

Thrombosis and Left Intraventricular Spontaneous Contrast Complicating Ischemic Cardiomyopathy Clinical case

Kimball-Kaky EG^{1,2}, Mongo Ngamami SF^{1,2}, Makani Bassakouahou JK³, Kouala Landa CM^{1,2}, Taty RJ³, Ngolo Letomo K¹, Bakekolo RP^{1,2}, Baragou S^{4,5*} and Ellenga Mbolla BF^{1,2}

¹Cardiology Department B, University Hospital Center of Brazzaville, Republic of the Congo.

²Faculty of Health Sciences, Marien Ngouabi University, Brazzaville, Congo.

³Cardiology Department A, University Hospital Center of Brazzaville, Republic of the Congo.

⁴Cardiology Department, Sylvanus Olympio University Hospital, Lomé, Togo.

⁵Faculty of Health Sciences, Lomé, Togo.

Correspondence

Baragou S, Cardiology Department, Sylvanus Olympio University Hospital, Lomé, Togo.

Received Date: 28 April 2026

Revised Date: 30 May 2026

Accepted Date: 03 June 2026

Publication Date: 15 June 2026

Copyright

© 2026 CME Online Library. This is an open- access article distributed under the terms of the Creative Commons Attribution 4.0 International license.

Abstract

Introduction: Thrombosis and spontaneous left ventricular contrast are complications of various cardiomyopathies, both ischemic and non-ischemic. Their recognition, prevention, and treatment remain important due to the potential risk of thromboembolic complications.

Observation: a 36-year-old male taxi driver, single, and father of five, was admitted to the ward on February 1, 2026, for congestive heart failure.

This patient, a smoker, had been hospitalized the previous year at the Military Hospital for a non-Q-wave myocardial infarction complicated by congestive heart failure. His discharge medication regimen included a low-sodium diet, furosemide, ramipril, nebivolol, atorvastatin, and acetylsalicylic acid. He had been non-adherent to his prescribed treatment. Dyspnea, which had occurred 10 days prior, was the reason for this hospitalization.

The clinical examination revealed : tachycardia, a gallop rhythm, a blood pressure of 108/62 mmHg, painful hepatomegaly, and edema of the lower limbs.

Paraclinical examinations

-Echocardiography showed biventricular systolic dysfunction, a reduced left ventricular ejection fraction (26%), tetracavitary dilation, and a fixed thrombus at the apex of the left ventricle.

-In the blood tests, the BNP level was 1200 pg/ml.

-The treatment included: fluid and sodium restriction, enoxaparin followed by acenocoumarol, losartan, spironolactone, dapagliflozin, and rosuvastatin. On day 12, the following were noted: resolution of heart failure, persistence of the thrombus. Discharge was authorized, nebivolol was reintroduced, a direct oral anticoagulant (DOAC) was prescribed, and weekly follow-up was obtained. On day 57, the thrombus had disappeared, spontaneous contrast enhancement was observed, and the DOAC was continued. The patient was again lost to follow-up.

Conclusion: Echocardiography is fundamental in the diagnosis and monitoring of thrombosis and spontaneous intracardiac contrast. Patient education is essential in our setting.

Keywords

Echocardiography, Thrombosis, Spontaneous contrast, Left ventricle, Brazzaville..



INTRODUCTION

Intracardiac thrombosis, in general, is the formation of a blood clot in one or more heart chambers. Located in the left ventricle, it complicates various forms of cardiomyopathy, both ischemic and non-ischemic. Its recognition, prevention, and management remain important due to the potential risk of thromboembolic

complications.

In developed countries, in addition to echocardiography, other diagnostic examinations such as computed tomography (CT) and magnetic resonance imaging (MRI) are common practice [1,2]. This is not the case in sub-Saharan Africa, where data on intracardiac thrombosis are scarce and difficult to collect [3-7]. Nevertheless, some

Citation: Kimball-Kaky EG, Mongo Ngamami SF, Makani Bassakouahou JK, Kouala Landa CM, Taty RJ, et al. Thrombosis and Left Intraventricular Spontaneous Contrast Complicating Ischemic Cardiomyopathy Clinical case. Int J Cardiol Res Rev. 2026;3(1):1-4. DOI: 10.52106/3066-3431.1010.

hospital studies have estimated the frequencies to be between 0.48% and 8.39% [1,4,8].

