Case Report

Acute Coronary Syndrome with Non-ST-Elevation Myocardial Infarction and Refractory Unstable Ventricular Tachycardia Complicated by Severe Acute Kidney and Liver Injury in Myxedema Crisis: A Case Report

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Highlights

- This case suggests that myxedema crisis often exist with AMI.
- Implementing a diagnostic scoring system to improve the recognition of myxedema crisis as a comorbidity in severe myocardial infarction is expected to reduce treatment delays, thereby preventing refractory shock and arrhythmias and minimizing the need for invasive interventions.

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ABSTRACT

Background: Myxedema crisis, which occurs due to hypothyroidism, is a rare and life-threatening condition that can lead to severe myocardial infarction and lethal arrhythmia, as presented in this case.

Case Presentation: A 63-year-old man presented with typical prolonged chest pain, palpitations leading to near syncope, severe fatigue, loss of appetite, dizziness, and somnolence 2 days before admission. The patient exhibited somnolence, hypotension, thin eyebrows, and pretibial pitting edema. Electrocardiography revealed sinus rhythm with a prolonged QT interval, inferolateral-anterior ischemia, and a troponin-T value five times above the upper limit of normal. Therefore, the working diagnosis included non–ST-elevation myocardial infarction Killip IV, severe biventricular heart failure, severe acute kidney injury, and severe acute liver injury. On the third day of treatment, the patient experienced two consecutive episodes of unstable ventricular tachycardia and one episode of return of spontaneous circulation cardiac arrest. Thyroid examination incidentally revealed severe hypothyroidism with severe hyperkalemia. After other causes were excluded, the diagnosis of myxedema crisis was assumed. Oral thyroid therapy, levothyroxine (100 µg once daily), was administered. Within 3 days of initiating all treatments, the patient experienced significant hemodynamic improvement, improved kidney function, and normalization of liver function, accompanied by the disappearance of dyspnea, chest pain, and edema, with a compos mentis status. The patient was discharged with stable hemodynamics without support on the tenth day of treatment and underwent a coronary computed tomography angiography at an outpatient facility, which showed near-normal coronary results. The patient has been on routine follow-up for almost 1 year with levothyroxine (50 µg once daily) and has recently demonstrated good left ventricular function (ejection fraction=50%) and good functional capacity on an exercise test.

Conclusion: Clinicians should consider hypothyroidism crisis in the differential diagnosis for myocardial infarction, heart failure, and lethal arrhythmia, and treatment should be initiated immediately.

Keywords: Hypothyroidism; Case Report; Myocardial Infarction; Levothyroxine; Myxedema

RHYTHM
Research in Heart Yield and Translational Medicine

Introduction

Myxoedema crisis (or coma) is a severe manifestation of hypothyroidism and represents a rare, life-threatening condition with an incidence of approximately 0.22 per million annually.[1] It results from severe and prolonged depletion of thyroid hormone.[2] This condition can lead to multi-organ dysfunction, including cardiovascular collapse, renal failure, and altered mental status,[3,4] and is often precipitated by stressors such as infection or myocardial infarction (MI).[5]

This case highlights the interplay between hypothyroidism and acute coronary syndrome (ACS), with unresolved coronary vasospasm or metabolic dysfunction considered potential mechanisms. Here, we describe a case of MI complicated by refractory unstable ventricular tachycardia and cardiogenic shock, which improved following appropriate management of the myxoedema crisis.

Case Presentation

A 63-year-old male presented with a two-day history of chest pain, palpitations, near-syncope, fatigue, anorexia, and somnolence. He had no prior hospitalizations, past medical history, or known comorbidities.

Physical examination revealed hypotension (blood pressure 100/70 mm Hg), hypothermia (35 °C), and tachypnea with a respiratory rate of 30 breaths per minute. Additional findings included periorbital edema, thinning of the eyebrows, bilateral pitting edema, and cool extremities. Cardiac and pulmonary examination showed a grade 3/6 pansystolic murmur at the lower left sternal border with a gallop rhythm, as well as basal rhonchi.

Initial investigations demonstrated an abnormal electrocardiogram (ECG) showing ST elevation in leads aVR and aVL, biphasic T-waves in V_3–V_4, inverted T-waves in V_5–V_6 and II/III/aVF, and a prolonged QT interval (Figure 1). Laboratory results showed an elevated thyroid-stimulating hormone (TSH) level of 55 mU/L, markedly reduced free thyroxine (0.001 mmol/L), elevated troponin-T (259; normal <50), elevated creatinine (6.9 mg/dL) with an estimated glomerular filtration rate (eGFR) of 8 mL/min/1.73 m², and hyperkalemia (K^+ 6.5 mEq/L).

