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Slit-Like Ventricles in Diffuse Cerebral Edema After Cardiac Arrest: A Case Report

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Abstract

Slit-like ventricles can accompany diffuse cerebral edema after cardiac arrest, and their significance depends on the associated parenchymal findings and clinical course. We describe a previously independent 65-year-old man who experienced recurrent cardiac arrest with nonshockable rhythms after a week of worsening dyspnea and peripheral edema. Following prolonged resuscitation, he remained comatose. Cranial computed tomography on hospital day 2 was reported to show diffuse cerebral edema, ventricular compression described as slit-like, and crowding at the foramen magnum. The intensive care assessment also recorded loss of cortical gray-white matter differentiation. Three available axial images illustrate effacement of visible ventricular and other cerebrospinal fluid spaces. Following multidisciplinary review and discussions with the family, life-sustaining treatment was withdrawn in the setting of reported severe brain injury, refractory shock, and multiorgan dysfunction. The patient died early on hospital day 3.

This case illustrates severe ventricular compression reported as slit-like, with complete effacement at the sites represented on the selected images. It cannot establish neurological prognosis or validate treatment-withdrawal decisions.

Categories: Neurology, Radiology, Emergency Medicine
Keywords: anoxic brain injury, cardiac arrest, computed tomography, diffuse cerebral edema, global cerebral edema, hypoxic-ischemic brain injury, neuroimaging, post-cardiac arrest syndrome, slit-like ventricles, ventricular compression

Introduction

Global hypoxic-ischemic brain injury is a major complication of cardiac arrest. Cranial computed tomography (CT) can identify an intracranial cause of collapse and demonstrate evolving brain injury after return of spontaneous circulation (ROSC) [1,2]. Established CT findings include diffuse swelling, loss of gray-white matter differentiation, and effacement of the cortical sulci, basal cisterns, and ventricular cerebrospinal fluid (CSF) spaces. Severe injury may also produce hypoattenuation of the basal ganglia and thalami and the pseudo-subarachnoid hemorrhage sign, in which cisternal and sulcal structures appear relatively hyperattenuating against diffusely hypoattenuating brain parenchyma [3]. The reversal sign denotes inversion of the normal attenuation relationship, with cortical gray matter appearing less attenuating than adjacent white matter [4].

Early CT may appear normal despite significant hypoxic-ischemic injury. Magnetic resonance imaging (MRI) with diffusion-weighted imaging (DWI) is more sensitive for detecting early cytotoxic injury, although diffusion abnormalities also evolve with time [5]. The gray-white matter attenuation ratio provides a quantitative measure of the relative CT attenuation of gray and white matter; a reduction can reflect cerebral edema [6]. Imaging findings require interpretation alongside the clinical course and appropriately timed multimodal neuroprognostication [1,2].

This report presents a case of diffuse cerebral edema after recurrent cardiac arrest in which the radiology account described slit-like ventricles. The purpose of this report is to place the reported ventricular morphology within the established imaging spectrum of global hypoxic-ischemic brain injury and to distinguish slit-like ventricular compression from complete ventricular effacement as illustrated in the selected CT images.

Case Presentation

A 65-year-old man who had previously been independent in activities of daily living developed progressive dyspnea and peripheral edema over one week. He had limited prior contact with healthcare services, no established medical diagnoses, and took no regular medications. He was a former smoker and had a history of substantial alcohol consumption. No history of hydrocephalus or CSF shunting was documented.

Emergency services were called because of worsening dyspnea. The patient was initially able to communicate with responders but subsequently developed cardiac arrest, and resuscitation reportedly

began immediately. The available narrative recorded approximately 20 minutes of basic life support followed by 25 minutes of advanced life support, with asystole followed by pulseless electrical activity. After ROSC, he was intubated and transported to the hospital. Two further episodes of pulseless electrical activity required approximately six and eight minutes of resuscitation, respectively. These durations could not be reconciled against precise source timestamps. A verified no-flow interval, bystander-cardiopulmonary resuscitation (CPR) details, shock count, and total epinephrine dose were unavailable; the recorded basic-life-support duration should not be interpreted as a delay before CPR.