We set out to report a case of thrombosis and spontaneous left intraventricular contrast, complicating ischemic cardiomyopathy, highlighting the contribution of echocardiography in diagnosis and monitoring.

CASE REPORT

A 36-year-old taxi driver, single and father of five, consulted the University Hospital Center (CHU) of Brazzaville on February 1, 2026, for dyspnea and edema of the lower limbs. This necessitated his hospitalization in the cardiology B ward for congestive heart failure.

This patient a smoker (6 packs per year) had been admitted to the Brazzaville Military Hospital a year prior for an acute myocardial infarction (MI) without a Q wave in the anteroapical territory, complicated by global heart failure. His discharge medication regimen included a low-sodium diet, furosemide 20 mg/day, losartan 50 mg/day, acetylsalicylic acid 100 mg/day, rosuvastatin 20 mg/day, and nebivolol 2.5 mg/day. It should be noted that this patient admitted to non-adherence to his treatment and was subsequently lost to follow-up. The current episode began ten days before admission with the recurrence of dyspnea, followed by hepatic pain and edema of the lower limbs. He consulted at the Brazzaville University Hospital and was admitted to the department.

The clinical examination revealed: normal consciousness, good skin and mucous membrane color, a temperature of 37°C, a weight of 70 kg, and a height of 168 cm, corresponding to a body mass index of 24 kg/m². Severe physical asthenia and anorexia were noted. Oxygen saturation on room air was 98%, the WHO performance status was 2, and urine output was 3500 ml/24 hours.

The chest was of normal configuration, the pulse regular at 100 beats/min, and the blood pressure 108/62 mmHg. There was tachycardia, a protodiastolic gallop rhythm, and crackles at the lung bases. The remainder of the examination revealed spontaneous jugular vein distension, painful hepatomegaly (liver span = 21 cm), hepatojugular reflux, and localized edema of the forefeet and malleoli. There was no Homans' sign.

Paraclinical Examinations

- The frontal chest X-ray showed cardiomegaly (cardiothoracic ratio = 60%), elevation of the right hemidiaphragm, and venous redistribution to the apices.
- The standard 12-lead electrocardiogram showed sinus tachycardia at 100 beats/min and a QS pattern in the anteroapical region.
- Two-dimensional apical four-chamber transthoracic echocardiography (TTE) (Figure 1), revealed : a) a large, fixed, left intraventricular thrombus located at the apex, adhering to the wall, measuring 34.73 x 25.61 mm; dilation of all four cardiac chambers and the inferior vena cava; and global hypokinesis, predominantly in the anteroapical and apicolateral territories. Left ventricular systolic function was reduced (LVEF = 26%), and pulmonary arterial pressure was elevated (mild right ventricular systolic dysfunction (TAPSE = 14 mm).
- The blood tests were normal, except for a BNP level of 1,200 pg/ml.

- Initial treatment included: a low-sodium diet and fluid restriction; furosemide 40 mg parenterally, then orally at the same dosage; ramipril 2.5 mg; enoxaparin 0.8 ml twice subcutaneously followed by

acenocoumarol 4 mg; dapagliflozin 10 mg; rosuvastatin 20 mg; and spironolactone 25 mg.

-At 12 days' admission, the patient's condition was characterised by: the resolution of signs of heart failure, and the persistence of the thrombus. Discharge was authorised, with the patient's medication regimen adjusted to include nebivolol 2.5 mg/day and rivaroxaban (a direct oral anticoagulant) in place of Sintrom, to ensure less restrictive and less costly follow-up for the patient. At day 57 of anticoagulant therapy, echocardiography revealed: the disappearance of the apical thrombus, and the presence of spontaneous contrast in the left ventricle (Figure 2). This necessitated the continuation of direct oral anticoagulants (DOACs) and the prescription of weekly echocardiographic monitoring. However, to date, this non-compliant patient has once again been lost to follow-up.

Comments

-Left intraventricular thrombosis is the formation of a blood clot within this heart chamber. In our patient's case, it was discovered incidentally during an echocardiographic assessment of global heart failure complicating ischaemic cardiomyopathy.