Chest radiograph demonstrated cardiomegaly with a cardiothoracic ratio of 66% and evidence of pulmonary edema (Figure 4). Based on these findings, the patient was diagnosed with non–ST-elevation myocardial infarction (NSTEMI, Killip class IV) complicated by cardiogenic shock, myxoedema crisis (diagnostic score: 135), severe acute kidney injury, urinary tract infection, and hyperkalemia.

During the first 3 days of management, the patient received comprehensive supportive care. Cardiac support consisted of norepinephrine and dobutamine, along with dual antiplatelet therapy and heparin. Renal management included furosemide and the glucose–insulin–calcium protocol for hyperkalemia. Ceftriaxone was initiated for a suspected urinary tract infection.

On the second day of ICU treatment, despite stabilization efforts and ACS management, the patient remained apathetic, with fluctuating blood pressure and evolving ECG changes in rhythm and ST segments. Echocardiography demonstrated severe biventricular heart failure, global hypokinesis, mild pericardial effusion, and adequate intravascular volume, although the patient continued to have low baseline cardiac output (Figure 5).

On the third day, recurrent episodes of ventricular tachycardia (Figure 2) were managed with two cardioversion attempts, resulting in return of spontaneous circulation after cardiac arrest. Because of recurrent ventricular tachycardia in the setting of ACS and refractory shock, emergency percutaneous coronary intervention was recommended; nonetheless, the patient declined the procedure.

Given the continued deterioration in MI status, a reassessment was performed. The clinical presentation and ECG findings were consistent with hypothyroidism, prompting evaluation of thyroid function. Laboratory testing revealed markedly elevated TSH and low free T4 levels. In the presence of severe systemic abnormalities, the diagnosis of NSTEMI complicated by myxedema crisis was established.

Subsequent diagnostic scoring yielded a total of 135 points (scores >60 indicate myxedema crisis), confirming the diagnosis and indicating a

poor prognosis.

Immediate thyroid hormone replacement was initiated; nevertheless, intravenous levothyroxine was unavailable. A literature review indicated that oral levothyroxine can be effective in treating myxedema crisis. After the confirmation of adequate intravascular volume, the patient received oral levothyroxine (250 µg), along with low-dose corticosteroid therapy and ongoing supportive treatments. By the afternoon of the fourth day, the patient's clinical status had begun to improve, and hemodynamics stabilized with minimal support. From days 4 to 10, management focused on maintaining hemodynamic stability, and vasopressors were successfully discontinued by day 5. By the sixth day, the patient maintained hemodynamic stability without pharmacologic support. Laboratory results obtained on days 6 and 9 demonstrated normalization of renal function and other metabolic parameters. The eGFR improved markedly from 8 to 99 mL/min/1.73 m². The patient reported relief with the timely therapy, which helped avoid progression to respiratory failure requiring mechanical ventilation.

Before discharge, a follow-up ECG showed normalization of the QTc interval and resolution of ST-segment depression in the lateral and inferior leads (Figure 3). At discharge on day 14, the patient was clinically stable and was prescribed a maintenance dose of levothyroxine (50 µg daily). Outpatient laboratory results obtained 1 month later showed normalization of thyroid hormone levels. Coronary computed tomography demonstrated near-normal coronary anatomy, with only mild stenosis in the left anterior descending and left circumflex arteries (Figure 6). At the 6-month follow-up, the patient's left ventricular ejection fraction had improved to 50% (Timeline of case presentation in Table 1).