Following ROSC, a Glasgow Coma Scale score of 3 was documented before the initiation of continuous sedation, along with dilated, poorly reactive pupils. This observation does not establish an examination free from residual resuscitation drugs or other confounders. Anterior ST-segment elevation was described in the clinical narrative and prompted coronary angiography. According to the catheterization report summarized in that account, the circulation was right-dominant, with minor wall irregularities and no significant obstructive coronary artery disease.

Bedside transthoracic echocardiography was reported to show right ventricular dilation, with limited assessment of regional left ventricular wall motion. Pulmonary CT angiography was performed to investigate pulmonary embolism. A possible partial right-sided embolus was mentioned in the clinical narrative, but the formal pulmonary angiography report was unavailable, and the selected screenshots did not establish a definite embolus. Pulmonary embolism therefore remains unconfirmed in this report. The cause of arrest remained uncertain. The available laboratory results are summarized in Table *1*.

Parameter	Recorded result	Unit	Published adult comparator
Arterial pH	7.29	Unitless	7.38-7.44
Arterial $PaCO_2$	55	mmHg	38-42
Arterial PaO_2	131	mmHg	Not applied on supplemental oxygen
Bicarbonate	26.4	mmol/L	23-26
Arterial oxygen saturation	99	%	Not applied on supplemental oxygen
Sodium	138	mmol/L	136-145
Potassium	4.9	mmol/L	3.5-5.0
Lactate	17	mmol/L	<1.3 for arterial lactate
Glucose	Unavailable	mmol/L	Not applied
Chloride	Unavailable	mmol/L	Not applied
Anion gap	Not calculable	mmol/L	Not applied
Base excess	Unavailable	mmol/L	Not applied

TABLE 1: Laboratory results recorded during emergency evaluation

The documented blood gas was obtained after resuscitation during mechanical ventilation with a fraction of inspired oxygen (FiO_2) of 0.60 and positive end-expiratory pressure of 5 cmH_2O. The relation of the recorded lactate value to the gas-sample time could not be verified. An earlier, separate blood gas sample recorded potassium of 6.5 mmol/L; its timing and treatment context were unavailable.

Comparators are source-specific published values from the American Board of Internal Medicine [7], not verified local laboratory intervals. The gas intervals reproduce the specific published source and should not be interpreted as universal limits. Sodium and potassium use serum comparators. Room-air oxygen ranges are omitted because they are not applicable to the documented oxygen conditions. Chloride and contemporaneous source measurements were unavailable, so no anion gap was calculated.

$PaCO_2$, arterial partial pressure of carbon dioxide; PaO_2, arterial partial pressure of oxygen

Cardiac and pulmonary investigations during emergency evaluation are summarized in Table *2*.

Investigation	Findings and source limitation
Electrocardiography	Anterior ST-segment elevation after ROSC was described in the clinical narrative; the original tracing was not available for reassessment.
Bedside transthoracic echocardiography	Reported right ventricular dilation; regional left ventricular wall motion was difficult to assess.
Pulmonary CT angiography	Performed to investigate pulmonary embolism. A definitive angiographic result cannot be provided because the formal report and complete examination were unavailable.
Coronary angiography	The account of the catheterization report described right dominance, minor wall irregularities without significant obstructive disease, and a discrete intramyocardial course of the mid left anterior descending artery.

TABLE 2: Cardiac and pulmonary investigations during emergency evaluation

Findings are attributed to the reports and clinical narrative summarized in the available case account. The full original investigations were unavailable for independent reassessment. A pulmonary embolus is not treated as an established angiographic finding.

CT, computed tomography; ROSC, return of spontaneous circulation

Representative coronary angiographic images show the right coronary artery (Figure *1A*) and left coronary system (Figure *1B*).

