ESC European Society of Cardiology

European Heart Journal - Case Reports (2025) **9**, ytaf040
https://doi.org/10.1093/ehjcr/ytaf040

CASE REPORT
Cardiovascular imaging

Myocardial infarction due to non-bacterial thrombotic endocarditis in a patient with oligodendroglioma: an unusual presentation and diagnostic challenge—a case report

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Received 9 July 2024; revised 23 October 2024; accepted 20 January 2025; online publish-ahead-of-print 4 February 2025

Background Non-bacterial thrombotic endocarditis (NBTE) represents a manifestation of thrombotic endocarditis characterized by the formation of thrombus on the heart valve leaflet. While neurological events are the most frequent initial presentation of NBTE, myocardial infarction also stands out as a major clinical manifestation among NBTE patients.

Case summary A 61-year-old female with no history of cardiovascular disease or episodes of chest pain suffered a left main trunk ST-segment elevation myocardial infarction (STEMI) after craniotomy for the treatment of oligodendroglioma. A comprehensive diagnostic workup, including coronary angiography, revealed no evidence of embolism. However, autopsy findings, in conjunction with cardiac ultrasound, contrast-enhanced computed tomography, and transoesophageal echocardiogram results, conclusively demonstrated that NBTE had obstructed the left main trunk of the coronary artery, leading to STEMI.

Discussion Non-bacterial thrombotic endocarditis should be strongly considered in the differential diagnosis for patients presenting with acute cerebrovascular events or coronary ischaemia, especially in the presence of predisposing factors such as a history of malignancy, systemic inflammation, or embolic phenomena of indeterminate origin. This consideration is critical for the timely identification and management of NBTE, ultimately mitigating the risk of severe complications and optimizing patient outcomes.

Keywords Non-bacterial thrombotic endocarditis • Myocardial infarction • Myocardial infarction with non-obstructive coronary arteries • Ventricular fibrillation • Oligodendroglioma • Case report

ESC curriculum 6.9 Cardiac dysfunction in oncology patients • 3.2 Acute coronary syndrome • 5.6 Ventricular arrhythmia • 4.11 Endocarditis

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Handling Editor: Massimo Mapelli
Peer-reviewers: Claudio Picariello; Armen Kocharian
Compliance Editor: Lucy Zheng

Learning points

- Understand the pathophysiology of NBTE and the mechanisms leading to thrombus formation.
- Recognize the importance of including NBTE in the differential diagnosis, particularly for patients presenting with unprovoked embolism, or those with underlying hypercoagulable states associated with malignancy and systemic inflammation.
- Understand the management and treatment options for NBTE, focusing on anticoagulation therapy to prevent recurrent emboli, treating the underlying cause, and exploring the potential surgical intervention in select cases.
- Understand the global increase in cancer incidences and the heightened need for increased awareness and proficiency in diagnosing and managing NBTE within the realm of oncology.

Introduction

Non-bacterial thrombotic endocarditis (NBTE) represents a manifestation of thrombotic endocarditis characterized by the formation of thrombus on the heart valve leaflet,[1] particularly on the aortic and mitral valves.[2,3] These thrombotic vegetations are typically composed of platelets, fibrin, and inflammatory cellular elements. Non-bacterial thrombotic endocarditis is thought to be a result of endothelial damage and hypercoagulable states, including advanced malignancies, systemic inflammatory conditions such as sepsis, antiphospholipid syndrome, rheumatic heart disease, collagen vascular diseases, and disseminated intravascular coagulation (DIC).[1]

We present a unique case of NBTE where, rather than typical coronary embolism,[4] thrombotic mass at the aortic valve root obstructed the left main coronary artery, which led to extensive myocardial ischaemia and required complex therapeutic decision-making.

