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CASE REPORT

Myocardial Infarction with Non-obstructive Coronary Arteries; Diffuse Large B-Cell Lymphoma Presenting as an Acute Coronary Syndrome

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Abstract: A 69-year-old man presented with sudden-onset crushing chest pain, diaphoresis, and syncope with ECG findings prompting treatment as a ST-elevation myocardial infarction (STEMI). Emergency coronary angiography demonstrated no obstructive coronary artery disease. Subsequent investigations including cardiac magnetic resonance imaging (MRI) and positron emission tomography (PET) scan demonstrated an infiltrative mass. Biopsy of a supraclavicular lymph node established a diagnosis of diffuse large B-cell lymphoma (DLBCL) with cardiac involvement. The patient was treated with corticosteroids and immunochemotherapy (DA-R-EPOCH), achieving complete remission on surveillance imaging. This case therefore contributes valuable evidence regarding the varied cardiovascular manifestations of lymphoma and reinforces the importance of maintaining a broad differential diagnosis in patients presenting with features of myocardial infarction with non-obstructive coronary arteries (MINOCA).

Keywords: Acute Coronary Syndrome; Emergency Medicine; Heart Neoplasms; Lymphoma, Large B-Cell, Diffuse; Magnetic Resonance Imaging; MINOCA

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1. Introduction

Patients presenting with chest pain, electrocardiographic evidence of myocardial ischemia, and elevated cardiac biomarkers are typically managed through expedited acute coronary syndrome pathways. However, a subset of these patients has no angiographically identifiable culprit lesion suitable for percutaneous coronary intervention (PCI), a condition referred to as myocardial infarction with non-obstructive coronary arteries (MINOCA) (1).

MINOCA represents a heterogeneous clinical entity with multiple potential underlying mechanisms, all of which lead to oxygen demand-supply mismatch of myocardium; including epicardial coronary artery spasm, coronary microvascular dysfunction, spontaneous coronary artery dissection, and coronary thromboembolism. In addition, myocarditis and Takotsubo cardiomyopathy are important differential diagnoses that may mimic MINOCA and should be excluded through appropriate diagnostic evaluation (2).

Cardiac neoplasms represent a very rare but important cause of MINOCA, particularly when accompanied by unexplained arrhythmias, myocardial wall thickening, or atypical imaging findings (2, 3). Diffuse large B-cell lymphoma (DLBCL) is the most common subtype of non-Hodgkin lymphoma and may rarely involve the heart through either primary cardiac lymphoma or secondary metastatic infiltration (4, 5). Although secondary cardiac involvement is identified in up to one-quarter of autopsy series of patients with advanced lymphoma, it remains infrequently recognized during life owing to its diverse and often non-specific clinical manifestations (6). Cardiac infiltration may involve the myocardium, pericardium, endocardium, or coronary vasculature and can present with heart failure, arrhythmias, pericardial effusion, conduction abnormalities, or, less commonly, symptoms mimicking acute coronary syndrome (6, 7).

We report a rare case of DLBCL presenting as presumed posterior ST-elevation myocardial infarction (STEMI). Multimodality imaging revealed infiltrative cardiac lymphoma causing myocardial injury and external coronary artery compression despite angiographically normal coronary arteries. This case highlights the diagnostic value of cardiac magnetic resonance imaging (MRI) and demonstrates the importance of maintaining a broad differential diagnosis in patients with STEMI like presentations and unobstructed coronary arteries.

1.1. *Case presentation*

The patient was a 69-year-old man who was awoken in the early hours of the morning by sudden-onset crushing central chest pain, associated with dyspnea and light-headedness. He had experiences two episodes of prehospital syncope. He denied any recent trauma, recreational drug use, or previous episodes of similar chest pain. He reported intermittent palpitations over the past few months, although these had not previously been investigated. Paramedics administered analgesia and glyceryl trinitrate, with partial symptomatic improvement.