-Generally speaking, in sub-Saharan Africa, the incidence of intracardiac thrombosis ranges from 0.48% to 8.39% of cases [1,4,8]. The condition affects both sexes [3-5,8,9], with a predominance of male cases in some series [3,4,9]. Our patient was male. The condition affects both young and elderly people [3-6,9]. Our patient was 36 years old and had several cardiovascular risk factors.

Cardiovascular risk factors (RFs) and factors contributing to thrombus formation

- In Abidjan, Ziguinchor in Senegal, and Cotonou [3,5,6], the most common cardiovascular risk factors were high blood pressure and smoking. In our young patient, in addition to heavy smoking (6 packets per year), stress was a significant RF, as evidenced by: his occupation as a taxi driver, his single status and the responsibility for his five children. These cardiovascular RFs likely contributed to the occurrence of the AMI one year prior.

-The risk factors for the formation of a left intraventricular thrombus are: ventricular dilatation, left ventricular (LV) systolic dysfunction, segmental hypokinesis and a reduced left ventricular ejection fraction (LVEF) [2-6,8, 9]. We also observed these in our patient. They constitute an important indicator of blood stasis and thrombotic risk.

-Transthoracic echocardiography (TTE) remains the first-line method. It is the most accessible and rapid diagnostic tool, particularly in our context, for the initial detection of thrombi, their number and any mobility [3-6,9]. In ischaemic cardiomyopathy, TEE also allows assessment of EF and segmental kinetics, detection of aneurysms and evaluation of global left ventricular remodelling.

-Echocardiographic signs of a thrombus include a hyperechoic mass with sharp margins, distinct from the endocardium, visible in at least two views and persisting throughout the cardiac cycle [1]. Delewi R et al. [2] noted a left ventricular thrombus in 39% of anterior MI cases. The same finding was reported in Abidjan by Soya E et al. [3] in a quarter of their patients. In our patient who presented with an apical anterior septal MI, the thrombus was apical, solitary, adherent to the wall and measuring 34.73 by 25.61 mm. There was dilatation of all four cardiac chambers, global hypokinesis, predominantly in the anteroapical-basal and apical-lateral regions, indicating severe LV systolic dysfunction (EF = 26%). This led to the diagnosis of ischaemic cardiomyopathy (ICM).

Clinical and Paraclinical Findings

The tell-tale clinical signs in our patient were: dyspnoea, hepatic pain and oedema of the lower limbs, indicative of a syndrome of global heart failure associated with IHD. Echocardiography, as noted above, enabled visualisation of the intra-LV thrombus, as in several other studies [3-6,8]. Among non-invasive radiological investigations, CT and, better still, MRI, play a significant role in the diagnosis of an intracardiac thrombus [1,2,10-12]. However, it is worth highlighting, in our context, the disadvantages for our patients in terms of cost and accessibility.

Therapeutic and prognostic aspects

The first-line treatment for intracardiac thrombus is systemic anticoagulation, whilst taking into account the management of the underlying heart condition. Immediate treatment aims to minimise the risk of thromboembolic events. In hospital, the administration of parenteral heparin combined with a vitamin K antagonist (VKA) remains the best option until therapeutic anticoagulation is achieved [1,3-6,8,9,13,14]. We have also adopted this approach. However, an OAC was preferred upon the patient's discharge, sparing them the burden of laboratory tests, whilst adhering to current guidelines [15-17]. Indeed, it is now established that the efficacy of DOACs appears to be even superior to that of VKAs in the management of intracardiac thrombi, subject to prospective studies to confirm these findings.

The echocardiographic scan performed on day 57 of anticoagulant therapy in our patient revealed that the apical thrombus within the left ventricle had disappeared. Authors from Senegal [5] and Guinea [4] had reported the complete disappearance of the left ventricular thrombus after 3 months of anticoagulant therapy in 60% and 64.7% of cases, respectively. However, in our case, the follow-up TEE revealed spontaneous contrast in the LV. In echocardiography, this phenomenon is a marker often associated with blood stasis. It is generally an important indicator of thrombotic risk [1]. Its detection is crucial, as it may signal an increased risk of thrombus formation, thereby increasing the risk of embolic events.