Summary figure

Time	Events
8 years ago	First craniotomy for oligodendroglioma
Day 1	Patient underwent a second craniotomy for recurrent oligodendroglioma.
Day 72	Myocardial infarction occurred, and the patient was resuscitated for ventricular fibrillation (VF). Subsequently, intra-aortic balloon pump (IABP) and extracorporeal membrane oxygenation (ECMO) were inserted. A substantial mass on the left coronary cusp was discovered, and heparinization therapy was initiated. She remained unconscious after being resuscitated.
Day 80	Even after sedation was discontinued, the patient remained unconscious. A follow-up computed tomography (CT) scan showed a massive ischaemic stroke.
Day 82	Left ventricular ejection fraction (LVEF) gradually improved from 10% to 35%. IABP was removed.
Day 86	ECMO was removed. Palliative care was initiated.
Day 95	Tracheotomy was performed.
Day 103	Patient deceased.
Day 104	Autopsy was performed.

Case presentation

A 61-year-old female with no history of cardiovascular disease or chest pain episodes underwent craniotomy for recurrent oligodendroglioma (IDH-mutant, 1p/19q-codeleted, grade 3 glioma). There were no signs of systemic metastasis on the positron emission tomography scan, and the tumour was completely resected with clear margins collectively suggesting a favourable prognosis. Post-operatively, the patient received chemotherapy with vincristine, nimustine hydrochloride (ACNU), carboplatin, and interferon-β, along with radiotherapy (VAC-feron-R). On the 72nd post-operative day, she experienced chest pain. While being transferred to the emergency department, she went into VF, requiring immediate resuscitation. Post-resuscitated electrocardiogram demonstrated ST-segment elevation in aVR and ST-segment depression in II, III, aVF, and V4–V6 (*Figure 1A*). The transthoracic echocardiogram (TTE) findings revealed akinesis in the anterior, lateral, and posterior walls, leading to a critically reduced LVEF of ∼10% (Supplementary material online, *Video S1*). Her cardiac enzymes including creatine kinase (CK), CK-MB, and troponin T, taken just before the patient went into VF, were within the normal range, and her C-reactive protein level was also negative. Due to persistent VF and pulseless electrical activity, urgent ECMO and IABP insertion were performed for haemodynamic stabilization. A subsequent coronary angiography revealed patent coronary arteries without any evidence of coronary stenosis or thrombotic occlusion (*Figure 1B*). A contrast-enhanced CT scan immediately after coronary angiography showed a significant mass on the left coronary cusp (*Figure 2A*), and a further examination using transoesophageal echocardiography (TEE) identified a $10 \times 10 \times 8$ mm mass at the same location (*Figure 2B*; Supplementary material online, *Video S2*). She remained unconscious, with a Glasgow Coma Scale score of 3, after being resuscitated.

The differential diagnosis for the mass included infectious endocarditis, malignant tumours, myxoma, NBTE, papillary fibroelastoma, and Lambl's excrescence. The absence of infectious symptoms and negative blood cultures excluded the possibility of infectious endocarditis. Laboratory tests for antinuclear antibodies were negative, and levels of proteins C and S were normal, effectively ruling out chronic systemic inflammatory conditions. After admission to the care unit, her CK, CK-MB, and troponin T rose, peaking at 7383 U/L, 432 U/L, and over 10 ng/mL, respectively. Despite heparinization therapy, the mass showed no reduction in size. With a significantly reduced LVEF, the patient relied on ECMO for haemodynamic stabilization. According to the Endocarditis Guideline 2023,[5] the patient was consequently referred to cardiovascular surgery. However, the surgical plan was ultimately cancelled when a follow-up head CT scan revealed a massive ischaemic stroke. The patient was medically treated, and although the mass' size remained unchanged, there were no subsequent MI events. The patient's LVEF recovered from 10% to 35%, achieving haemodynamic stability and allowing for the discontinuation of both the ECMO and IABP within a 2-week timeframe. Despite these cardiac improvements, the patient remained unconscious, prompting the initiation of palliative care, and she passed away on Day 103.