His past medical history was significant for hypertension, renal colic, obesity, dyslipidemia, and hyperuricemia. His regular medications included lercanidipine, irbesartan, allopurinol, rosuvastatin, and meloxicam. He was an ex-smoker, having ceased tobacco use many years prior, and reported minimal alcohol intake. He was of Filipino heritage.

On arrival to the emergency department (ED), the patient was awake but appeared diaphoretic. Chest pain remained at 3/10. Initial observations were: respiratory rate 16/minute, SpO2

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93% on room air, heart rate 100-120 beats/minute (irregularly irregular), blood pressure 131/99 mmHg and afebrile. Examination revealed no increased work of breathing, irregular dual heart sounds with no murmur, strong peripheral pulses and equal blood pressures bilaterally. The jugular venous pressure was not elevated. Abdominal examination was normal, but with the presence of a pulsatile mass. There were no pedal edema or clinical features of deep vein thrombosis (DVT).

The first electrocardiography (ECG) demonstrated atrial fibrillation with a ventricular rate of approximately 90–110 beats per minute, with occasional premature ventricular complexes. There was diffuse ST-segment depression across the precordial leads, most marked in V4–V5 (Figure 1). These findings were concerning for acute coronary syndrome, particularly subendocardial ischemia. A posterior ECG was subsequently performed and demonstrated approximately 1 mm of ST-segment elevation in the posterior leads (Figure 2).

A limited bedside transthoracic echocardiogram (TTE) performed by the treating emergency physician demonstrated an ill-defined abnormality adjacent to the posterior cardiac wall. This finding could not be further characterized on the available images. There was no evidence of pericardial effusion or cardiac tamponade. No definite regional wall motion abnormality was identified, and there were no echocardiographic features suggestive of pulmonary embolism.

The initial troponin level was elevated at 811 ng/L, supporting myocardial injury. Venous blood gas analysis revealed mild lactic acidosis (3.9 mmol/L) and serum electrolytes were within normal limits.

After administration of loading doses of antiplatelet agents (aspirin + ticagrelor) and anticoagulant therapy (heparin), the patient was transferred to the cardiac catheterization laboratory with a working diagnosis of posterior STEMI.

Coronary angiography demonstrated no obstructive coronary artery disease and no identifiable target lesion for PCI. These findings prompted concern for an embolic event, particularly in the context of newly diagnosed atrial fibrillation.

A bedside TTE performed by the cardiology team demonstrated a normal-sized left ventricle (LV) with preserved systolic function and posterolateral hypokinesia, mild mitral regurgitation, a normal right ventricle, and a dilated left atrium. A formal TTE confirmed preserved LV systolic function and additionally demonstrated apparent inferolateral and anterolateral LV wall thickening, raising concern for pericardial effusion, hematoma, or intramyocardial hematoma.

During the first 24 hours following admission to the cardiac care unit (CCU), he experienced a self-terminating episode of tachyarrhythmia, documented as alternating irregular broad-complex tachycardia and irregular narrow-complex tachycardia, without associated symptoms. Later the same day, he developed pulseless ventricular tachycardia (VT), with return of spontaneous circulation (ROSC) achieved after 2 minutes of cardiopulmonary resuscitation (CPR). The recurrent arrhythmias prompted initiation of metoprolol and amiodarone.

Given the clinical concern for myocardial rupture versus aortic dissection, ECG-gated cardiac and chest computed tomography (CT) angiography was performed. Imaging demonstrated a large hematoma with associated bulging of the lateral left ventricular wall, extending posteriorly and causing displacement of the circumflex coronary branches, left inferior pulmonary vein, and left atrium. There was mild compression of the right ventricle, without evidence of cardiac tamponade. The coronary arteries were patent, with no evidence of dissection or active contrast extravasation. Additional findings included a small pericardial effusion, cardiomegaly, small bilateral pleural effusions with adjacent atelectatic change, mediastinal lymphadenopathy, and an incidental left adrenal lesion with imaging features most consistent with a benign adenoma (Figure 3).