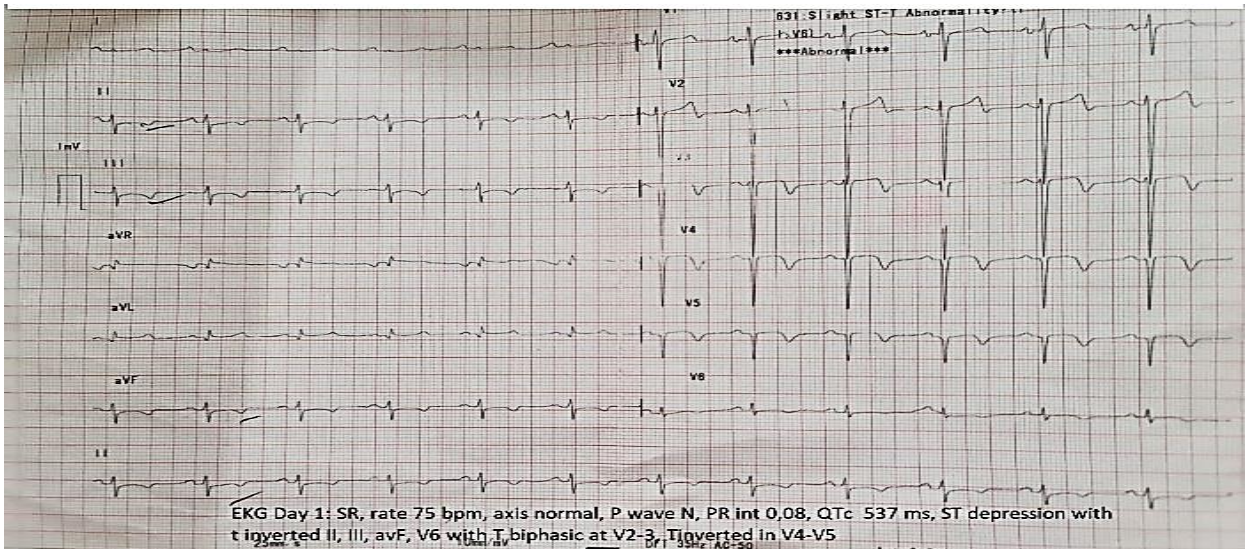

Figure 1. Day 1 ECG showing inferolateral ischemia and a prolonged QT interval

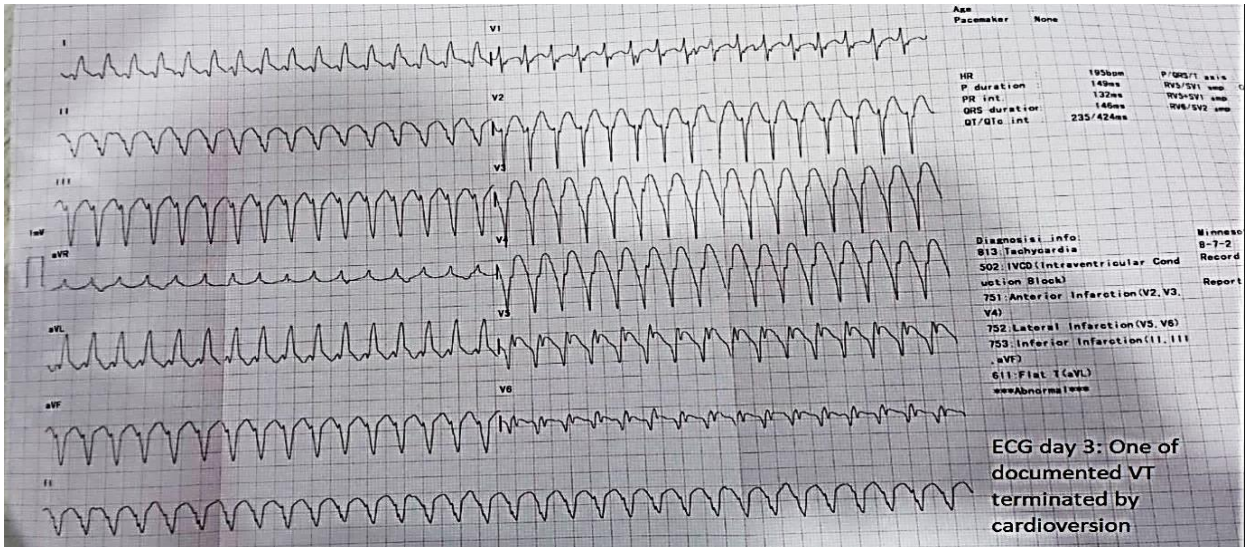

Figure 2. Day 3 ECG showing ventricular tachycardia

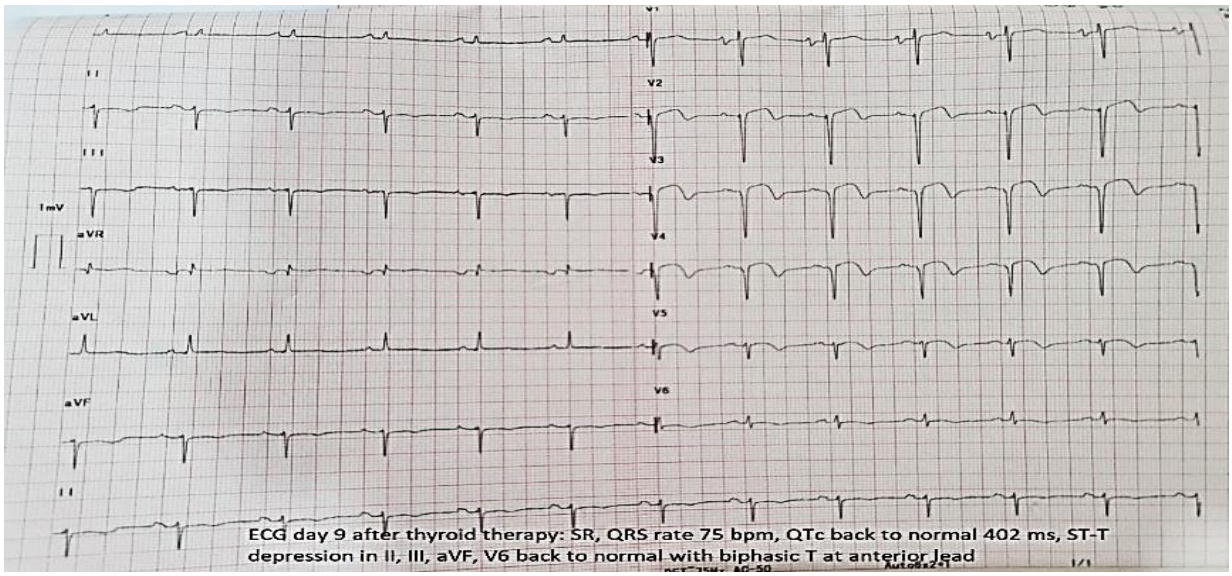