An autopsy revealed a pedunculated mass, anchored to the left coronary cusp of the aortic valve (*Figure 3*). Microscopic evaluation using both Gram and periodic acid-Schiff (PAS) stains detected no bacterial presence. The mass was characterized by a lack of structural

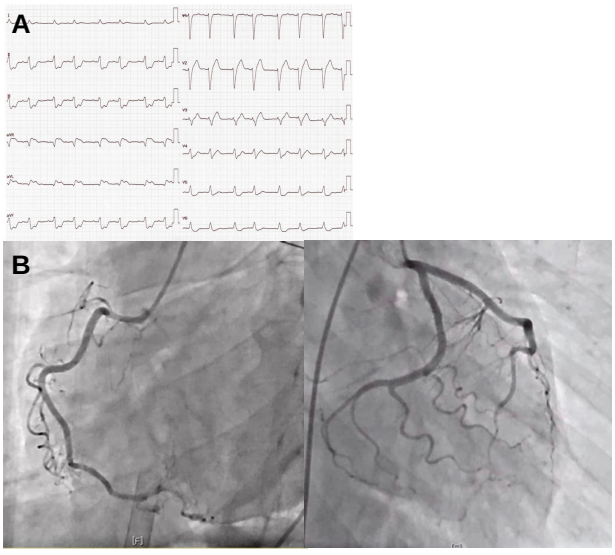

Figure 1 (*A*) Electrocardiogram. ST-segment elevation in lead aVR and ST-segment depression in leads II, III, aVF, and V4–V6. (*B*) Coronary angiogram. There was no evidence of coronary artery occlusion or stenosis.

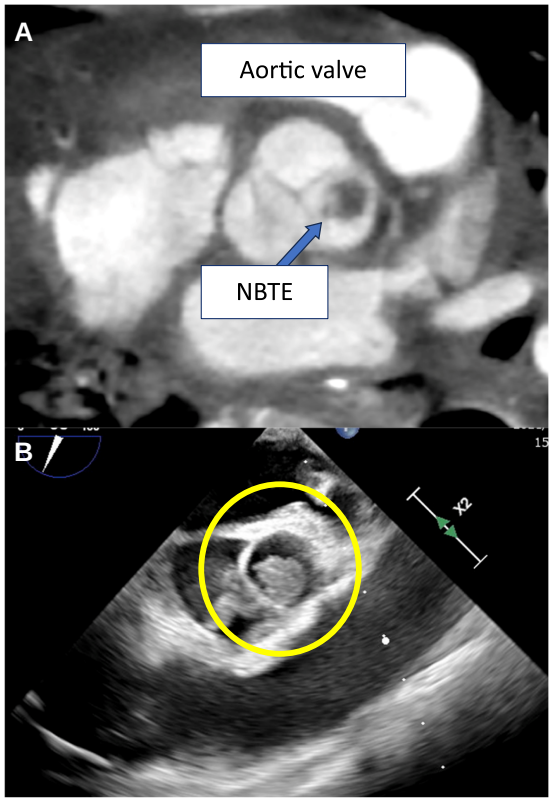

Figure 2 (*A*) Contrast-enhanced computed tomography. A mass on the left coronary cusp (arrow). (*B*) Transoesophageal echocardiogram. Intermittent obstruction of the left coronary artery ostium occurred due to the transient proximity of the mobile mass (10 × 10 × 8 mm) (circle).

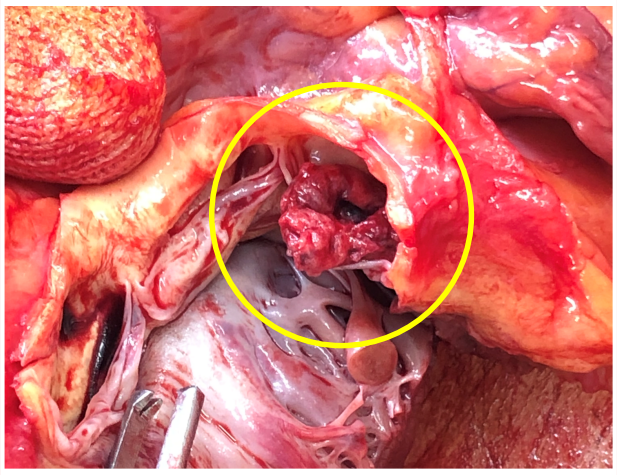

Figure 3 Autopsy (macroscopic). The mass was discovered on the left coronary cusp (circle).