Cardiac MRI was subsequently performed and demonstrated an unusual pattern of thickening of the lateral and anterior left ventricular walls with internal hemorrhagic change, particularly in the mid-to-epicardial region of the mid lateral wall. There was focal akinesia with compromise of the left ventricular cavity, heterogeneous myocardial enhancement, and T2 hyperintensity consistent with edema throughout the thickened myocardium. No cardiac rupture was identified. LV systolic function was moderately to severely impaired whilst right ventricle (RV) function was moderately reduced. These appearances raised concern for an infiltrative process with superimposed hemorrhages (Figure 4).

Positron emission tomography-computed tomography (PET-CT) demonstrated findings highly suspicious for a locally invasive primary cardiac malignancy with widespread metastases involving nodal, osseous, gallbladder, pancreatic, and bilateral adrenal sites, as well as possible involvement of the prostate/left seminal vesicle and soft tissue sites (Figure 5).

Based on the imaging findings, the patient was referred to the hematology/oncology team for further evaluation. An ultrasound-guided biopsy of the right supraclavicular lymph node was performed, and histopathological analysis demonstrated immunohistochemical positivity for MYC and BCL6, with a high mitotic count (Figure 6). MYC rearrangement was detected by fluorescence in situ hybridization (FISH), but no definitive BCL2 or BCL6 rearrangement. Histological features were consistent with a high-grade B cell lymphoma, rather than Burkitt's lymphoma. He was initially commenced on corticosteroids and pre-phase rituximab to alleviate symptoms and prevent progression of the lymphoma. A week later, chemotherapy was initiated with two cycles of R-CHOP and was subsequently escalated to 4 further cycles of DA-R-EPOCH.

PET-CT was performed after 2nd and 6th cycles of chemotherapy, which demonstrated impressive metabolic response. End of treatment PET-CT after cycle 6 was Deauville 4 due to persistent mild avidity at the left adrenal nodule that was deemed to be a separate indolent etiology. However, a repeat PET-CT three months later showed complete morphologic and metabolic response, with Deauville 2 (Figure 7).

Cardiac MRI performed approximately seven months after his initial ED presentation demonstrated preserved biventricular systolic function with normal ventricular volumes. There was interval improvement compared with the prior study, with Left Ventricular Ejection Fraction (LVEF) increasing from 30% to 52%. There was also a significant reduction in the infiltrative left ventricular myocardial mass, with only minimal residual myocardial thickening. His ECG has normalized compared to presentation (Figure 8). The patient subsequently returned to primary care follow-up, with an ongoing plan for regular surveillance.

2. Discussion

This case illustrates the diagnostic challenge posed by cardiac involvement of DLBCL, presenting as an acute coronary

syndrome (ACS) with ECG changes suggestive of posterior STEMI. Although ACS remains the most common cause of acute chest pain accompanied by ST-segment deviation and elevated cardiac biomarkers, clinicians should remain alert to alternative diagnoses when coronary angiography demonstrates non-obstructive coronary arteries.

Cardiac involvement by lymphoma is uncommon and may occur as either primary cardiac lymphoma or, more frequently, secondary involvement in disseminated systemic disease. Primary cardiac lymphoma accounts for fewer than 2% of primary cardiac tumours and less than 1% of extranodal lymphomas. Secondary cardiac infiltration is considerably more common, with autopsy studies demonstrating myocardial or pericardial involvement in approximately 10–25% of patients with advanced high-grade lymphoma, although antemortem diagnosis remains rare because of its highly variable clinical presentation (5, 8).

