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Septic cerebral embolism from a nontraumatic atrioesophageal fistula: a case report

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Case Report

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Abstract

Background

Nontraumatic atrioesophageal fistula (AEF) is an exceedingly rare and usually fatal communication between the esophagus and the left atrium. Neurological symptoms may dominate its presentation, yet they are easily attributed to more common causes.

Case presentation:

A 48-year-old man with epilepsy, heavy alcohol use and six months of progressive dysphagia — evaluated once by a normal otolaryngological examination four weeks earlier but not further investigated — was brought to the emergency department after being found at home confused and disoriented, with septic shock and upper gastrointestinal bleeding; no seizure activity had been witnessed, although an unwitnessed epileptic seizure could not be excluded. Initial non-contrast head CT was unremarkable, and the presentation was attributed to a possible postictal state in the context of septic-metabolic encephalopathy. The following day, new gaze deviation, central facial palsy, severe hemiparesis and tactile neglect on the left prompted repeat imaging. CT showed a new left thalamic lesion, and CT angiography revealed a gas-containing embolus at the left carotid bifurcation. These findings led to reassessment of the echocardiographic and thoracic findings and to the diagnosis of a left-sided AEF. Despite emergency embolectomy and atrial wall repair, a further ischemic lesion appeared in the right parietal region one week after surgery, and the patient died on postoperative day 10; biopsy confirmed esophageal squamous cell carcinoma.

Conclusions

Septic embolism from an occult AEF can present as a stroke mimic or be masked by postictal and septic-metabolic explanations. Evolving focal deficits warrant early neurological consultation, vascular imaging and reassessment of earlier findings, even when initial imaging is negative.

Introduction

Septic emboli are an uncommon but clinically important cause of ischemic stroke and are associated with disproportionately high mortality [1]. One of their rarest sources is an atrioesophageal fistula (AEF), an abnormal communication between the esophagus and the left atrium. Most published cases are iatrogenic and follow catheter or surgical ablation for atrial fibrillation [2, 3]; nontraumatic fistulas, arising from esophageal ulceration or malignancy, are exceptional and have been described only in isolated reports and small series [4–7]. Malignant esophageal perforation into the left atrium can give rise to cerebral gas embolism and sepsis [8].

Although the lesion is thoracic, AEF is very often a neurological disease at presentation. Embolization of air, thrombus or esophageal contents produces embolic strokes, seizures, encephalopathy, coma or meningitis, and in reviews of ablation-related cases neurological symptoms accompany the clinical picture in most patients and predict a worse outcome [9–11].

The internal medicine, gastroenterological and cardiac surgical aspects of the present patient have been published previously [12]. In this report we describe the same case from the neurological perspective. The evolution of the neurological deficits, from a presentation initially explained by a possible seizure and septic encephalopathy to persistent focal signs incompatible with that explanation, prompted repeat neuroimaging and vascular imaging. The finding of a gas-containing septic embolus, in turn, led to reassessment of the echocardiographic, esophageal and thoracic findings and to the correct diagnosis. We believe this diagnostic pathway, which is not covered by the earlier report, is of practical value to neurologists and emergency physicians. The report was prepared in accordance with the CARE guidelines [13].

Case presentation

Patient history

A 48-year-old man with secondary (toxic-metabolic) epilepsy, arterial hypertension and heavy alcohol use presented with six months of progressive dysphagia accompanied by interscapular pain and an 8-kg weight loss over five weeks. An otolaryngological examination four weeks before admission was normal, and no further work-up of the dysphagia had been performed. The day before admission he vomited twice. On the morning of admission he was still communicating normally with his family; in the afternoon he was found at home sitting on the bed, without verbal contact and with no seizure activity witnessed. He was brought to the emergency department disoriented.

Neurological presentation and course

On admission, the clinical picture was dominated by neurological findings: altered consciousness with disorientation, and deviation of the head and gaze. The patient was hypotensive (blood pressure 60/40 mmHg) and tachycardic (heart rate 109/min), with a temperature of 37.3°C, requiring vasopressor support for septic shock; a nasogastric tube yielded hemorrhagic content, indicating upper gastrointestinal bleeding. Laboratory testing showed a marked systemic inflammatory response (CRP 260 mg/L, procalcitonin > 114 ng/mL, leukocytosis and neutrophilia) and a coagulopathy (INR 1.65); detailed results have been reported previously [12].

