Bilateral Intracardiac Microbubbles in a Patient With Giant Hiatus Hernia: A Case Report

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Abstract

Intracardiac microbubbles may occur inadvertently during a cardiac procedure, which are typically reported in patients with central venous catheters or cardiac prosthetic valves. Here, we report a case wherein a microbubble filling in the bilateral atriums and ventricles was revealed during echocardiography despite the patient not having the aforementioned risks. An 87-year-old man with hypertension was admitted with a diagnosis of heart failure caused by a giant hiatal hernia. While awaiting hernia surgery, he started vomiting and suddenly went into a coma. A contrast-enhanced computed tomography (CT) scan of the abdomen showed a thickening of the gastric wall, intramural gas, and portal vein gas. Considering these findings, a giant esophageal hiatus hernia was suspected as the cause of the intracardiac microbubbles. In addition, an echocardiogram showed a patent foramen ovale, and the magnetic resonance imaging (MRI) of the head showed multiple cerebral infarctions bilaterally in the cerebral hemispheres. Therefore, a paradoxical air embolism was suspected to cause the coma in this patient. A giant esophageal hiatus hernia can cause portal vein gas triggered by an increased intragastric pressure (which causes vomiting). Then, the portal vein gas flows into the right heart via the sinusoids. Cerebral air embolism can also develop via a shunt, such as a patent foramen ovale, and trigger a foreign body reaction via inflammation and cause coma. When microbubbles are observed in the heart on an echocardiogram, it is necessary to seek the place of entry because it can be a lethal sign due to complications that could follow, such as a cerebral air embolism or pulmonary air embolism.

Introduction

Iatrogenic detection of microbubbles is common in patients with central venous lines, certain medical devices, or cardiac prosthetic valves [1]. They have been associated with intestinal ischemia and colon cancer as well and may be generated by absorbed intestinal gas that reaches the heart through the portal system and systemic vein shunts [1-3]. The routes for the air in the heart can vary based on abnormal anatomy of shunts such as a patent foramen ovale, aorta-esophageal, and vena cava-duodenal fistula [1-3].

Here, we report a case of an 87-year-old patient with hypertension, in whom the microbubbles occurred in the right heart system unexpectedly. The microbubbles circulated throughout the body via a right-to-left shunt. The echocardiography showed the microbubble filling up the cardiac cavities bilaterally.

Case Presentation

An 87-year-old man presented to the hospital with a complaint of anorexia for two weeks. He had been well until two weeks prior to the consultation, when he had a coffee-ground emesis. On his first visit to our hospital, he was diagnosed with gastroesophageal reflux disease and macrocytic anemia, and lansoprazole and folic acid were administered. Although his vomiting improved, he developed anemia and his family doctor referred him to our hospital. In the past two weeks, he gained 5 kg in weight and developed edema in both legs. He had no complaints of dyspnea, chest pain, fever, or chills. His medical history included hypertension and gastroesophageal reflux disease, and no history of smoking. His medications at the time of admission included ferrous citrate, folic acid, lansoprazole, mosapride, and bifidobacteria. There were no abnormal vital signs except for tachypnea at 24 beats per minute.

On physical examination, the jugular vein was distended, and the chest auscultation revealed bilateral crackles. There was bilateral pitting edema in the lower extremities. Blood tests revealed a decreased hemoglobin of 8.2 g/dL, albumin of 2.5 g/dL, and elevated brain natriuretic peptide (BNP) level of 448 pg/mL. Other laboratory test results are shown in Table 1.

| Marker       | Day 1 | Day 17 | Range |
|--------------|-------|--------|-------|
|              |       |        |       |