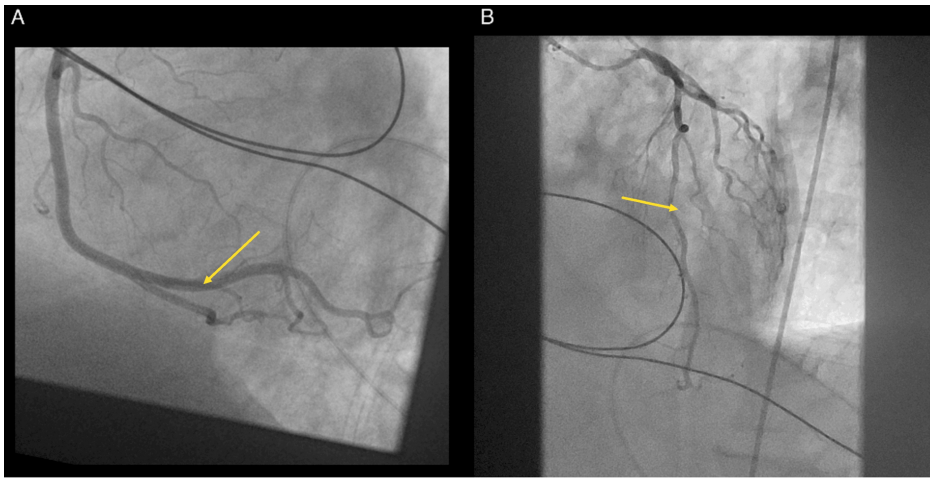


FIGURE 1: Representative coronary angiographic images after cardiac arrest

(A) Right coronary artery. (B) Left coronary system. Yellow arrows identify the contrast-opacified coronary vessels. The complete catheterization report described minor wall irregularities without significant obstructive coronary artery disease. These selected still frames illustrate the investigation; that conclusion is based on the complete clinical study and its report.

Two selected axial screenshots document the pulmonary vascular investigation performed on hospital day 1 (Figures *2A*-*2B*). The suspected right-sided embolus reported in the clinical notes could not be independently established from these limited images.

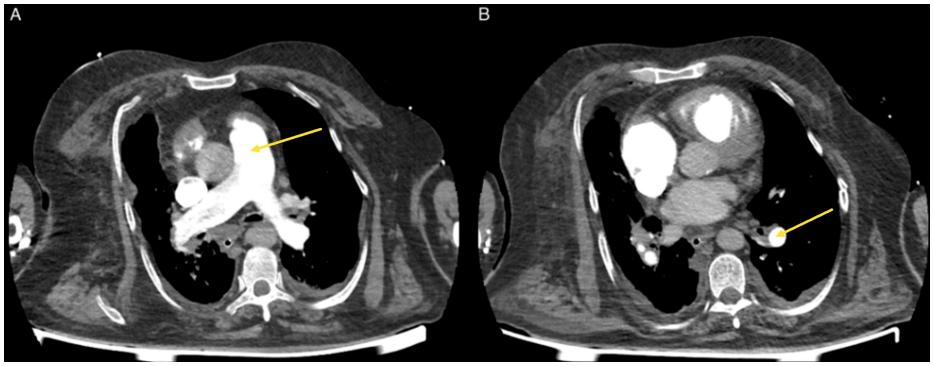


FIGURE 2: Selected axial pulmonary CT angiography images during emergency evaluation

(A) Image at the level of the central pulmonary arteries. (B) More caudal image through the heart and lower pulmonary arterial branches. Yellow arrows identify a central pulmonary arterial structure in A and a lower pulmonary arterial branch in B; they do not indicate a verified embolus. These screenshots document the investigation and do not independently confirm or exclude pulmonary embolism. The suspected embolus could not be localized from the available material.

CT, computed tomography

Cranial CT on hospital day 2 was reported to show diffuse cerebral edema, "slit-like ventricles," and crowding at the foramen magnum, without a skull fracture. The intensive care assessment additionally recorded loss of cortical gray-white matter differentiation and extensive ventricular effacement, interpreted as hypoxic-ischemic brain injury. The figure material available for this report consists of three axial screenshots from that examination (Figure *3*). They illustrate complete effacement of the ventricular CSF spaces represented on the selected supratentorial images, together with sulcal and basal cisternal effacement. Hospital day 2 does not establish an interval of at least 48 hours after the last ROSC; the exact interval was unavailable.