Initial non-contrast head CT showed no acute findings, and contrast-enhanced CT of the thorax was interpreted as acute phlegmonous esophagitis, with a possible diverticulum of the ventral esophageal wall extending toward the pulmonary veins. In a patient with known epilepsy and septic shock, in whom no seizure activity had been witnessed but an unwitnessed epileptic seizure could not be excluded, the

altered consciousness and gaze deviation were provisionally attributed to a postictal state in the context of septic-metabolic encephalopathy. During the morning ward round the following day, the deviation of the head and eyes to the right became irrepressible, and the patient acutely developed a left-sided central facial nerve palsy, severe left-sided hemiparesis and left-sided tactile neglect; he remained conscious, with adequate one-word answers. At this stage, the differential diagnosis was postictal (Todd's) paresis versus an evolving acute cerebrovascular event.

Because the focal deficits persisted, a repeat non-contrast head CT and CT angiography of the cervical and intracranial vessels were performed the same day. Non-contrast CT demonstrated a new hypodensity, up to 8 mm, in the left thalamus (Fig. 1). CT angiography showed, at the bifurcation of the left common carotid artery extending into the origins of the external and internal carotid arteries, a hypodensity containing foci of gas and causing hemodynamically significant stenosis to occlusion of the internal carotid artery — a finding highly suggestive of a septic, gas-containing embolus (Fig. 2). Intraluminal gas within a cerebral embolus is highly atypical of atherothrombotic or cardioembolic stroke and pointed to an embolic mechanism from a proximal, air-containing source rather than postictal paresis or a primary atherothrombotic event.

Diagnostic reassessment

The neurovascular findings prompted a targeted search for the embolic source. Echocardiography demonstrated a thrombus in the left atrium together with external compression of the atrial wall, suggesting an extracardiac process adjacent to the atrium. On reassessment (“second look”) of the admission chest CT, and on a repeat contrast-enhanced CT of the thorax, aorta and esophagus with oral contrast performed the same day, a ventral esophageal diverticulum/ulcer was seen extending into the region of the left atrium, raising suspicion of a developing covered communication or perforation between the esophagus and the left atrium, without CT evidence of active perforation into the mediastinum, pneumomediastinum or pneumoperitoneum; the thoracic and abdominal aorta showed no other thromboemboli (Fig. 3). These findings established the diagnosis of a left-sided AEF.

Treatment and outcome

The same day, within approximately 24 hours of admission, the patient was transferred emergently to a cardiac surgery center, where he underwent embolectomy of the left common, internal and external carotid arteries and reconstruction of the left atrial wall with a pericardial patch; the surgical and perioperative management has been described in detail previously [12]. He was transferred back to the intensive care unit of our institution on postoperative day 2. Septic shock nevertheless persisted and progressed to multiorgan failure. Postoperative gastroscopy, deferred earlier because of the risk of enlarging the perforation, revealed an ulcerated esophageal lesion, and biopsy confirmed high-grade squamous cell carcinoma. On postoperative day 7, follow-up non-contrast CT of the brain revealed a new ischemic lesion in the right high parafalcine (parietal) region, measuring 3 × 2.5 cm (Fig. 4), of uncertain

embolic versus hemodynamic (watershed) etiology in the perioperative and septic shock setting. Given the critical condition and the histological findings, care was transitioned to palliative management, and the patient died on postoperative day 10. The main events are summarized in Table 1.

Table 1
Timeline of key clinical, neurological and diagnostic events.

Time point	Event
6 months before admission	Progressive dysphagia with interscapular pain and an 8-kg weight loss over 5 weeks
~ 4 weeks before admission	Otolaryngological evaluation for dysphagia; normal findings, no further work-up performed
Day − 1	Vomiting (twice)
Day 0 – admission	Found at home confused and disoriented, without verbal contact; no seizure activity witnessed; septic shock requiring vasopressors; hemorrhagic nasogastric aspirate
Day 0	Non-contrast head CT without acute findings; chest CT interpreted as phlegmonous esophagitis. Altered consciousness and gaze deviation attributed to a possible postictal state (unwitnessed seizure could not be excluded) with septic-metabolic encephalopathy
Day 1	During morning ward round: irrepressible right head/gaze deviation, left central facial palsy, severe left hemiparesis, left tactile neglect
Day 1	Repeat non-contrast CT (new left thalamic hypodensity) and CT angiography (gas-containing embolus, left carotid bifurcation)
Day 1	Echocardiography: left atrial thrombus with external compression of the atrial wall. Reassessment of chest CT and repeat contrast-enhanced CT with oral contrast: esophageal ulcer/diverticulum with suspected perforation into the left atrium → diagnosis of left-sided AEF
Day 1	Emergency transfer for carotid embolectomy and pericardial patch reconstruction of the left atrial wall
Postoperative day 2	Transfer back to the intensive care unit of our institution
Postoperative period	Persistent septic shock and multiorgan failure; gastroscopy and biopsy: high-grade esophageal squamous cell carcinoma
Postoperative day 7	Follow-up CT: new ischemic lesion, right parafalcine (parietal) region (3 × 2.5 cm)
Postoperative day 10	Death after transition to palliative care