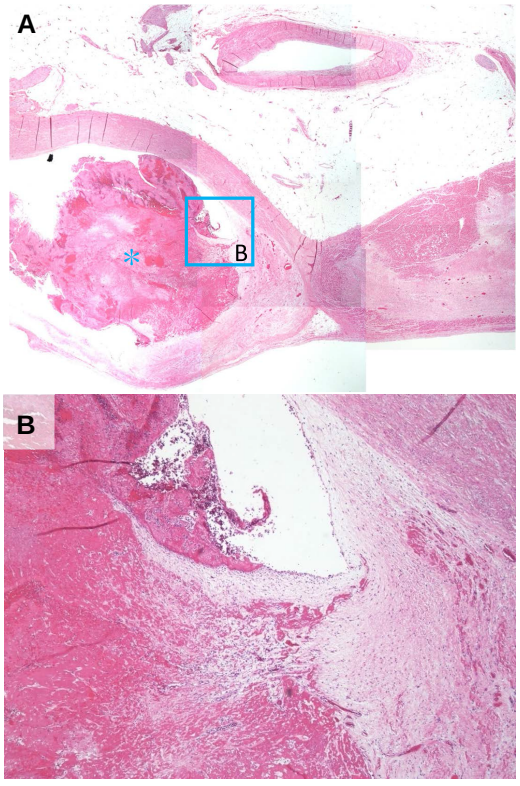

Figure 4 Autopsy (microscopic, haematoxylin eosin stain). (*A*) There were no bacterial presence, and the mass was characterized by a lack of structural organization. (*B*) The mass consisted of white clots made up of platelets and fibrin.

organization and was predominantly composed of platelets (*Figure 4*). These comprehensive pieces of evidence led to the final diagnosis of NBTE. Additionally, a scar was identified on anterior and lateral walls

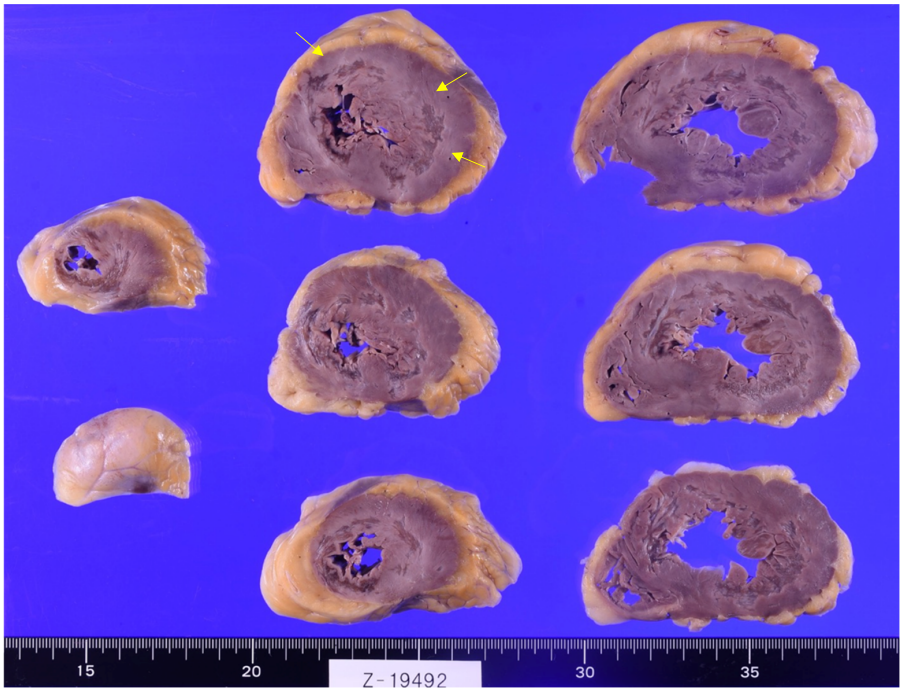

Figure 5 Myocardial ischaemia. The scar was identified on the anterior, lateral, and posterior walls of the left ventricle.

of the left ventricle (*Figure 5*), indicative of myocardial ischaemia associated with the left coronary artery. Notably, no thrombus or arteriosclerosis lesion was found in any coronaries. Macroscopic and histological examination of the brain showed evidence of multiple cerebral infarctions, corresponding to the regions identified by the head CT scan. In addition, white thrombi were found within the arteries of the otherwise normal brain parenchyma, suggesting the possibility that the mass found at the aortic valve had detached and embolized shortly before death.