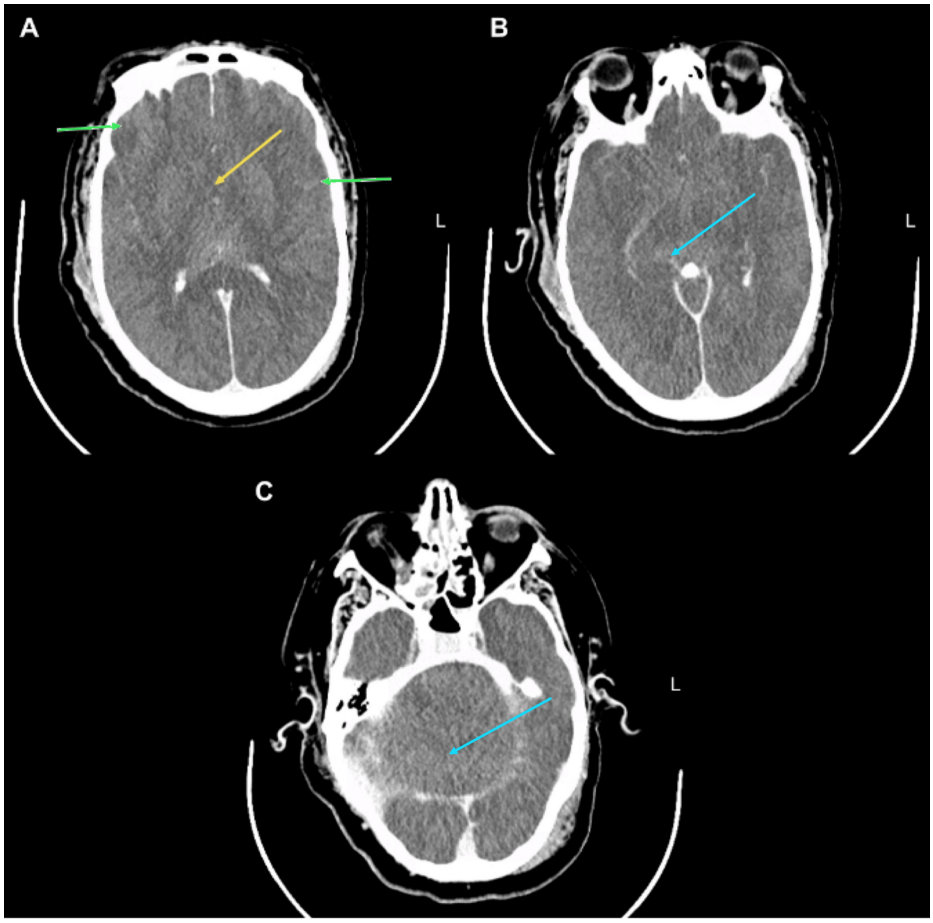


FIGURE 3: Selected axial CT images showing severe ventricular compression and CSF space effacement

Images were obtained on hospital day 2. Panel A shows the supratentorial ventricular region with no discernible CSF lumen at the yellow arrow and cortical sulcal effacement at the green arrows. Panel B shows the basal cisternal region with effacement of the visible CSF spaces at the cyan arrow. Panel C shows the posterior fossa; the cyan arrow identifies this anatomical region and does not demonstrate foramen-magnum crowding. Original image content and annotations are retained. The description is restricted to the selected images; reported foramen-magnum crowding is not directly demonstrated. L indicates the patient's left.

CT, computed tomography; CSF, cerebrospinal fluid

Following multidisciplinary review and discussions with the family, the treating team documented a decision to withdraw life-sustaining treatment because of reported severe brain injury in the setting of refractory shock and multiorgan dysfunction. Vasopressor support and supplemental oxygen were subsequently withdrawn, and the patient died early on hospital day 3. The exact ROSC-to-withdrawal interval and the relation to normothermia or sedative clearance were unavailable.

Beyond the initial examination, details of sedative or neuromuscular-blocking drugs, temperature management, a later examination after drug clearance, electroencephalography (EEG), somatosensory evoked potentials, neurological biomarkers, and MRI were unavailable. No prior brain imaging was supplied. Glucose and osmolality data, details of anticoagulation or thrombolysis decisions, and autopsy status were also unavailable. Lack of this information does not establish that an investigation or treatment was not performed.

Discussion

The central feature of this case is severe ventricular compression described as "slit-like ventricles" in the radiology account. The selected images illustrate complete effacement at the depicted ventricular sites, together with other CSF-space effacement in diffuse cerebral swelling after prolonged, recurrent cardiac arrest. The clinical course and reported loss of cortical gray-white matter differentiation are consistent with hypoxic-ischemic brain injury. This is an established imaging pattern, and the teaching point is to assess ventricular visibility alongside the cortical sulci, basal cisterns, and parenchymal attenuation. These descriptive observations do not provide a quantitative measure of the severity of injury.

