular gas in the central venous catheter-related CVG (48 cases, 77.4%) with 39.5% mortality. Our local series had a patient with CVG-related cerebral embolism in which no intracranial gas but 1.2 mL of the left ventricular gas was shown in Figure 1. In this patient, the presence of the left ventricular gas supported the paradoxical embolism although no intra-cardiac right to left shunt was shown in the color Doppler imaging.

In the local case series, massive (>100 mL) hepatic portal venous gas was observed in the remaining two patients with iatrogenic complications. The patient experienced cardiac arrest during the endoscopic procedure with balloon dilatation. Post-resuscitation MDCT images (Figure 2) demonstrated multiple collections of gas in the right ventricle, the pulmonary arterial trunk, infrarenal inferior vena cava, the ascending thoracic aorta and cerebral vessels. The barotrauma during the endoscopic procedure with balloon dilatation for bowel is a possible entity of massive hepatic portal venous gas, according to four published reporting cases. Massive hepatic portal venous gas associated with cystic venous gas in another local case occurred due to decompression sickness with sub-atmospheric intraperitoneal pressure and the entry of gas into injured gastric wall vein via a mal-positioned gastrostomy tube

(Figure 3B). One published case reported massive hepatic portal venous gas in decompression sickness with injured jejunum. Other etiologies of iatrogenic cerebral gas in published cases included percutaneous lung biopsy or radiofrequency ablation.

Aggressive resuscitation was the most frequent (66.7%, n = 8) cause of vascular gas in non-iatrogenic local cases: intracranial gas in three patients, hepatic vascular gas in three patients and both in two patients. MDCT images did not provide the conclusive evidence of cerebral or hepatic vascular anatomies. MDCT images of craniofacial trauma in the local series showed gas in the overt cerebral vein (Figure 4C). Non-iatrogenic hepatic portal venous gas occurred in patients with acute colonic pseudo-obstruction in two local cases. The local case series included gas replacement of systemic arteries caused by positive pressure ventilation in the patient with aortobronchial fistula secondary to the aneurysmal rupture. MDCT in the patient showed conclusive images of intrahepatic arterial gas (Figure 3A) and cerebral arterial gas (Figure 4A–B).

Table 1: Local cases with iatrogenic-caused vascular gas on MDCT

Etiology	No. of cases	Vascular gas on MDCT	Volume (mL) of gas on MDCT	Hypothesis for traveling gas to the vein	No. of death
Central venous catheter manipulation	1	Left ventricle	1.2	Paradoxical embolism	0
Barotrauma during endoscopic procedure with balloon dilatation	1	HPV Cerebral vessel Right ventricle Ascending thoracic aorta Pulmonary arterial trunk Infrarenal IVC Pneumoretroperitoneum	256 2.4 16.3 3.1 1.7 2.7 Massive	Transmitted gas via portal vein-hepatic artery capillary anastomosis at rapid flow Intra-cardiac right-left Shunt?	1
Malposition of gastrostomy tube	1	HPV	161	Intraperitoneal subatmospheric pressure	0
Decompression disease		Cholecystic vein Pneumoperitoneum	10.4 Massive		

HPV: Hepatic portal vein, IVC: Inferior vena cava

Table 2: Local cases with non-iatrogenic caused vascular gas on MDCT

Etiology	No. of cases	Vascular gas on MDCT	Volume (mL) of gas on MDCT	Hypothesis for traveling gas to the vein	No. of death
Craniofacial trauma	1	Cerebral vein	1.65	Communicating with paranasal sinus	1
Aggressive resuscitation	8	Indeterminate cerebral vessel	0.6–3.4	Retrograde movement	7
		Indeterminate hepatic vessel	0.4–1.2		
Acute colonic pseudo-obstruction	2	HPV	0.11–0.18	Barotrauma increased pressure	0
Ruptured aortic dissection*	1	Systematic artery, Left ventricle, atrium	Massive	Positive pressure	1
		Pulmonary artery		Ventilation in aortobronchial fistula	

HPV: Hepatic portal vein *Our published case report [4]

Table 3: Selected published case reports with iatrogenic caused venous gas on MDCT