The presenting features in our patient, including crushing chest pain, diaphoresis, syncope, posterior ST-segment elevation, atrial fibrillation and markedly elevated troponin, appropriately prompted activation of the STEMI pathway. Immediate reperfusion therapy remains the standard of care in such presentations, and the initial management was entirely appropriate. However, coronary angiography demonstrated angiographically normal coronary arteries, shifting the diagnostic focus towards MINOCA and other STEMI mimics. In patients presenting with MINOCA, clinicians should systematically consider coronary embolism, myocarditis, Takotsubo syndrome, spontaneous coronary artery dissection, infiltrative cardiomyopathies, and cardiac tumours as potential underlying causes (2).

Several clinical features subsequently emerged that suggested an alternative pathological process rather than conventional myocardial infarction. The presence of newly diagnosed atrial fibrillation, recurrent ventricular arrhythmias culminating in ventricular tachycardia arrest, unexplained myocardial wall thickening on echocardiography, and the absence of obstructive coronary disease collectively indicated diffuse myocardial pathology. Cardiac involvement by lymphoma is recognised to produce electrical instability through direct infiltration of the myocardium and specialised conduction system, predisposing patients to atrial and ventricular arrhythmias (5, 6). These manifestations may precede the diagnosis of lymphoma and should heighten suspicion for infiltrative cardiac disease when occurring in conjunction with non-obstructive coronary arteries.

A major strength of this case lies in the sequential use of multimodality imaging. Bedside echocardiography identified an abnormality adjacent to the posterolateral left ventricular wall but lacked sufficient tissue characterisation to establish the diagnosis. Cardiac CT excluded aortic dissection and myocardial rupture, while demonstrating a mass-like infiltrative process causing displacement of surrounding cardiac structures.

Cardiac MRI, however, proved pivotal. The combination of myocardial thickening, heterogeneous late gadolinium enhancement, extensive myocardial edema and hemorrhagic change strongly favoured an infiltrative malignant process over myocardial infarction or intramyocardial hematoma. CMR remains the imaging modality of choice for evaluating cardiac masses because of its superior spatial resolution and tissue characterisation, allowing differentiation between inflammatory, infiltrative and neoplastic myocardial diseases (9).

PET-CT further demonstrated extensive metabolically active disease, identifying accessible supraclavicular lymphadenopathy for biopsy. Histopathological confirmation of high-grade DLBCL enabled timely commencement of immunochemotherapy. Importantly, the patient's dramatic radiological response and subsequent recovery of left ventricular systolic function following treatment strongly support myocardial infiltration by lymphoma as the underlying mechanism of presentation.

The mechanism responsible for the patient's myocardial injury was likely multifactorial. In contrast to classical Type 1 myocardial infarction caused by acute plaque rupture and coronary thrombosis, myocardial ischemia in this case appears to have resulted predominantly from external compression and displacement of the circumflex coronary artery by infiltrative myocardial tumour, together with direct myocardial infiltration and associated edema. These mechanisms produced ECG abnormalities and myocardial injury despite angiographically patent coronary arteries, emphasising that malignant cardiac infiltration should remain within the differential diagnosis of STEMI mimics (3).

This case also highlights the importance of multidisciplinary collaboration. Emergency physicians, interventional cardiologists, cardiac imaging specialists, pathologists and hematologists each played an essential role in establishing the diagnosis and facilitating prompt treatment. Early recognition of atypical features following normal coronary angiography allowed rapid progression to advanced imaging and tissue diagnosis, ultimately leading to complete metabolic remission and significant recovery of ventricular function.

To our knowledge, reports describing DLBCL presenting as posterior STEMI with ventricular tachycardia arrest and complete functional myocardial recovery following chemotherapy remain exceptionally uncommon. This case therefore contributes valuable evidence regarding the varied cardiovascular manifestations of lymphoma and reinforces the importance of maintaining a broad differential diagnosis in patients presenting with apparent acute coronary syndrome but unobstructed coronary arteries.