AEF, atrioesophageal fistula; CT, computed tomography

Discussion

This case shows how a rare thoracic-cardiac communication can present primarily as a neurological syndrome and how neurological reasoning can open the path to the diagnosis. The initial presentation — altered consciousness and gaze deviation in a patient with known epilepsy and septic shock — was plausibly explained by a possible seizure and septic-metabolic encephalopathy, and the normal admission CT did not challenge that explanation. Such an explanation carries a risk of premature closure. Early ischemic changes are frequently invisible on non-contrast CT, and a history of epilepsy readily invites the interpretation of new focal deficits as postictal (Todd's) paresis. In our patient it was the persistence and evolution of the focal signs, rather than any single test, that made the working diagnosis untenable and triggered repeat and vascular imaging.

Several features, viewed together, point to septic embolism rather than a primary neurological event. First, intraluminal gas within a carotid embolus is highly unusual in atherothrombotic or cardioembolic stroke and should prompt a search for a communication between the air-containing gastrointestinal tract and the systemic circulation. Second, a further ischemic lesion appeared in a second arterial territory (the right parafalcine/parietal region) in the postoperative period, consistent with an ongoing or recurrent embolic process before and around the time of definitive surgical repair, although a contribution from perioperative hemodynamic compromise (a watershed mechanism) cannot be excluded. Third, the combination of chronic dysphagia, hematemesis and acute neurological signs has been described as the characteristic triad of nontraumatic esophago-atrial fistula [5]; all three were present in our patient, although the dysphagia had already prompted an otolaryngological evaluation four weeks before admission that did not identify its cause. In ablation-related AEF, neurological manifestations, including embolic stroke, seizures, coma and cerebral air embolism, are present in the majority of patients and are associated with higher mortality [2, 9–11].

The neurological findings also had direct consequences for further management. They prompted reassessment and reinterpretation of the chest CT, previously read as phlegmonous esophagitis, as a perforation into the left atrium. Contrast-enhanced chest CT is the preferred diagnostic test for AEF, whereas endoscopy, transesophageal echocardiography and other esophageal instrumentation may enlarge the fistula and precipitate air or food embolism [9, 10]. Nontraumatic AEF secondary to esophageal malignancy is exceedingly rare, and its prognosis is poor even with surgical treatment [4, 6, 8]. Nevertheless, surgery is the only therapy that offers a chance of survival [9, 11], and time to diagnosis matters. Early neurological involvement, recognition of the stroke-mimic pattern and early vascular imaging may shorten the diagnostic interval.

This report has limitations. It describes a single patient, the neurological examination was limited by the critical condition, it remains uncertain whether the patient experienced an unwitnessed epileptic seizure at presentation, and the diagnostic sequence was reconstructed retrospectively from the medical records.

Conclusions

Neurological symptoms, including seizures, gaze deviation, hemiparesis and other stroke-like presentations, may be the presenting feature of septic embolism from an occult atrioesophageal fistula rather than a primary neurological event. In a patient with sepsis, upper gastrointestinal bleeding or a history of dysphagia, evolving focal deficits should raise a high level of vigilance. Such patients warrant early neurological consultation, CT angiography and contrast-enhanced chest CT, and repeated reassessment of earlier findings, even when initial neuroimaging and other investigations are reported as negative. Attributing new deficits solely to postictal or septic-metabolic causes risks delaying a diagnosis whose only effective treatment is urgent surgery.

Declarations

Ethics approval and consent to participate

The research was conducted in accordance with the Declaration of Helsinki and approved by the institutional ethics committee of Jessenius Faculty of Medicine in Martin (Approval No. EK 32/2023). Written informed consent to participate was obtained from the patient's next of kin.

Consent for publication

Written informed consent for publication of this case report and the accompanying images was obtained from the patient's next of kin.

Availability of data and materials

All data relevant to this case are included in this article. Further details are available from the corresponding author on reasonable request, subject to patient confidentiality.