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|                      | Admission | Day 17 | Normal Range                  |
|----------------------|-----------|--------|------------------------------|
| **White blood cells**| 4.5       | 5.3    | 3.5–9.1 × 10^3/μL            |
| Neutrophils          | 78.7      | 76.4   | 44.0–72.0%                   |
| Lymphocytes          | 7.7       | 17.1   | 18.0–59.0%                   |
| Monocytes            | 12        | 5.9    | 0.0–12.0%                    |
| Eosinophils          | 0.7       | 0.4    | 0.0–10.0%                    |
| Basophils            | 0.5       | 0.2    | 0.0–3.0%                     |
| Red blood cells      | 2.37      | 2.74   | 3.76–5.50 × 10^6/μL          |
| Reticulocytes (%)    | 4.2       |        | 0.2–2.0%                     |
| Hemoglobin           | 8.2       | 9.1    | 11.3–15.2 g/dL               |
| Hematocrit           | 25.4      | 28.1   | 33.4–44.9%                   |
| Mean corpuscular volume | 107.2    | 102.4  | 79.0–100.0 fl                |
| Platelets            | 19.8      | 16.0   | 13.0–36.9 × 10^4/μL          |
| **Total protein**    | 4.8       | 5      | 6.5–8.3 g/dL                 |
| **Albumin**          | 2.5       | 2.4    | 3.8–5.3 g/dL                 |
| **Total bilirubin**  | 0.3       | 0.3    | 0.2–1.2 mg/dL                |
| Aspartate aminotransferase | 23    | 22     | 8–38 IU/L                    |
| Alanine aminotransferase | 16      | 21     | 4–43 IU/L                    |
| Alkaline phosphatase | 88        | 71     | 106–322 IU/L                 |
| γ-glutamyl transpeptidase | 35       | 26     | <48 IU/L                     |
| Blood urea nitrogen  | 17.3      | 29.1   | 8–20 mg/dL                   |
| Creatinine           | 0.71      | 0.57   | 0.40–1.10 mg/dL              |
| Serum sodium         | 134       | 131    | 135–150 mEq/L                |
| Serum potassium      | 4.8       | 3.2    | 3.5–5.3 mEq/L                |
| Serum chloride       | 101       | 92     | 98–110 mEq/L                 |
| Serum calcium        | 7.8       | 8      | 3.5–10 mg/dL                 |
| **C-reactive protein** | 0.61   |        | <0.30 mg/dL                  |
| Ferritin             | 39.5      |        | 14.4–303.7 ng/mL             |
| Brain natriuretic protein | 448   |        | <40 pg/mL                     |
| Troponin I           | 0.018     |        | <0.02 ng/mL                  |
| D-dimer              | 4.1       |        | <1 μg/mL                      |
| pH                   |           | 7.458  |                              |
| PaO₂                 | 56.4      |        | 80–100 mmHg                   |
| PaCO₂                | 41.6      |        | 35–45 mmHg                    |
| Serum bicarbonate    | 29.5      |        | 22–26 mmol/L                  |
| Lactate              | 3.1       |        | 0.26–1.39 mmol/L              |

**TABLE 1:** Laboratory data on admission and on day 17 of hospitalization.

PAO₂: partial pressure of oxygen; PACO₂: partial pressure of carbon dioxide
An electrocardiogram (ECG) revealed a sinus rhythm and complete right bundle branch block. Chest radiography showed a bilateral costophrenic angle blunt (Figure 1).

![Chest X-ray at admission](image)

**FIGURE 1: Chest X-ray at admission**

The bilateral costophrenic angles were dull.

The patient was admitted to the hospital and treated for acute heart failure with intravenous furosemide at 40 mg/day. The patient responded to treatment over several days, with weight loss and an improvement in both leg edema and tachypnea; however, his anorexia did not improve.

On the fifth day of hospitalization, a computed tomography (CT) scan was performed to investigate the cause of his anorexia, which revealed a giant esophageal hiatus hernia (Figure 2).
FIGURE 2: Abdominal CT scan on the fifth day
The CT revealed a giant esophageal hiatal hernia.

CT, computed tomography

FIGURE 3: Abdominal CT scan on the fifth day
The giant esophageal hiatus hernia compressed the heart anteriorly (Figure 3).

