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# Sealed Post-infarction Left Ventricular Free-Wall Rupture in a Patient Taking Dabigatran: A Case Report

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## Abstract

Left ventricular free-wall rupture is a rare but life-threatening mechanical complication of acute myocardial infarction. In its subacute form, thrombus and pericardium temporarily contain the bleeding, opening a narrow window for diagnosis and surgery. We report a 76-year-old man with paroxysmal atrial fibrillation treated with dabigatran and with impaired renal function, who presented with syncope and cardiogenic shock after five days of untreated chest pain. The electrocardiogram (ECG) showed ST-segment elevation with QS complexes in leads I and aVL and reciprocal depression in the inferior leads, a pattern typical of first diagonal branch occlusion. Bedside echocardiography revealed a large, organized hemopericardium, and CT excluded aortic dissection. Coronary angiography confirmed occlusion of the first diagonal branch. Antithrombotic loading was withheld. At emergency surgery, a 1 × 1 cm tear of the mid-lateral free wall, matching the culprit territory, was found already sealed by thrombus; it was repaired with a sutureless patch technique combined with off-pump internal mammary artery grafting. After surgical re-exploration for recurrent postoperative bleeding, the patient recovered and was alive at 18 months. This case highlights the value of electrocardiographic localization, the central role of bedside echocardiography, and the practical issues raised by a direct oral anticoagulant at the time of rupture.

**Categories:** Cardiology, Emergency Medicine, Cardiac/Thoracic/Vascular Surgery
**Keywords:** cardiac surgery, cardiac tamponade, dabigatran, echocardiography, first diagonal artery, hemopericardium, idarucizumab, left ventricular free-wall rupture, myocardial infarction, sutureless repair

## Introduction

Left ventricular free-wall rupture (LVFWR) is a rare but life-threatening mechanical complication of acute myocardial infarction (AMI). Its incidence has declined from up to 6% of patients with ST-elevation AMI before the widespread use of primary percutaneous coronary intervention [1] to between 0.01% and 0.5% in contemporary series [2,3]. Outcomes, however, remain poor: mortality approaches 90% with medical treatment alone, and in-hospital mortality after surgical repair ranges from 17% to 36%; in a recent nationwide registry of 148 surgically treated patients, it was 31.1% [4]. Advanced age, late presentation, and absent reperfusion are classic risk factors, and current guidelines recommend urgent heart team evaluation and surgery once the diagnosis is suspected [5,6]. Rupture usually occurs within the first week after a transmural infarction, when the necrotic myocardium has been softened by inflammatory infiltration and enzymatic lysis but has not yet been reinforced by collagen deposition [5,7]. It may cause sudden electromechanical dissociation or, in its subacute form, be temporarily contained by thrombus and the pericardium, which seal the tear and allow time for diagnosis and surgical repair [7,8]. A sealed rupture should be distinguished from a ventricular pseudoaneurysm, in which the contained rupture becomes organized into a sac whose wall consists of pericardium and thrombus without myocardium and which communicates with the ventricular cavity through a neck [5].

We report a 76-year-old man taking dabigatran, with impaired renal function, who presented with syncope and cardiogenic shock five days after the onset of chest pain. A spontaneously sealed LVFWR caused by occlusion of the first diagonal branch was suspected at the bedside, confirmed at surgery, and repaired with a sutureless technique combined with coronary artery bypass grafting.

The principal contribution of this case report is the management of a contained rupture in a patient taking dabigatran with impaired renal function, a scenario that is likely to become more frequent. Direct oral anticoagulants (DOACs) are increasingly prescribed to elderly patients with atrial fibrillation, the same population at highest risk of rupture, and dabigatran is eliminated predominantly by the kidneys, with approximately 80% renal clearance, so its effect may persist when renal function is impaired [9,10]. We do not suggest that dabigatran caused the rupture, which is a complication of the infarct itself; rather, residual anticoagulant activity may increase pericardial bleeding and complicate surgical hemostasis. DOAC-associated hemorrhagic tamponade has been described mainly in the absence of structural cardiac injury: a

systematic review of 26 cases, nine of them involving dabigatran, found elevated serum creatinine in 15 patients and included no cases of ventricular rupture [11]. Data on post-infarction LVFWR in patients taking a DOAC, and on the timing of reversal in this setting, are therefore scarce. A secondary contribution is the diagnostic sequence, in which electrocardiographic localization and bedside echocardiography identified the culprit territory and the rupture in a high-lateral infarction, a presentation that is easily overlooked.