Etiology	Authors	No. of cases	Vascular gas on MDCT	Hypothesis for traveling gas to the vein	No. of death(s)
Central venous catheter manipulation	Phino et al. [5]*,	46	Cerebral gas bubbles	Paradoxical embolism	18
	Yesilaras, et al. [6]	1	Pneumocranium	Retrograde movement	1
Lung biopsy/ ablation	Bou-Assaly et al. [7]	1	Cerebral artery	Sub-atmospheric pressure in the pulmonary vein	0
	Jeannin [8]		Coronary artery		1
	Mokhes et al. [9]	1	Left ventricle, Atrium, Aorta, Coronary artery	Communicating pulmonary artery	0
	Rapicetta et al. [10]	1			0
		1	Pulmonary vein, Left atrium ,Ventricle, Coronary artery		
Barotrauma during endoscopic procedure with balloon dilatation	Lee et al. [11]	2	HPV	Damage of intestinal mucosa combined with bowel dilatation	0
	Eoh et al. [12]	1	Cerebral artery		0
	Ma et al. [13]	1	HPV		0
	Pandurangadu et al. [14]	1	Cerebral vessel		0
Laparoscopy and decompression sickness	Shatz et al. [15]	1	HPV, Pneumoperitoneum Pneumatosis intestinalis	Perforated jejunum Sub-atmospheric pressure	0
Oxygen-supply tube connected to intravenous catheter	Makino [16]	1	HPV Pneumothorax, Pneumoperitoneum, pneumoretroperitoneum	Transmitted gas via portal vein-hepatic artery capillary anastomosis	1
Connection between the endotracheal tube and oxygen flow meter	Yang et al. [17]	1	Systemic artery Intracranial artery pneumocranium	Massive gas entering the pulmonary vein	1

HPV: Hepatic portal vein

Table 4: Selected published case reports with non-iatrogenic- caused non-inflammatory, non-ischemic vascular gas on MDCT

Etiology	Authors	No. of cases	Vascular gas on MDCT	Mechanism of venous gas	No. of death(s)
Craniofacial trauma	Pampin-Huerta et al. [18]	2	Cerebral vessel	Communicating with paranasal sinus	2
Aggressive resuscitation	Shiotani et al. [19]	25	Cerebral vein	Retrograde movement	25
	Arata et al. [20]	27	Hepatic vein		27
	Shah et al. [21]	4	Hepatic vein		4
Diver decompression sickness	Morita et al. [22]	4	HPV Mesenteric vein IVC	Sub-atmospheric pressure	1
Acute colonic pseudo-obstruction (Ogilvie's syndrome),	Inokuchi et al. [23]	7	HVP pneumatosis intestinalis	Barotrauma	0
	Kearns et al. [24]	1		Increased pressure	0

HVP: Hepatic portal vein, IVC: Inferior vena cava

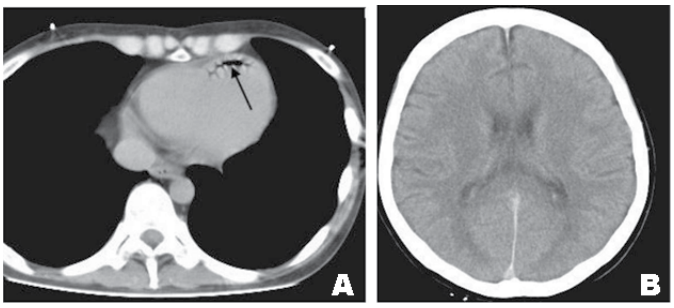

Figure 1: Central venous catheter- related cerebral arterial paradoxical embolism. (A) Left ventricular gas (arrow) on MDCT. (B) No gas was shown on head CT, but symmetrical hyperintensity was shown in bilateral occipital lobes on diffusion weighted magnetic resonance image (not shown).

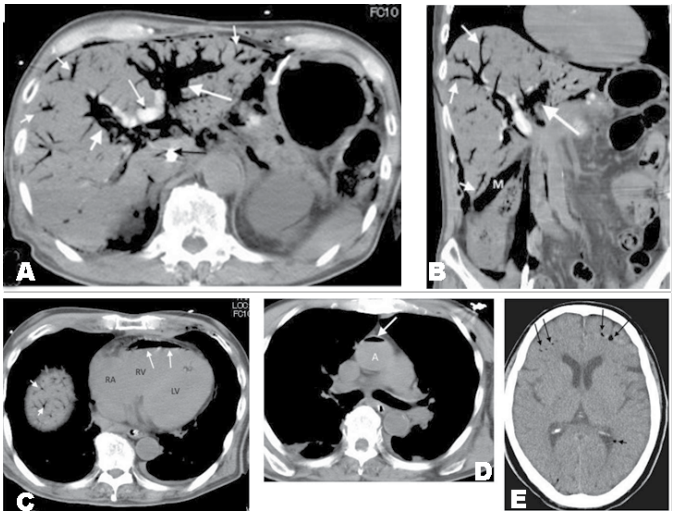

Figure 2: Post-resuscitation MDCT images of various locations of vascular gas in a patient with cardiac arrest during duodenal endoscopic procedure with balloon dilatation. (A) Axial image. Small short arrows: gas in peripheral branches of hepatic portal veins. Large long arrow: the left main trunk. Large short arrow: the right main trunk. Small long arrow: contrast material in the biliary track. Black arrow: central venous catheter in the inferior vena cava. (B) Coronal reconstructed image. Hepatic portal vein is replaced by massive collections of gas Large long arrow: the left main trunk, Small short arrows: peripheral branches. M: Morrison's pouch. (C) Gas (long arrows) in the right ventricle (RV), LV: left ventricle. RA: right atrium. Short arrows: gas in the peripheral portal veins (D) Gas (arrow) in the ascending aorta (A). (E) Cerebral subarachnoid gas bubbles (arrows) in undetermined vessel.