CT, computed tomography
There were no other abnormalities in the abdominal cavity that could cause anorexia or vomiting. We concluded that his anorexia was caused by a giant hiatus hernia, and the compression of the hernia in the atrium caused heart failure. Surgery was planned for the repair of a giant esophageal hiatal hernia one week later. However, he could not progress to the surgery.

On the 17th day, the patient suddenly went into a coma after vomiting. Regarding his consciousness, his Glasgow Coma Scale (GCS) score was 6. His respiratory rate was 24 breaths per minute, pulse was 85 beats per minute, blood pressure was 110/80 mmHg, and SpO2 was 85%, while breathing oxygen through an oxygen mask at a rate of 4 L/minute. On physical examination, the pupils were 3 mm in diameter, equal, and reacted to light, and the jugular vein was not distended. The lungs were clear on auscultation, and the cardiac auscultation revealed no murmur. Blood gas analysis showed a lactate level of 3.1 mmol/L; other laboratory data are shown in Table 1. Only transthoracic echocardiography was performed to investigate the hypoxemia and elevated lactate levels, which revealed microbubbles in the bilateral cardiac atriums and ventricles with a tiny foramen ovale (Figure 4).

FIGURE 4: Echocardiography on the 17th day

The echocardiography revealed microbubbles in bilateral cardiac cavities.

The contrast-enhanced CT showed portal vein gas (Figure 5) in the liver gastric wall thickening (Figure 6).
FIGURE 5: Contrast CT scan on the 17th day
The CT revealed portal vein gas in the liver.
CT, computed tomography

FIGURE 6: Contrast CT scan on the 17th day
The CT revealed gastric wall thickening.
CT, computed tomography

There were no contrast defects in the main pulmonary artery trunk or the intestinal wall. A head MRI on the
Giant esophageal hiatus hernia can cause portal vein gas and intracardiac microbubbles triggered by point of entry should be found, which suggests gastrointestinal ischemia or increased internal pressure; may be due to the characteristics of such air embolization.

Microbubbles were observed bilaterally in the cardiac cavities. This suggests that the bubbles from the right ventricular system entered the left ventricular system. Usually, bubbles in the right ventricle are trapped in the pulmonary capillaries after passing through the pulmonary artery and would not flow into the left ventricle. There are several mechanisms underlying this, of which one is a right-to-left shunt through the heart or lungs, such as a patent foramen ovale. For example, the Valsalva maneuver can transiently increase right atrial pressure, creating a right-to-left shunt and transferring the potential embolic source into the systemic circulation. Similarly, increasing pulmonary artery pressure caused by a pulmonary embolism also increases right-to-left shunt flow. In this case, transthoracic echocardiography showed blood flow between the right and left atria, suggesting a patent foramen ovale. Vomiting (observed in this case before the onset of the coma) might have caused an increased intrathoracic pressure and accelerated shunt flow. The other mechanism involves the volume of the microbubbles. When it is a large volume of microbubbles, a passage of air emboli from the hepatic vein to the inferior vena cava was observed, suggesting that the bubbles passed through the sinusoids to the inferior vena cava.

Finally, the microbubbles that entered the systemic circulation had reached the brain through the aorta, causing a cerebral air embolism. More than 5% of cases of cerebral infarction are speculated as caused by a patent foramen ovale and called as a paradoxical embolism. In cerebral infarction, the clinical presentation is determined by the areas of the brain that are affected, and complete disorientation is rare. However, cerebral infarction due to an air embolism is known to cause coma. This may be related to the pathophysiology of the microbubble embolization. In addition to ischemia (due to vascular occlusion), an inflammatory response occurs because the body reacts with the bubble as a foreign substance. Both processes result in vasogenic edema, and neuronal injury extends beyond the site of obstruction. In this case, the degree of disorientation was much more significant than the imaging findings, which may be due to the characteristics of such air embolization.