The terms slit-like ventricles and complete ventricular effacement should not be used interchangeably. Slit-like morphology implies a thin residual lumen, whereas complete effacement denotes loss of a discernible CSF lumen on the relevant image. We retain "slit-like ventricles" in the title as the descriptor recorded in the radiology account, while explicitly describing complete effacement at the ventricular sites depicted in the selected screenshots. The title does not imply that a residual lumen is demonstrated on those images. Shunt-associated slit ventricle syndrome is a separate clinical entity [8]; with no documented CSF shunt, it is not a plausible competing diagnosis in this case.

The precipitating cause of arrest remains unresolved. The earlier potassium value of 6.5 mmol/L raises possible hyperkalemia as a contributor, but its relation to arrest and any treatment could not be established. Dyspnea, right ventricular dilation, and the narrative mention of possible pulmonary embolism warrant consideration but do not confirm pulmonary embolism without a definitive pulmonary angiographic result. Reported anterior ST elevation prompted appropriate coronary investigation, which was described as showing no significant obstructive disease. The available data cannot assign a single cause of arrest or determine whether these abnormalities preceded or followed resuscitation. Anticoagulation, thrombolysis, and autopsy details were unavailable.

The blood gas values also require cautious interpretation. A pH of 7.29 with $PaCO_2$ of 55 mmHg and bicarbonate of 26.4 mmol/L is compatible with a respiratory acidemic component. Lactate of 17 mmol/L denotes marked hyperlactatemia, but its relationship to the bicarbonate cannot be resolved without verified sampling times and treatment details. Bicarbonate administration, a coexisting metabolic process, or noncontemporaneous samples are possible explanations, not documented findings. Hypercapnia can increase cerebral blood flow and blood volume [1], but its contribution to swelling in this patient cannot be established from a single recorded value.

Prolonged CPR, nonshockable rhythms, and rearrest are relevant clinical context. In the cohort reported by Esdaille et al., early severe cerebral edema was also associated with younger age and noncardiac arrest etiology [9]. Applicability to this 65-year-old patient with an uncertain cause is therefore partial. These population-level associations cannot establish the mechanism or neurological outcome in this individual. Similarly, Lee et al. studied serial CSF-volume proportions in patients surviving to a delayed CT at 72-96 hours; the association of change with outcome was attenuated after adjustment for the early baseline measurement [10]. That study does not validate an inference from this single examination without baseline imaging.

Quantitative gray-white ratio studies rely on defined CT acquisition and measurement methods [6]; no Hounsfield-unit measurements, gray-white ratio, or calibrated ventricular or cisternal measurements were available for this report, and these could not be derived reliably from the screenshots. The 2023 neuroprognostication guideline considers diffuse loss of gray-white differentiation with sulcal effacement on CT at least 48 hours after ROSC as one component of multimodal assessment [2]. The 2025 AHA guideline recommends consolidating multimodal prognostic interpretation at least 72 hours after normothermia and discontinuation of sedatives, with attention to confounders [11].

This single case is limited by incomplete retrospective clinical information. Findings concerning the full CT examination, cortical gray-white differentiation, and the cardiac and pulmonary investigations are attributed to the radiology or clinical accounts summarized above, rather than to a new review of the complete original datasets. Baseline imaging, original digital CT data, calibrated quantitative measurements, acquisition settings, and sagittal reconstructions were unavailable. The reported foramen-magnum crowding could not be demonstrated. These limitations restrict the report to a descriptive imaging example and preclude causal or prognostic validation.

Conclusions

Slit-like ventricular compression can form part of the CT appearance of diffuse cerebral edema after recurrent cardiac arrest. In this case, ventricular narrowing, reported loss of gray-white matter differentiation, and sulcal effacement supported the interpretation of severe hypoxic-ischemic brain injury. The practical value of this appearance is to prompt a systematic review of the surrounding brain parenchyma and CSF spaces in the clinical context. It should be distinguished from shunt-associated slit ventricle syndrome. Early CT findings and ventricular caliber alone cannot establish brain death or a definitive long-term neurological prognosis; appropriately timed multimodal assessment remains essential.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Mykhailo Iashchuk

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Drafting of the manuscript: Mykhailo Iashchuk

Critical review of the manuscript for important intellectual content: Mykhailo Iashchuk, Sofia Pires, Nelson Barros

Supervision: Mykhailo Iashchuk

Disclosures

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