## Case Presentation

### Clinical presentation

A 76-year-old man was brought to the emergency department after a syncopal episode at home. His medical history included paroxysmal atrial fibrillation, arterial hypertension, diabetes mellitus, hypercholesterolemia, and hyperuricemia; his home therapy included dabigatran 150 mg twice daily, olmesartan 20 mg once daily, allopurinol 150 mg once daily, ezetimibe/atorvastatin, metformin, insulin degludec, and insulin lispro. He reported oppressive chest pain that had begun approximately five days earlier, for which he had not sought medical attention. On the morning of admission, he lost consciousness at home and was found unconscious. On arrival at the emergency department, he was in cardiogenic shock: at triage, blood pressure was 70/50 mmHg, heart rate 87 beats per minute, and oxygen saturation 98%, and blood pressure subsequently became unrecordable. He was awake but poorly cooperative. The timeline of clinical events is summarized in Table *1*.

| Time | Event |
|---|---|
| About five days before admission | Onset of oppressive chest pain; no medical contact |
| Day 0, morning | Syncope at home; found unconscious |
| Day 0, 11:39 | Arrival at the emergency department by ambulance; triage blood pressure 70/50 mmHg, heart rate 87 beats per minute, oxygen saturation 98% |
| 11:58 | Medical and cardiology evaluation; ECG with high-lateral ST-segment elevation |
| 12:20 | Repeat ECG: persistent ST-segment elevation in I, aVL, V5, and V6 |
| 13:13 | Troponin I 20,559 ng/L; creatine kinase-MB 24.9 ng/mL |
| Before 13:28 | Bedside TTE: large organized hemopericardium; LVEF 45%-50%; norepinephrine started; pericardiocentesis avoided |
| 13:28 | Thoracic CT angiography without and with contrast: extensive hemopericardium; no aortic dissection |
| 14:09 | Emergency transfer to the referral center with cardiac surgery |
| 15:16 | Coronary angiography: occluded first diagonal branch, 70% mid-LAD stenosis; ventriculography: anterolateral akinesia, no extravasation |
| Day 0, afternoon | Sternotomy: about 500 mL of blood and clots evacuated; sealed 1 × 1 cm mid-lateral tear; sutureless repair; off-pump left internal mammary artery to LAD grafting |
| By postoperative day 5 | Recurrent hemorrhagic tamponade on echocardiography; surgical re-exploration |
| About two weeks after surgery | Transfer to the ward |
| About three weeks after surgery | Discharge on aspirin, clopidogrel, and apixaban |
| Six months | Antiplatelet therapy discontinued; apixaban alone |
| 18 months | Follow-up: alive; LVEF 50%; no pericardial effusion or pseudoaneurysm |

**TABLE 1: Timeline of clinical events.**

ECG, electrocardiogram; TTE, transthoracic echocardiography; LVEF, left ventricular ejection fraction; CT, computed tomography; LAD, left anterior descending artery

### Diagnostic evaluation

The 12-lead electrocardiogram (ECG) showed sinus rhythm with ST-segment elevation and QS complexes in leads I and aVL and reciprocal ST-segment depression in leads III and aVF; a subsequent tracing showed persistent ST-segment elevation in leads I, aVL, V5, and V6 (Figure *1*). Laboratory tests showed cardiac troponin I of 20,559 ng/L, creatine kinase of 500 U/L, creatine kinase-MB of 24.9 ng/mL, C-reactive protein of 9.18 mg/dL, white blood cell count of 17.62 × $10^9$/L, hemoglobin of 15.6 g/dL, platelet count of 408 × $10^9$/L, and blood glucose of 232 mg/dL. Serum creatinine was 1.84 mg/dL, corresponding to an estimated glomerular filtration rate of approximately 38 mL/min/1.73 $m^2$ (Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) 2021 equation) and a creatinine clearance of approximately 34 mL/min (Cockcroft-Gault formula, body weight 70 kg). Serum creatinine before admission was 1.6 mg/dL (creatinine clearance approximately 39 mL/min), indicating pre-existing chronic kidney disease. Coagulation tests could not be obtained because the sample was insufficient, and no specific dabigatran assay was performed. The time of the last dabigatran intake could not be established.