Spontaneous contrast echocardiography presents as dense, mobile echoes exhibiting circular or spiral motion, swirling within the left ventricle (Figure 2) and resembling wisps of smoke ("swirling smoke"). This situation necessitated the continuation of anticoagulant therapy with DOACs in our patient and the continuation of weekly outpatient echocardiographic monitoring. The course of the condition was characterised by stable haemodynamics and the persistence of spontaneous contrast within the LV. This led to the continuation of anticoagulant therapy with DOACs. However, we have had no news of the patient for the past fortnight. The persistence of spontaneous contrast enhancement beyond the third month of anticoagulant therapy may be due to left ventricular dilatation and significant segmental hypokinesis, associated with IHD and reduced LVEF. Furthermore, in this patient of modest social means, who has once again been lost to follow-up, non-adherence to treatment cannot be formally ruled out. Consequently, patient education in our context presents a real challenge. Moreover, the promotion of health insurance by the public authorities is crucial.

CONCLUSION

Left intraventricular thrombus is a serious complication often associated with dilated cardiomyopathy, characterised by a reduced ejection fraction. Diagnosis relies on transthoracic echocardiography, which is the primary diagnostic tool available in developing countries. Treatment involves long-term anticoagulation with vitamin K antagonists or direct oral anticoagulants. Intracardiac thrombosis

in general should be given greater attention and systematically investigated due to the high risk of systemic embolism and morbidity and mortality.

Consent

The patient has given their informed written consent for the publication of this case report.

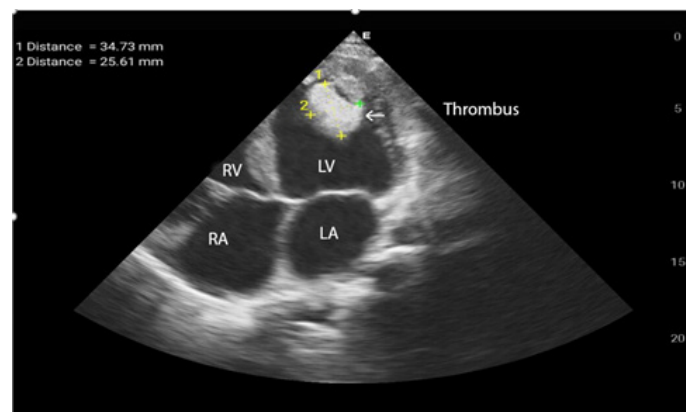


Figure 1: Two-dimensional transthoracic echocardiography. Apical view of the four chambers. Dilated left ventricle, containing a fixed apical thrombus.

Legend : LA = left atrium; LV = left ventricle; RA = right atrium; RV= right ventricle

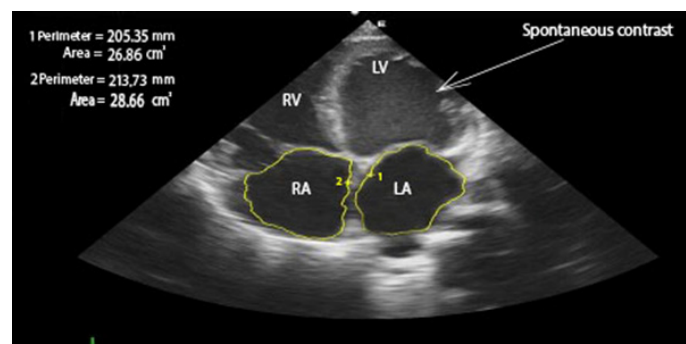


Figure 2: Two-dimensional transthoracic echocardiography. Apical view of the four chambers. Disappearance of the apical thrombus in the left ventricle. Presence of spontaneous contrast in the left ventricle.