3. Conclusion

Cardiac diffuse large B-cell lymphoma is an uncommon but important cause of acute coronary syndrome mimics. This case demonstrates that myocardial infiltration and external coronary artery compression may produce electrocardiographic changes, biomarker elevation and clinical features indistinguishable from acute myocardial infarction despite the absence of obstructive coronary artery disease. In patients presenting with MINOCA, particularly when accompanied by unexplained arrhythmias or abnormal myocardial thickening, clinicians should maintain a high index of suspicion for infiltrative cardiac malignancy.

Early use of multimodality imaging, especially cardiac MRI, combined with prompt histopathological confirmation can facilitate timely diagnosis and initiation of disease-specific therapy. Successful treatment of the underlying lymphoma may result not only in complete oncological remission but also substantial recovery of myocardial function.

4. Declarations

4.1. *Acknowledgments*

The authors thank the Emergency Department, Cardiology, Cardiac Imaging, Radiology, Pathology and Hematology teams at Gold Coast Hospital and Health Service for their contribution to the diagnosis and management of this patient.

4.2. *Conflict of interests*

The authors declare that they have no competing interests.

4.3. *Funding and supports*

The authors received no specific funding for the preparation of this manuscript.

4.4. *Authors' contributions*

All authors fulfilled the four ICMJE authorship criteria, including substantial contributions to the conception or design of the work or the acquisition, analysis, or interpretation of data; drafting the work or revising it critically for important intellectual content; final approval of the version to be published; and agreement to be accountable for all aspects of the work.

4.5. *Ethical statement*

Ethical approval was not required for publication of this single case report in accordance with institutional policy. Written informed consent was obtained from the patient.

4.6. *Informed consent*

Written informed consent for publication of this case report and the accompanying clinical images was obtained from the patient.

4.7. *Availability of supporting data*

All data relevant to this case are included within the article. Additional information is available from the corresponding author upon reasonable request.

4.8. *Using artificial intelligence chatbots statement*

ChatGPT was used only to identify the most up-to-date relevant references.

5. References

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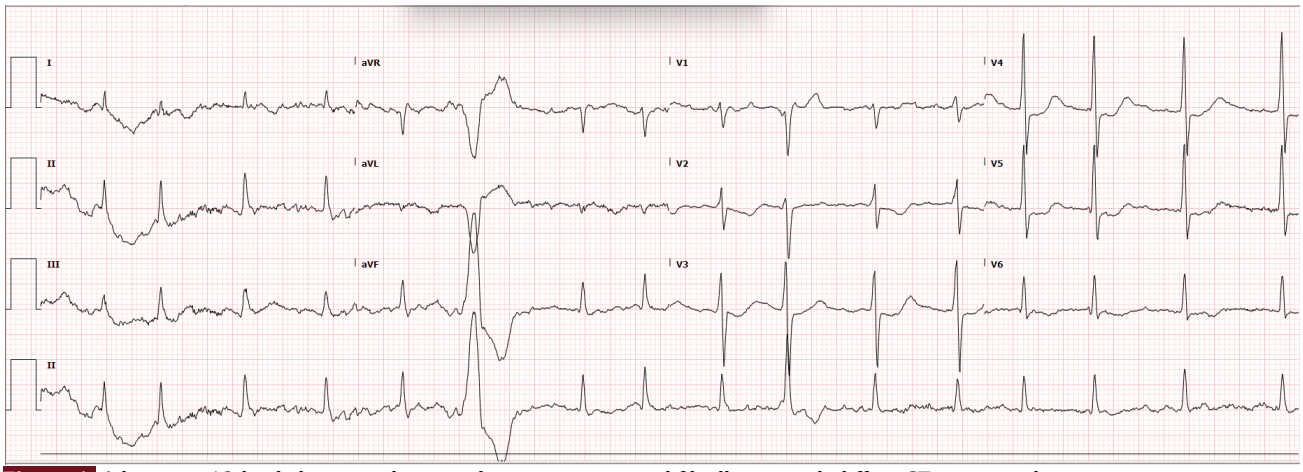

Figure 1: Admission 12-lead electrocardiogram demonstrating atrial fibrillation with diffuse ST-segment depression, most prominent in leads V4–V5, consistent with widespread subendocardial ischemia.