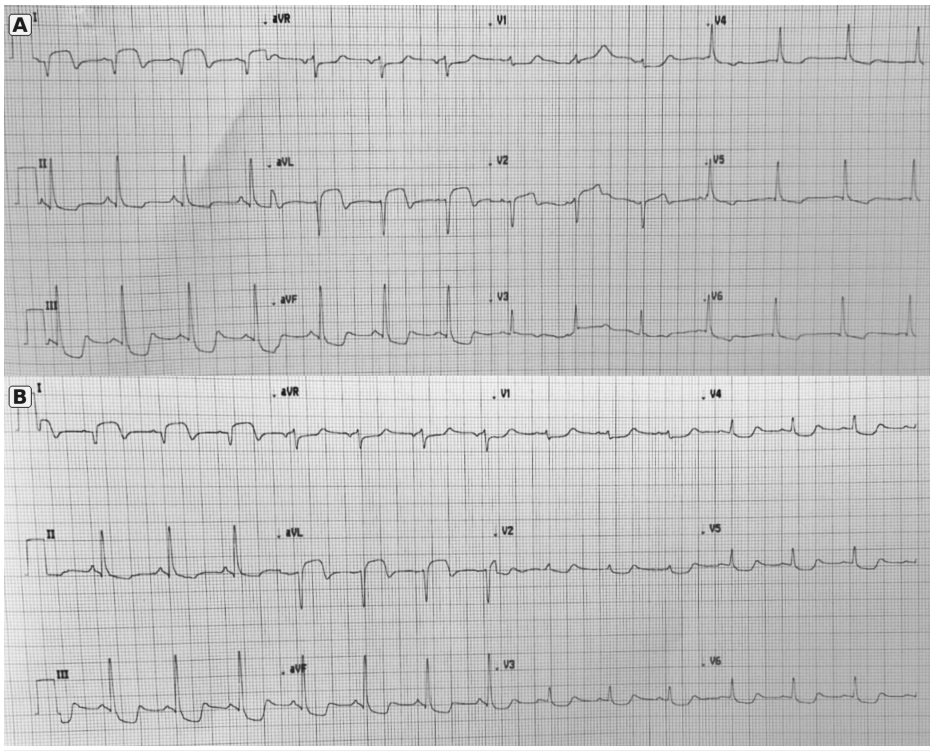



**FIGURE 1: Twelve-lead electrocardiograms at presentation.**

(A) ST-segment elevation with QS complexes in leads I and aVL and reciprocal ST-segment depression in leads III and aVF, a high-lateral pattern consistent with first diagonal branch occlusion. (B) Subsequent tracing showing persistent ST-segment elevation in leads I, aVL, V5, and V6.

Bedside transthoracic echocardiography (TTE) revealed a large pericardial effusion containing echogenic material consistent with organized clot. Because the collection was largely organized, the classic echocardiographic signs of tamponade, such as chamber collapse, were absent; the hemodynamic compromise was nevertheless attributed to the hemopericardium, and compression of the right-sided chambers was later confirmed by intraoperative echocardiography. Left ventricular ejection fraction (LVEF) was 45%-50%, with lateral and apical akinesia (Figure *2A*). The combination of a late-presenting infarction, syncope, and hemopericardium raised the suspicion of LVFWR. Because acute aortic dissection with hemopericardium could not be excluded, particularly in an anticoagulated patient, urgent contrast-enhanced thoracic CT was performed. It showed a circumferential pericardial collection up to 60 mm thick in the right anterolateral recess, with attenuation consistent with hemopericardium, and no aortic dissection (Figures *2B*, *2C*). Subacute post-infarction LVFWR was therefore strongly suspected, although the diagnosis could be confirmed only at surgery.