Discussion

The reported prevalence of NBTE ranges from 0.9% to 1.6%, although its true incidence remains elusive as it is often discovered incidentally during post-mortem autopsies.[1] The aortic valve is the most affected (46.1%), followed by the mitral valve (40.6%), with concurrent involvement of both valves occurring in 8.3%.[3] Thus, it is crucial to add NBTE in differentiation diagnosis and thoroughly evaluate the left heart system in patients at high risk of NBTE, those with unexplainable thrombotic events, or in individuals experiencing recurrent thrombotic events despite anticoagulation therapy.

As mentioned earlier, NBTE is thought to result from endothelial damage and hypercoagulable states. Among malignancies, gliomas are known to induce a hypercoagulable state, significantly increasing the risk of thrombotic events.[6] In particular, oligodendroglioma, classified as a grade 3 glioma, is frequently associated with venous thromboembolism. Despite the lack of documented cases directly linking gliomas with arterial thrombosis or NBTE, the presence of oligodendroglioma is considered the probable aetiological factor in our case, attributed to its inherent capacity to promote a hypercoagulable condition.[6]

One of the major complications associated with NBTE is the heightened risk of systemic embolism. Notably, while neurological events are the most frequent initial presentation of NBTE, MI also stands out as a major clinical manifestation in patients with NBTE.[2,7–9] It is important to note that this can occur even in the absence of significant coronary artery stenosis, underscoring that MI in the context of NBTE is predominantly due to embolic events originating from the NBTE lesions themselves, a phenomenon referred to as myocardial infarction with non-obstructive coronary arteries (MINOCA).[5]

In our specific case, a comprehensive diagnostic workup revealed no evidence of embolism. However, autopsy findings, corroborated by cardiac ultrasound, contrast-enhanced CT, and TEE results, conclusively demonstrated that NBTE caused obstruction in the left main trunk, leading to MI. This represents the first documented case of such a unique scenario of ST-segment elevation myocardial infarction (STEMI) associated with NBTE.

Anticoagulation therapy stands as the primary treatment for NBTE. Due to a scarcity of comprehensive data, heparin, both unfractionated and low-molecular weight, is generally preferred as the anticoagulant of choice.[10] Surgical intervention is contemplated only under specific conditions such as in patients with tumours larger than 10 mm despite heparin therapy and in those experiencing recurrent embolic events. However, the decision to proceed with surgery is complicated by the high mortality rate associated with surgery coupled with the frequent presence of underlying carcinoma in these patients, making the surgical option clinically challenging in many cases. Regardless of surgical treatment, the prognosis for NBTE remains poor, with a reported 1-year survival rate of ~66% and an even more dismal prognosis in cases associated with underlying malignancy.[11]

Conclusion

To the best of our knowledge, this is the first report to describe STEMI caused by cancer-related NBTE. The timely recognition and appropriate management of NBTE are crucial for preventing potentially life-threatening complications and optimizing patient outcomes. By raising

awareness of this atypical presentation, our report emphasizes the need for considering NBTE in the differential diagnosis and adopting a multidisciplinary approach in the care of patients with malignancy who develop thrombotic complications.

Lead author biography

Yuto Miura graduated from Kanazawa University, Japan, in 2018. He is currently working as a fellow in the Department of Heart Failure at the National Cerebral and Cardiovascular Center, Japan. His main areas of interest are heart failure, cardio-oncology, and preventive medicine.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

Acknowledgements

We would like to express our gratitude to Mr John Martin for his grammatical assistance.

Consent: The authors confirm that written consent for submission and publication of this case report including the images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

Funding: None declared.

Data availability

The data underlying this article will be shared on reasonable request to the corresponding author.

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