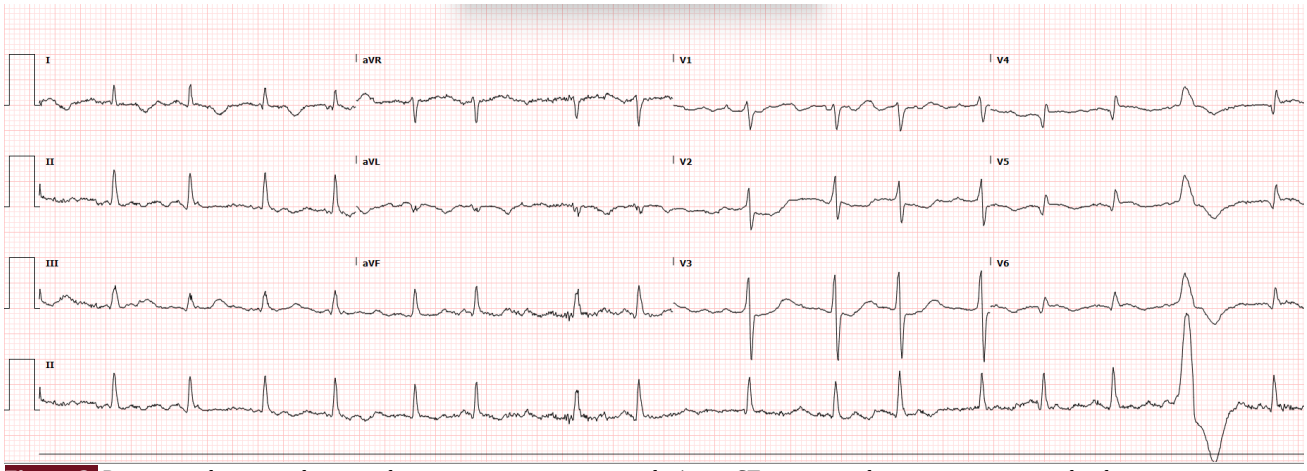

Figure 2: Posterior electrocardiogram demonstrating approximately 1 mm ST-segment elevation in posterior leads, prompting activation of the STEMI pathway. STEMI: ST-segment elevation myocardial infarction.

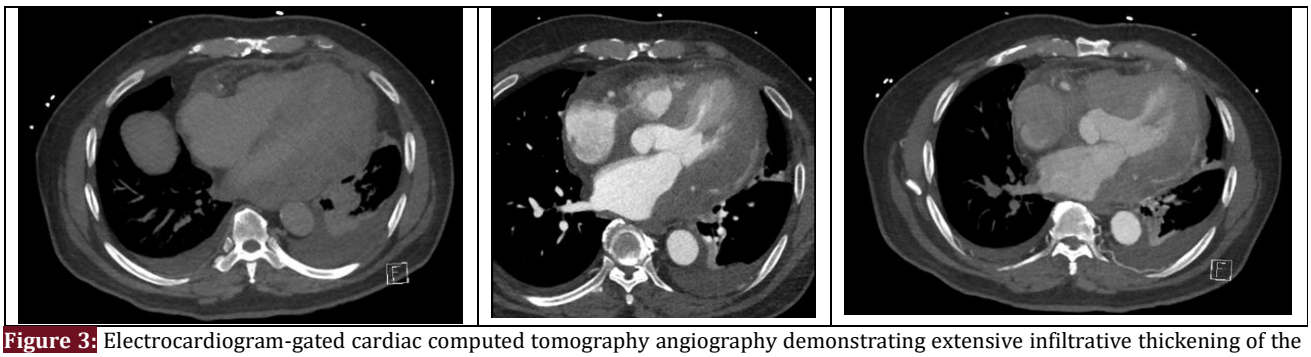

Figure 3: Electrocardiogram-gated cardiac computed tomography angiography demonstrating extensive infiltrative thickening of the lateral left ventricular wall with associated hemorrhagic change causing displacement of the circumflex coronary artery and adjacent cardiac structures. No coronary dissection or active contrast extravasation was identified (Left: non-contrast; middle: arterial/coronary phase; right: delayed phase).