Competing interests

PS is an Editorial Board Member of BMC Neurology. He was not involved in the editorial handling or peer review of this manuscript. The remaining authors declare that they have no competing interests.

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Authors' contributions

PS and ML conceived the report, collected the neurological data and drafted the manuscript. PS and RM were involved in the neurological management of the patient and revised the manuscript. MM and RM provided the internal medicine data and revised the manuscript. EK and VN supervised the work and critically revised the manuscript. All authors read and approved the final manuscript.

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Declarative Use of AI

Claude (Anthropic) was used to assist with structuring the manuscript and correcting grammar and language. The authors reviewed and edited all content and take full responsibility for the content of this publication.

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Figures

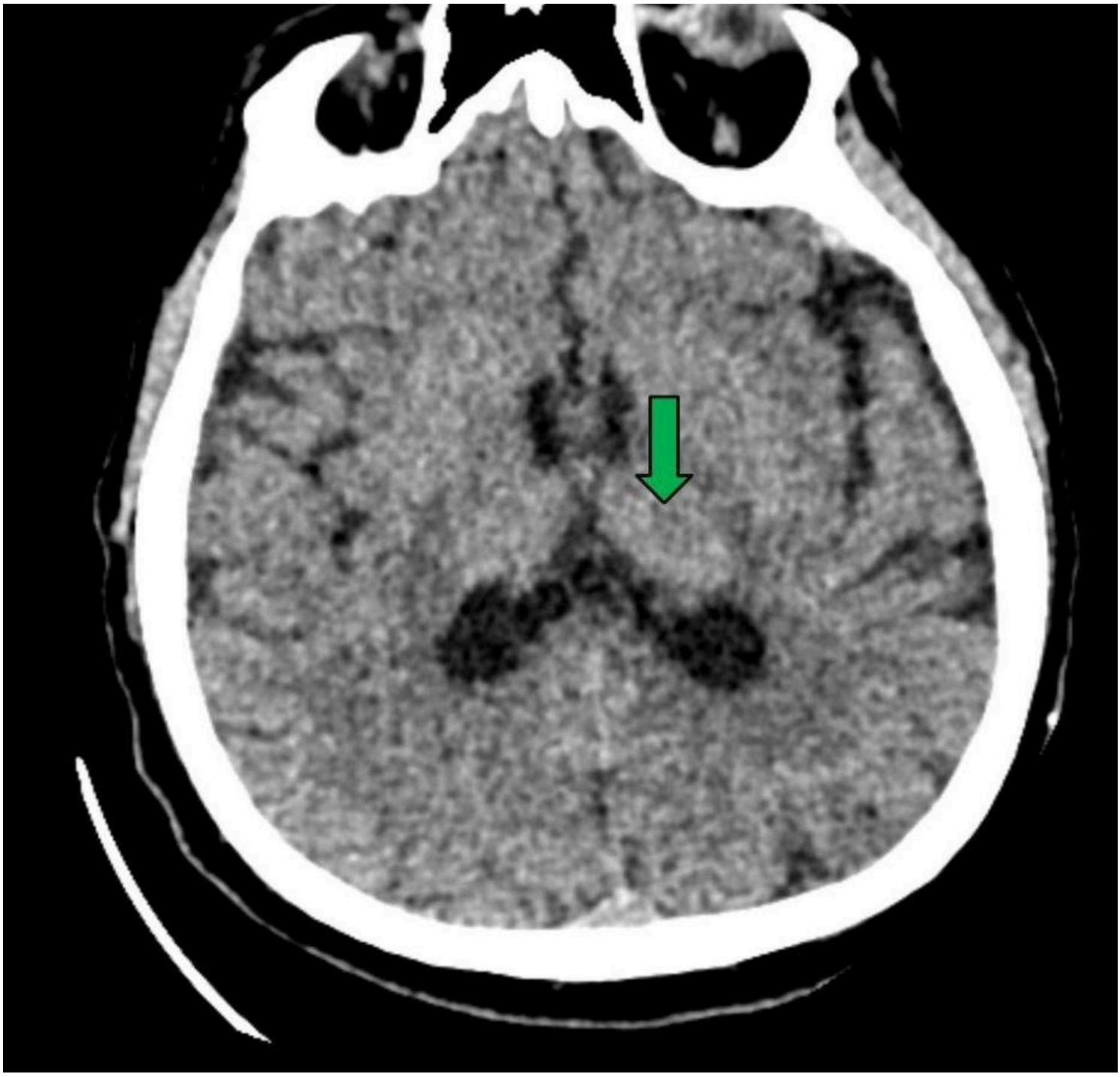


Figure 1

Non-contrast CT of the brain, Day 1, showing a new hypodensity up to 8 mm in the left thalamus (arrow).