Legend: LA = left atrium; LV = left ventricle; RA = right atrium; RV= right ventricle

REFERENCES

1. Deux JF, Mayer J, Colombier D, Guendouz S, Lapeyre M, et al. Masses and Thrombi. In: Cardiac Imaging: CT and MRI. Elsevier. 2011; 205-230.
2. Delewi R, Nijveldt R, Hirsch A, Marcu CB, Robbers L, et al. Left ventricular thrombus formation after acute myocardial infarction as assessed by cardiovascular magnetic resonance imaging. *Eur J Radiol.* 2012; 81: 3900-3904.
3. Soya E, Angoran I, Gbassi C, Koffi F, Avoh A, et al. Intracardiac thrombosis: Aspects Épidémiologiques, Cliniques, Echocardiographiques, Thérapeutiques et Evolutifs à Abidjan. *Health Res Afr.* 2024; 2: 16-19.
4. Baldé EY, Baldem D, Beavogui M, Barry IS, Kimso O, et al. Thrombus intracavitaire: aspects épidémiologique, clinique, échographique et évolutif au service de Cardiologie de l'Hôpital

- National Ignace Deen de Conakry. Tropical cardiology. 2014; 2014; 140: 59.
5. Kwatar EB. Intracardiac thrombose: épidémiologiques, diagnostics, therapeutics and evolution aspects at the Hôpital de la Paix de Ziguinchor. 2022.
 6. Houenassi DM, Tchabi Y, Sacca-Vehoukpé J, Akindès Dossou-Yovo R, Kohoun RK, et al. Echographic study of thrombose and spontaneous intra-auriculaire contrast in patients with auricular fibrillation. Tropical Cardiology. 2013; 131: 20.
 7. Kane AD, Ndiaye MN, Mbaye A, Diao M, Mboup C, et al. Screening for intracardiac thrombi in peripartum cardiomyopathy. Médecine d'Afrique Noire. 2010; 5704: 203-211.
 8. Ralamboson SA, Miandrisoa RM, Riel A, Rakotomanga D, Rajomarison M, et al. Epidemiological clinical and ultrasound aspects of cardiac intracavitary thrombosis seen in the Hospital Center of Soavinandriana. Rev méd Madag. 2011; 1: 58-62.
 9. Tagueniti J, Samih A, Raissouni M, Benyass A. Intracardiac thrombi: positive and etiological diagnosis. Cardiology Center, Mohammed V Military Teaching Hospital, Rabat, Morocco. 408-2019-64.pdf.
 10. Camaj A, Fuster V, Giustino G, Bienstock SW, Sternheim D, et al. Left Ventricular Thrombus Following Acute Myocardial Infarction: JACC State-of-the-Art Review. J Am Coll Cardiol. 2022; 79: 1010-1022.
 11. Meurin P, Carreira B, Dumaine V, Shqueir R, Milleron A, et al. Incidence, diagnosis methods, and evolution of left ventricular thrombus in patients with anterior myocardial infarction and low ventricular ejection fraction: a prospective multicenter study. Am Heart J. 2015; 170: 256-262.
 12. Paul T, Vaitkus PT, Elliot S, Facc MD. Embolic potential, prevention and management of mural thrombus complicating anterior myocardial infarction: a meta-analysis. J Am Coll Cardiol. 1993; 22: 955-962.
 13. Renotte T, Tamakloe T, Mariage JL, Michel X, Gubin B, et al. Diagnostic approach and management of left intraventricular thrombus complicated by unexpected embolism : A case report. Ann Cardiol Angeiol. 2025; 74: 101863.
 14. Guyatt GH, Akl EA, Crowther M, Gutterman DD, Schünemann HJ. Executive summary: antithrombotic therapy and prevention of thrombosis, 9th ed; American College of Chest Physicians evidence-based clinical practice guidelines. Chest. 2012; 141: 75-475.
 15. Iqbal H, Straw S, Craven TP, Stirling K, Wheatcroft SB, et al. Direct oral anticoagulants compared to vitamin k antagonist for the management of left ventricular thrombus. ESC Heart Fail. 2020; 7: 2032-2041.
 16. Huang L, Tan Y, Pan Y. Systematic review of efficacy of direct oral anticoagulants and vitamin k antagonists in left ventricular thrombus. ESC Heart Fail. 2022; 9: 3519-3532.
 17. Backaus JF, Pflaumbaum A, Krogias C, Kreimer F, Mügge A, et al. Short-and long-term outcome of patients with spontaneous echocontrast or thrombus in the left atrial appendage in the era of the direct acting anticoagulants. Clin Res Cardiol. 2021; 110: 1811-1821.