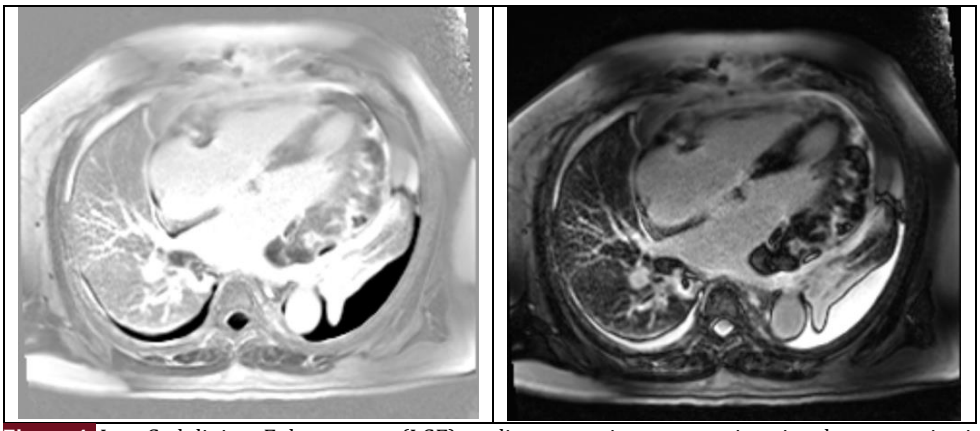

Figure 4: Late Gadolinium Enhancement (LGE) cardiac magnetic resonance imaging demonstrating infiltrative thickening of the lateral and anterior left ventricular myocardium with heterogeneous late gadolinium enhancement, myocardial edema and intramyocardial hemorrhagic change, highly suggestive of infiltrative malignancy (Left: phase-sensitive inversion recovery; right: magnitude reconstruction).

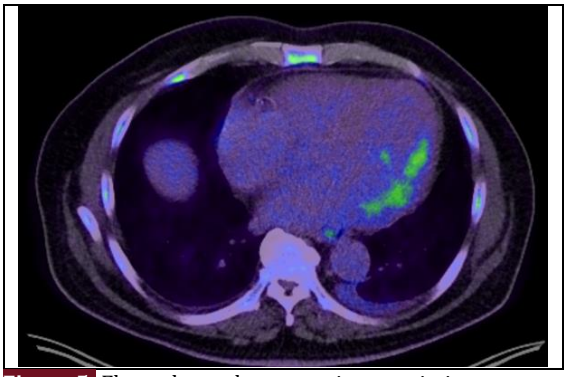

Figure 5: Fluorodeoxyglucose positron emission tomography-computed tomography demonstrating intense metabolic activity within the cardiac lesion together with widespread nodal and extranodal disease, consistent with disseminated lymphoma.