Figure 3. Day 9 ECG showing anterior ischemia with normalization of the QT interval

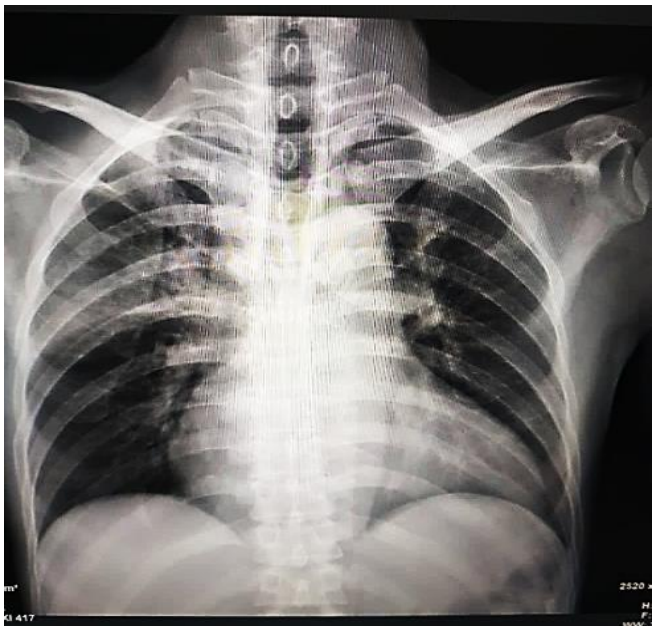

Figure 4. Chest radiograph (PA view) demonstrating cardiomegaly with signs of acute pulmonary edema