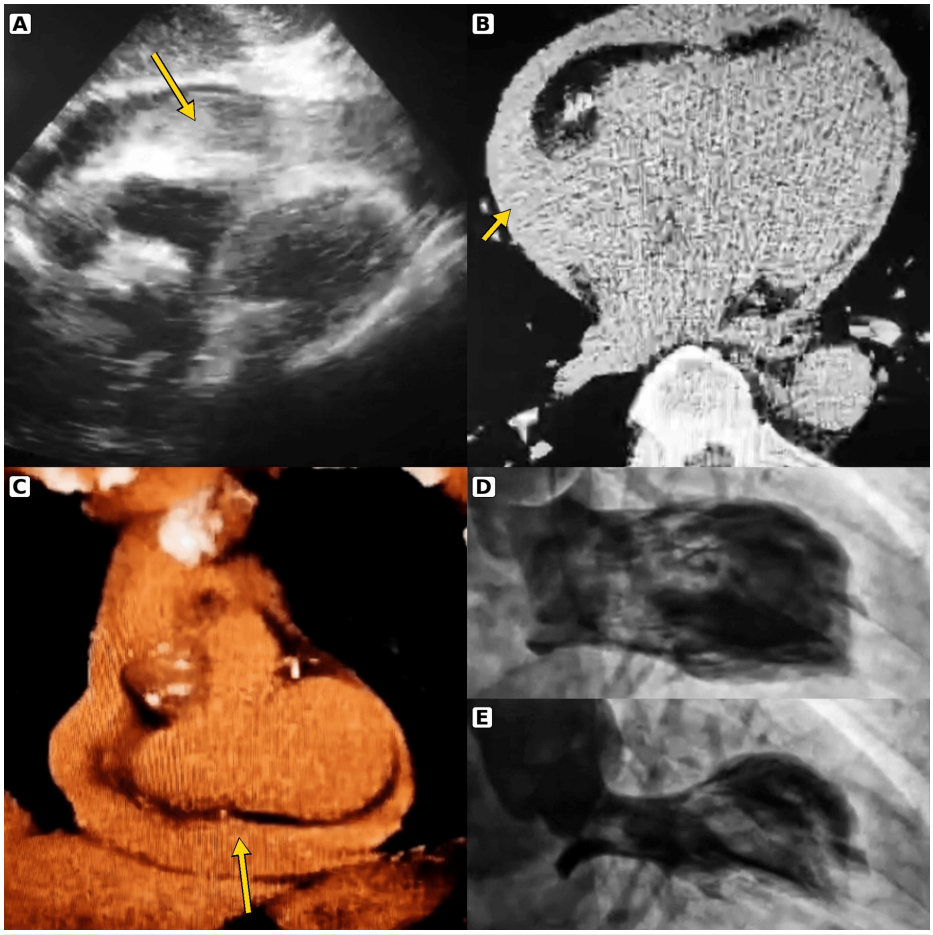



**FIGURE 2: Multimodality imaging.**

(A) Subcostal transthoracic echocardiographic view showing a large pericardial collection consistent with organized hemopericardium (arrow). (B) Axial contrast-enhanced CT image showing the circumferential hemopericardium (arrow); no aortic dissection was identified. (C) Three-dimensional volume-rendered CT reconstruction showing the hemopericardium below the heart (arrow). (D, E) Left ventriculography in diastole (D) and systole (E) showing anterolateral akinesia without contrast extravasation. CT: computed tomography.

## Initial management and transfer

Norepinephrine was started before CT, and pericardiocentesis was deliberately avoided because rupture was suspected; bedside TTE and CT were completed within approximately two hours of arrival. Antiplatelet and heparin loading were withheld. Idarucizumab was not administered, because the hemopericardium was attributed to a mechanical complication of the subacute infarction rather than to anticoagulant-related bleeding. The patient was transferred emergently to a cardiac surgical center, where coronary angiography showed occlusion of the first diagonal branch at its origin and a long, eccentric, approximately 70% stenosis of the mid left anterior descending artery (LAD); the right coronary artery had no significant obstructive disease apart from a moderate lesion of the posterolateral branch. Left ventriculography was performed to confirm containment of the rupture and showed anterolateral akinesia without contrast extravasation (Figures *2D*, *2E*).