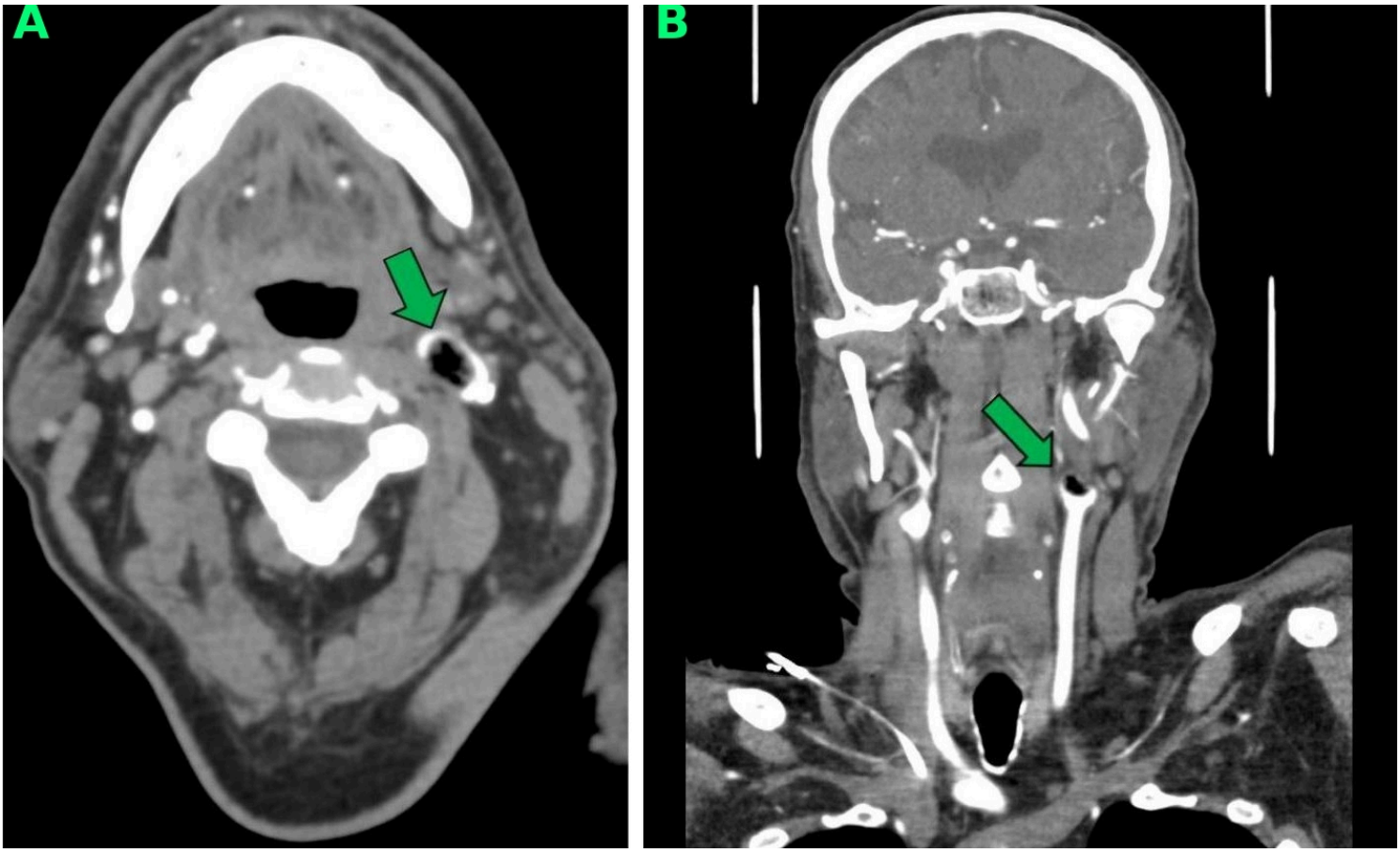


Figure 2

CT angiography of the neck vessels, axial (A) and coronal (B) reconstructions, performed on Day 1. At the bifurcation of the left common carotid artery, extending into the origins of the external and internal carotid arteries, a hypodensity containing foci of gas is seen (arrows), causing hemodynamically significant stenosis to occlusion of the internal carotid artery — consistent with a septic gas-containing embolus.