The learning points from this case report are that if right ventricular air is present on echocardiography, the point of entry should be found, which suggests gastrointestinal ischemia or increased internal pressure; giant esophageal hiatus hernia can cause portal vein gas and intracardiac microbubbles triggered by...
increased intragastric pressure due to vomiting; if echocardiography shows bubbles in both heart cavities, an intracardiac shunt (such as an open foramen ovale) should be sought; and if echocardiography shows intracardiac microbubbles in patients a comatose condition, a cerebral air embolism should be included in the differential diagnosis.

Conclusions

Microbubbles entering the venous system can travel to the systemic vein, which can then enter the systemic circulation via a right-to-left shunt and cause a paradoxical cerebral air embolism. Since the underlying cause is lethal and could lead to life-threatening complications, it is important to investigate the entry point whenever intracardiac microbubbles are observed. A giant esophageal hiatus hernia is a rare but possible cause. Surgical treatment of a giant esophageal hiatus hernia may prevent the development of microbubbles.

Additional Information

Disclosures

Human subjects: Consent was obtained or waived by all participants in this study. Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: Payment/services info: All authors have declared that no financial support was received from any organization for the submitted work. Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. Other relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Barak M, Katz Y: Microbubbles: pathophysiology and clinical implications. Chest. 2005, 128:2918-32. 10.1378/chest.128.4.2918
2. Patel H, Boateng S, Singh G, Feinstein S: Spontaneous right-sided microcavitations in a healthy adult. Echo Res Pract. 2015, 2:K53-6. 10.1530/ERP-15-0021
3. Graf A, Steffen C, Frick S: Spontaneous intracardiac microcavitations in a patient with a colonic carcinoma. BMJ Case Rep. 2019, 12:10.1136/bcr-2019-229932
4. Abboud B, El Hachem J, Yazbeck T, Doumit C: Hepatic portal venous gas: physiopathology, etiology, prognosis, and treatment. World J Gastroenterol. 2009, 15:3585-90. 10.3748/wjg.15.3585
5. Liebman PR, Patten MT, Manny I, Benfield JR, Hechtman HB: Hepatic--portal venous gas in adults: etiology, pathophysiologist and clinical significance. Ann Surg. 1978, 187:281-7. 10.1097/00000658-197805000-00012
6. Mittal K, Annandpara K, Dey AK, Kedar P, Hira P, Kale S: Left aberrant gastric vein causing isolated left hepatic portal venous gas secondary to an incarcerated diaphragmatic hernia. Pol J Radiol. 2015, 80:364-7. 10.12659/PJR.894103
7. Mallens WM, Scheppers-Bok R, Nicolai JJ, Jacobs FA, Heyerman HG: Portal and systemic venous gas in a patient with cystic fibrosis: CT findings. AJR Am J Roentgenol. 1995, 165:338-9. 10.2214/ajr.165.2.7618551
8. Kamikado C, Nagano S, Takumi K, et al.: Gas embolism caused by portal vein gas: case report and literature review. Case Rep Gastroenterol. 2008, 2:262-71. 10.1159/000146664
9. Kriegshauser JS, Reading CC, King BF, Welch TJ: Combined systemic and portal venous gas: sonographic and CT detection in two cases. AJR Am J Roentgenol. 1990, 154:1219-21. 10.2214/ajr.154.6.12170731
10. Palmon SC, Moore LE, Lundhberg J, Young T: Venous air embolism: a review. J Clin Anesth. 1997, 9:251-7. 10.1016/S0952-8180(97)00024-X
11. Gronert GA, Messick IM Jr, Cucchiara RF, Michenfelder JD: Paradoxical air embolism from a patent foramen ovale. Anesthesiology. 1979, 50:548-9. 10.1097/00000542-197906000-00018
12. Saver JL, Mattle HP, Thaler D: Patent foramen ovale closure versus medical therapy for cryptogenic ischemic stroke: a topical review. Stroke. 2018, 49:1541-8. 10.1161/STROKEAHA.117.018153