DISCUSSION

The entry of gas into the circulation is mostly an iatrogenic problem but may also occur in many fields of medicine including emergency medicine. The entry of gas into the blood stream requires a pressure gradient favoring the passage of gas into the blood vessel. This occurs when venous pressure is negative relative to

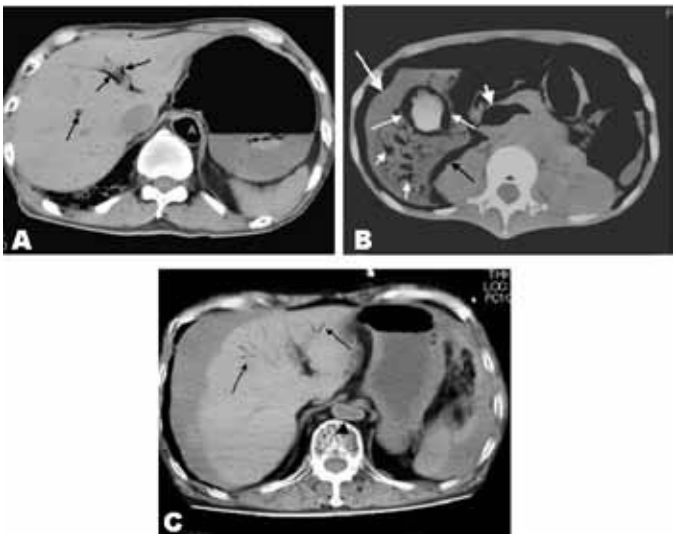

Figure 3: Various locations of intrahepatic vascular gas on MDCT. (A) Hepatic arterial gas (arrows) in a patient with a ruptured aortic arch aneurysm and aortobronchial fistula under positive ventilation. A: the abdominal aorta replaced by gas (B) Hepatic portal venous gas (small short arrows) and fluid pneumoperitoneum (large long arrow) due to mal-positioned gastrostomy tube and injured gastric wall with a pressure gradient under atmosphere. Small long arrows: gas in the cystic vein joining the right portal vein (normal variation). Large short arrow: extrahepatic portal vein Black arrow; the Morrison's pouch. Note scanty of retroperitoneal fat. (C) Post-resuscitation intrahepatic gas (arrows) in undetermined vessels.

atmospheric pressure or when gas is forced under pressure directly or indirectly into the bloodstream [3, 16]. A 14 G catheter enables the gas to flow at a rate of about 100 mL/sec, with a pressure difference of only 5 cm H_2O [16]. Entering gas into the circulation occurs by barotrauma caused by positive pressure ventilation and insufflation of gas into the peritoneal cavity during endoscopy can also cause gas to enter the circulation [13].

The most frequently iatrogenic vascular gas was central venous catheter-related cerebral gas embolisms. Approximately, 70% of the patients with gas embolisms had intracranial gas bubbles, which were most frequently located in the subarachnoid space [5]. MDCT provides no conclusive anatomy with regards to the gas in the subarachnoid space although it can depict a small gas bubble. Therefore, the gas bubble was interpreted by some authors as intra-arterial caused by paradoxical embolism and by others as intra-venous caused by retrograde embolism [6]. Hagen et al. [25] demonstrated in a large number of autopsy specimens of human hearts that 27.3% had foramen ovales of 1 to 10 mm in diameter. Increasing right atrium pressure from entering gas is suggested to cause right to left shunting in the fossa ovale which is usually occluded [26]. In the majority