Figure 3

Contrast-enhanced CT of the thorax. (A) Sagittal reconstruction from reassessment of the admission (Day 0) study. (B) Axial image from the repeat CT with oral contrast performed on Day 1. Both show a ventral esophageal diverticulum/ulcer extending toward the left atrium (arrows), with findings suspicious for a developing communication/perforation between the esophagus and the left atrium.

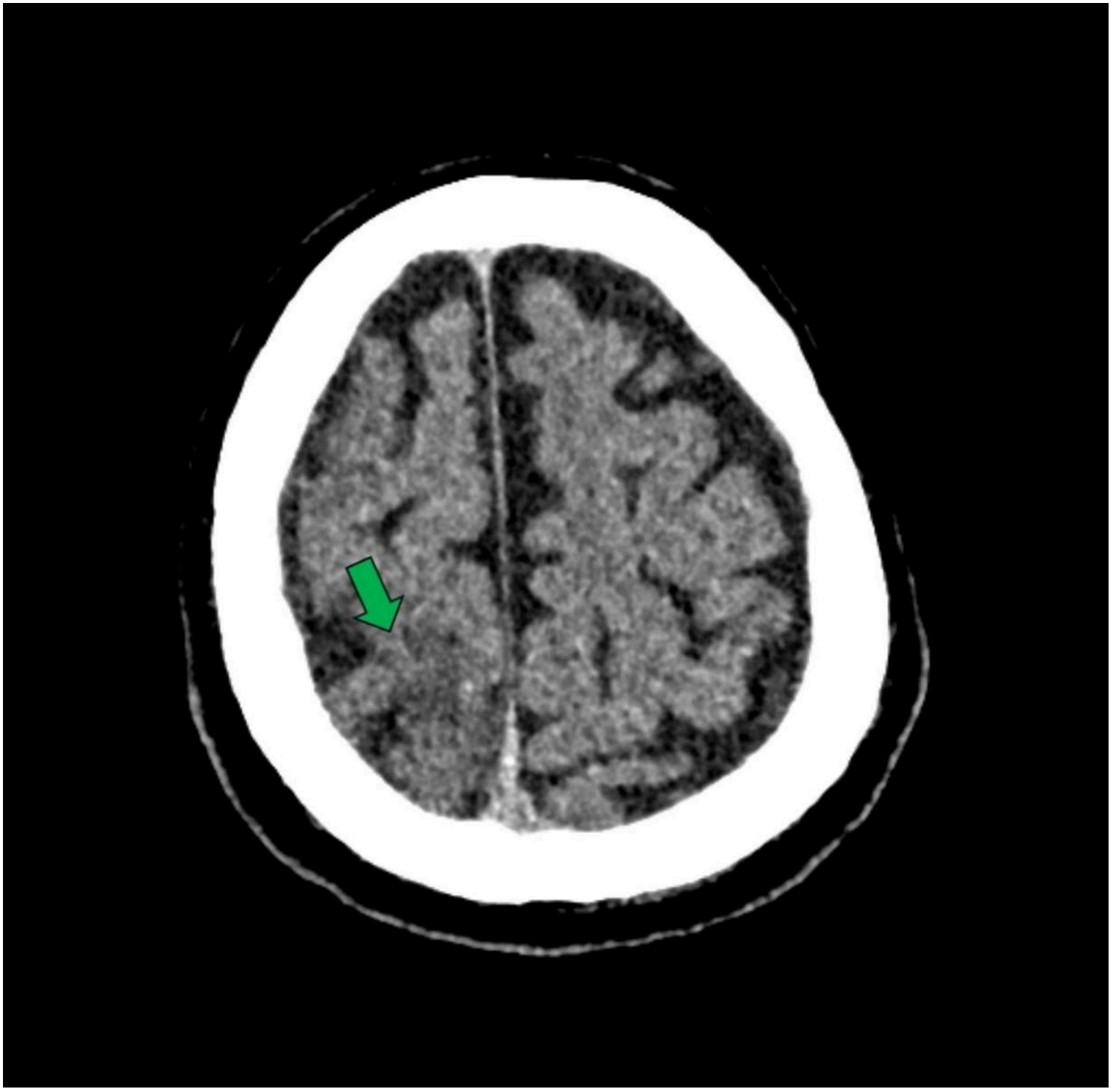


Figure 4

Non-contrast CT of the brain, postoperative day 7, showing a new ischemic lesion in the right high parafalcine (parietal) region, measuring 3 × 2.5 cm (arrow).

Supplementary Files

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- CAREchecklist.pdf