## Surgical management

The patient underwent emergency surgery in profound cardiogenic shock. Intraoperative echocardiography confirmed a large mixed fluid-and-clot pericardial collection compressing the right-sided chambers, and LVEF was 40%-45%. Through a median sternotomy, approximately 500 mL of blood and clots were evacuated. A 1 × 1 cm macroscopic tear was identified in the mid-lateral left ventricular free wall, within the first diagonal territory, without active bleeding, which confirmed a spontaneously sealed rupture. The defect was repaired with a sutureless technique, using a hemostatic patch and surgical adhesive reinforced with a pericardial patch, and off-pump grafting of the left internal mammary artery to the LAD was performed.

## Postoperative course and follow-up

The postoperative course required inotropic support. Within the first five postoperative days, bedside

echocardiography detected recurrent hemorrhagic tamponade, which required surgical re-exploration and mediastinal drainage. Recovery was thereafter uneventful. The patient was transferred to the ward approximately two weeks after the first operation and was discharged one week later with LVEF at the lower limit of normal. After discharge, the patient received aspirin, clopidogrel, and apixaban 5 mg twice daily, together with ezetimibe/atorvastatin, insulin, and metformin; triple therapy was chosen by the cardiac surgeon because of the high ischemic risk in a patient with multiple risk factors, and after six months antiplatelet therapy was discontinued and apixaban was continued alone. At 18-month follow-up, the patient was alive, and echocardiography showed an LVEF of 50%, with no pericardial effusion or pseudoaneurysm.

## Discussion

Our patient had the subacute form of LVFWR, in which the interval between rupture and hemodynamic collapse can be used for diagnosis and surgical repair. Two morphological types are classically described: the blow-out type, a macroscopic defect with brisk bleeding, and the oozing type, slow bleeding through infarcted myocardium without a discrete tear [7]. Oozing ruptures predominate in surgical series; in a single-center experience of 35 patients operated on over 30 years, they accounted for 82.8% of cases, and in-hospital mortality was 28.6% [12]. Operative mortality approaches one-third overall: it was 32% in a meta-analysis of 363 patients [13]. Our patient had a definite 1 × 1 cm tear without active bleeding, that is, a sealed macroscopic rupture, an intermediate form in which thrombus and pericardial containment plausibly provided the time needed for diagnosis, transfer, and repair [8]. No sac communicating with the ventricular cavity was found, which distinguishes this presentation from a pseudoaneurysm [5].

The ECG anticipated both the culprit vessel and the site of rupture. ST-segment elevation in leads I and aVL with reciprocal changes in the inferior leads is typical of first diagonal occlusion [14], and in our patient this pattern matched the angiographic culprit and the mid-lateral tear found at surgery. High-lateral infarction may produce subtle changes and is easily overlooked, which may contribute to late presentation and absent reperfusion, two of the main risk factors for rupture [5]. Bedside echocardiography was decisive: TTE established hemopericardium and regional dysfunction within minutes and later detected recurrent postoperative bleeding. Because the hemopericardium was organized, the classic echocardiographic signs of tamponade were absent despite shock and right-sided chamber compression at surgery, which shows that their absence does not exclude a hemodynamically significant clotted collection. CT was useful to exclude aortic dissection, an important differential diagnosis of hemopericardium in an anticoagulated patient, and angiography guided concomitant grafting. Nevertheless, surgery is the definitive treatment of LVFWR, and preoperative investigations should not delay it in unstable patients [5,6]. Left ventriculography, in particular, is usually avoided when rupture is suspected, because contrast injection may disrupt a fragile seal; in our patient, it was performed to confirm containment. Neither CT nor ventriculography showed contrast extravasation, which indicates that contrast imaging cannot exclude a sealed rupture.