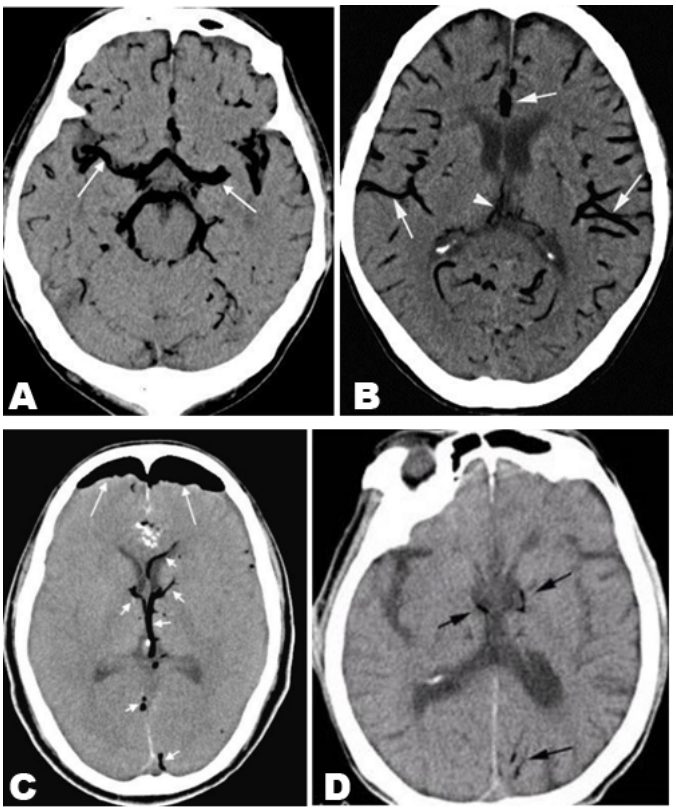

Figure 4: Various locations of cerebral vascular gas on MDCT. (A) Gas replacement of systemic arteries in cardiac arrest patients with ruptured aortic arch aneurysm and aortobronchial fistula under positive ventilation. At the level of Willis ring. Arrows: the middle cerebral arteries (B) Same case to (A), At the level of lateral ventricular body. Arrow in the anterior portion: the anterior cerebral artery. Arrows in bilateral portions: the temporal arteries. Arrowhead: the medial posterior choroid artery (C) Gun-shot wound patient with cerebral venous gas (short arrows from the anterior to posterior: the anterior vein of septum pellucidum, the internal cerebral vein, the straight sinus and the superior sagittal sinus) communicating with pneumocranium (long arrows) and paranasal sinus. (D) Post-resuscitation patient with cerebral gas (arrow) in undetermined vessels.

of published cases, there was no reference to shunt investigation. The presence of the intra-cardiac right-left shunting on transesophageal echocardiogram is not always shown in patients with paradoxical embolisms. In a local case with central venous catheter trouble, the presence of left ventricular gas supported a paradoxical embolism, although no intra-cardiac right to left shunt was shown in the echocardiogram. Paradoxical arterial embolism is an acceptable mechanism because many cases reported central venous catheter-related coronary arterial gas embolism. But Yeslaris et al. [6] claimed that retrograde gas embolism possibly occurred through the internal jugular vein into intracranial veins when massive amounts of air entered the jugular vein via a large bored central venous catheter. Retrograde gas movement through the internal jugular vein to the intracranial venous sinus is considered as the mechanism of post-resuscitation cerebral gas or hepatic gas [20, 21] Post-resuscitation cerebral and hepatic gas are explained by the retrograde gas movements; at rapid infusion of large fluid volumes, contaminated air via the jugular or femoral central venous catheter is allow to enter the cerebral vein or hepatic vein, respectively [21].

Hepatic portal venous gas has been described in non-obstructive dilatation of bowels in acute colonic pseudo-obstruction (Ogilvie's syndrome) and Crohn's disease [23, 24]. Massive amounts of hepatic portal venous gas occurred due to the barotrauma during endoscopic procedure with balloon dilatation. The increased intra-luminal pressure causes mucosal tears within the bowel, which allows gas to enter submucosal veins and flow to the hepatic portal vein. A large amount of hepatic portal venous gas is not dangerous because the gas is not rapidly or easily transmitted to the central circulation. However, it may enter the inferior vena cava via the portal vein-hepatic artery capillary anastomosis when the gas is forced to be infused at a rapid and massive flow. The mechanical obstruction of the right ventricular pulmonary outflow tract and pulmonary vasculature occurs when a large amount of gas enters the right ventricle. Hepatic portal gas was observed in 20 cases with a 15% mortality rate, in the published case reports and our local case series.

CONCLUSION

Gas embolus does produce clinical signs and symptoms which may mimic an acute cardiopulmonary or cerebrovascular event. The spectrum of vascular gas causes detected with MDCT is widening. Radiologists as well as emergency department physicians, should be familiar with the increasing number of vascular gas causes in addition to knowledge of the patient's clinical history. A careful search for clues on MDCT images was useful in discussing the etiology of vascular gas entry points and increased awareness of the emergent clinical settings where the vascular gas occurred.

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Author Contributions

Kazuya Sugimori – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Izumi Torimoto – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Kyota Nakamura – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Masaaki Kondo – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published
Kazushi Numata – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published
Shigeo Takebayashi – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor
The corresponding author is the guarantor of submission.

Conflict of Interest
Authors declare no conflict of interest.

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