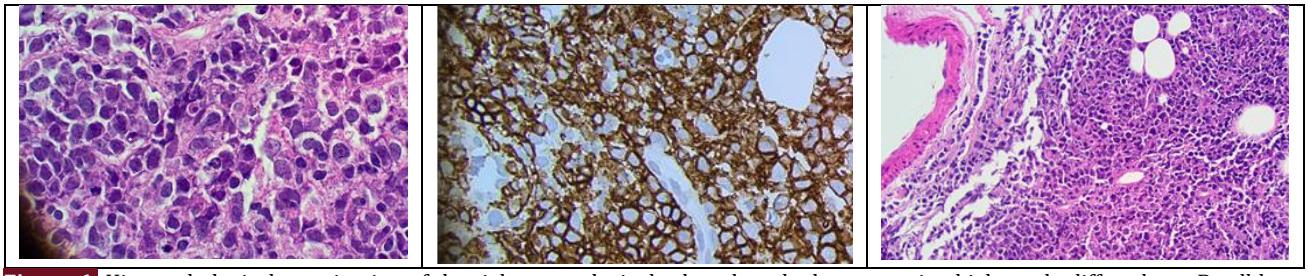

Figure 6: Histopathological examination of the right supraclavicular lymph node demonstrating high-grade diffuse large B-cell lymphoma with MYC and BCL6 expression. Fluorescence in situ hybridisation confirmed MYC rearrangement.

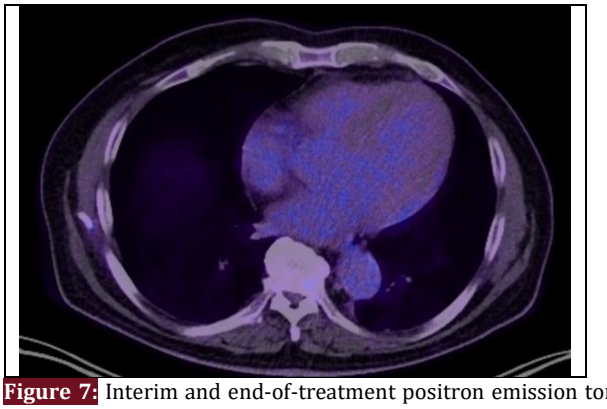

Figure 7: Interim and end-of-treatment positron emission tomography-computed tomography demonstrating marked metabolic response following immunochemotherapy and complete metabolic remission on surveillance imaging (Deauville score 2).

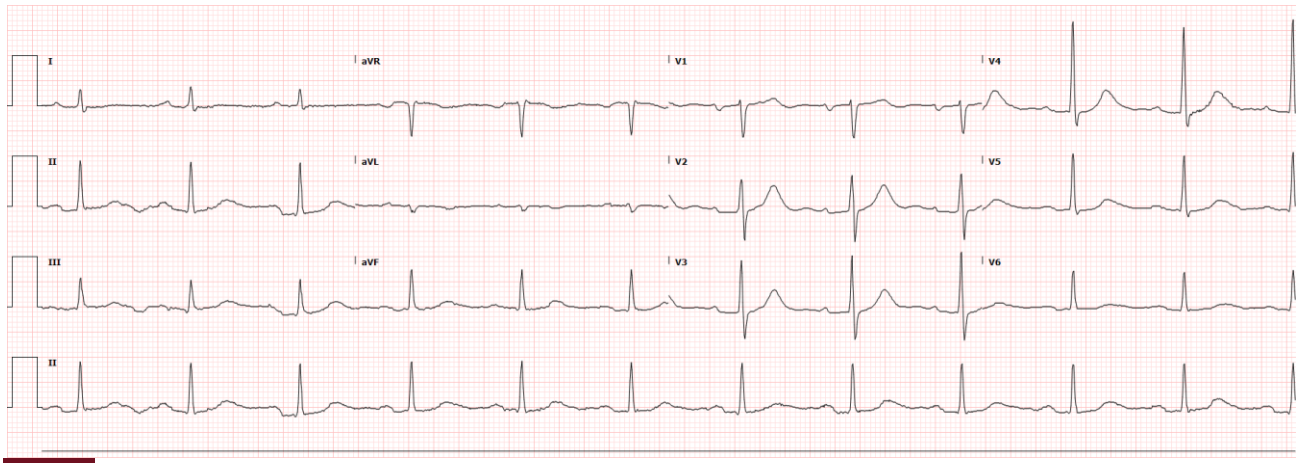

Figure 8: Follow-up electrocardiogram following completion of chemotherapy demonstrating resolution of the initial ischemic changes.