The DOAC added a further layer of complexity. Dabigatran is cleared mainly by the kidneys, and its effect is prolonged in renal impairment; current recommendations extend its interruption before high-risk surgery to up to 96 hours when creatinine clearance is 30-49 mL/min [9,10]. In patients aged 75-80 years or with a creatinine clearance of 30-50 mL/min, the reduced dose of 110 mg twice daily may be considered when bleeding risk is high [9]; our patient, aged 76 years with a baseline creatinine clearance of approximately 39 mL/min, was taking 150 mg twice daily. The modest rise in serum creatinine at admission (from 1.6 to 1.84 mg/dL) suggests that impaired clearance was largely chronic, which makes drug accumulation plausible, although the actual residual activity could not be measured. With an unknown time of last intake, residual anticoagulant activity could not be excluded; this may have favored pericardial bleeding and complicated surgical hemostasis, without implying a causal role in the rupture itself. Antithrombotic loading should be withheld as soon as a mechanical complication is suspected, and targeted reversal with idarucizumab, which has an onset of action of 10-30 minutes and provides rapid reversal of dabigatran in patients requiring urgent surgery [10,15], should be considered before the operation. Hemodialysis and prothrombin complex concentrate are nonspecific alternatives when idarucizumab is not available [10].

Sutureless patch-and-glue techniques are attractive in fragile necrotic myocardium [7,16]; in the single-center series mentioned above, in-hospital mortality was similar after sutured and sutureless repair (29% and 25%, respectively) [12]. Sealing, however, does not guarantee stability: in a series of 35 patients treated with sutureless repair, re-rupture occurred in 17%, mostly within 24 hours, and was associated with worse survival [16]. Close echocardiographic surveillance therefore remains essential, as in our patient, in whom recurrent tamponade was detected at the bedside and treated promptly. This report has limitations. The operative findings at re-exploration were not documented, so we cannot determine whether postoperative bleeding originated from the repaired tear, from adjacent myocardium, or from another surgical source. Coagulation tests could not be obtained at presentation, and no specific dabigatran assay was performed, so the contribution of residual anticoagulation can be inferred only indirectly. Finally, idarucizumab was not given, because the bleeding was attributed to the mechanical complication rather than to anticoagulation; in retrospect, given the need for emergency surgery, targeted reversal could have been considered, and we cannot exclude that residual dabigatran activity contributed to postoperative bleeding.

## Conclusions

In elderly patients with a late-presenting AMI, syncope or shock with hemopericardium should be considered LVFWR until proven otherwise, and surgery should not be delayed by nonessential imaging. In high-lateral infarction, the ECG pattern of first diagonal occlusion can point to the likely site of rupture. In patients taking a DOAC, antithrombotic loading should be withheld as soon as a mechanical complication is suspected, and targeted reversal should be considered before surgery, especially when renal function is impaired. A spontaneously sealed rupture is not mechanically stable, and close echocardiographic surveillance after repair is warranted to detect recurrent bleeding early.

# 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:** Raffaele Carluccio, Fabio Minicucci

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**Drafting of the manuscript:** Raffaele Carluccio, Amalia Siracusa, Crescenzo Testa

**Critical review of the manuscript for important intellectual content:** Raffaele Carluccio, Amalia Siracusa, Giuseppe Cioffi, Crescenzo Testa, Fabio Minicucci

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## Disclosures

**Human subjects:** Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **AI use statement:** The authors used ChatGPT (OpenAI); Claude (Anthropic) as follows: language polishing, structure or formatting in Case Presentation, Figure 2. ChatGPT (OpenAI) and Claude (Anthropic) were used only for manuscript organization, English-language editing, and formatting to journal requirements; they did not generate clinical data, images, or scientific conclusions. All AI-edited text was reviewed line by line by the authors. Every clinical detail, laboratory value, measurement, and time interval was checked against the patient's medical records, imaging reports, and operative notes. Each reference was verified against the original article on PubMed, including authors, title, journal, year, volume, pages, and DOI, and each citation was checked to confirm that it supports the statement it accompanies. The annotation arrows in Figure 2 were added at the authors' direction, and their placement was confirmed by the authors on the original images. The authors take full responsibility for the content of the manuscript. The authors confirm that they have reviewed and verified all AI-assisted content and take full responsibility for the work. **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.

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