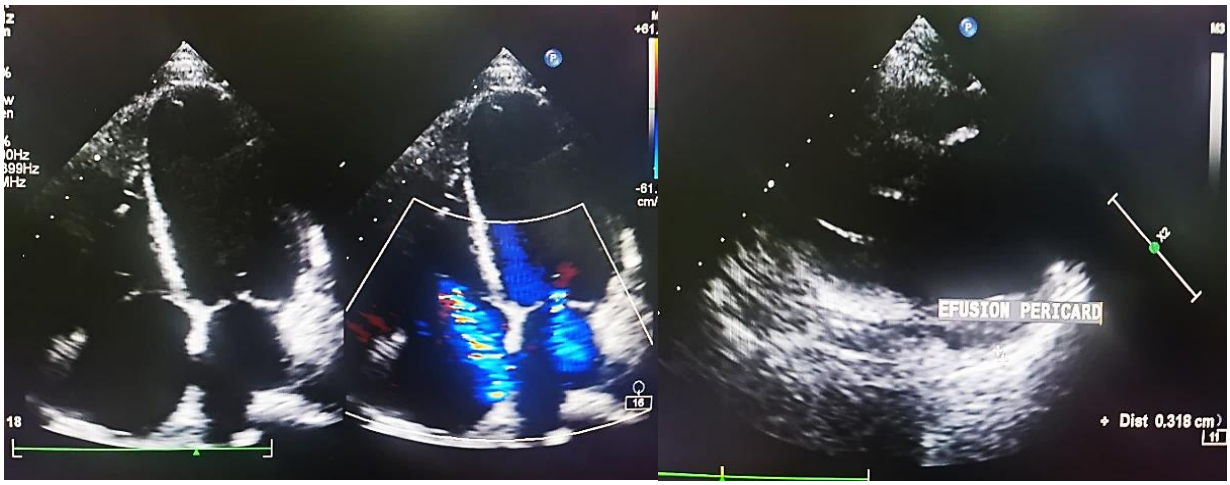

Figure 5. Echocardiography showing dilation of the left ventricle, right atrium, and right ventricle, with mild pericardial effusion. Moderate mitral and tricuspid regurgitation was also observed.

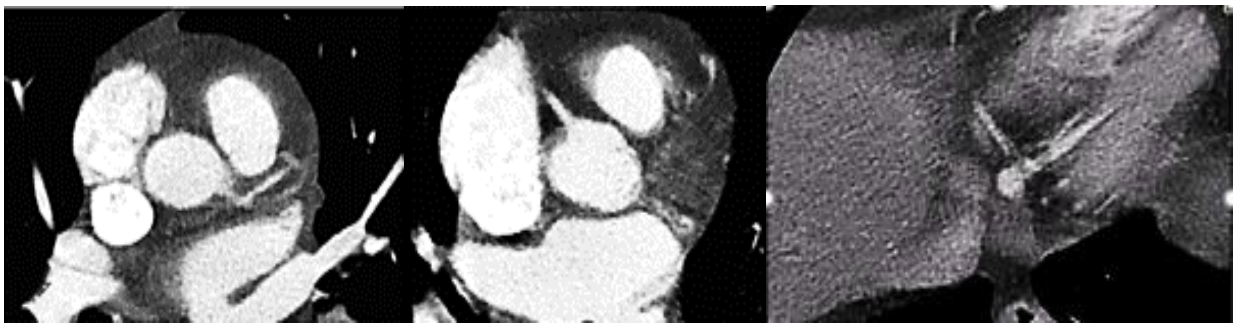

Figure 6. Coronary CT showing near-normal coronary anatomy, with only mild stenosis of the left anterior descending and left circumflex arteries

Table 1. Timeline and follow up of patient

Day	Clinical Events & Findings	Interventions & Outcomes
Day 0 (Pre-Admission)	2-day history of: • Chest pain • Palpitations, near-syncope • Fatigue, anorexia, somnolence	Stabilization with intravenous fluids in primary care
Day 1 (Admission)	Somnolence, hypotension (BP 100/70 mm Hg), hypothermia (35 °C) • ECG: Prolonged QT, inferolateral ischemia • Labs: ↑troponin-T (259), ↑TSH (55 mU/L), ↓FT4 (0.001 mmol/L), ↑creatinine (6.9 mg/dL), hyperkalemia (K^+ 6.5) • Chest radiograph: Cardiomegaly, pulmonary edema	Diagnosed with NSTEMI (Killip IV), cardiogenic shock, acute kidney injury, and UTI • Started dual antiplatelet therapy, norepinephrine, dobutamine, furosemide, and glucose–insulin–potassium protocol • Ceftriaxone for UTI
Day 2	Consciousness deteriorated (apathetic) • ECG rhythm changes, fluctuating BP • Echocardiography: Severe biventricular failure, global hypokinesia	Continued ICU supportive care and acute coronary syndrome management
Day 3	Refractory ventricular tachycardia (2 episodes), 1 cardiac arrest → ROSC via cardioversion • Refused PCI • Suspected hypothyroidism confirmed by TSH/FT4	Diagnosed with myxedema crisis (score:135); intravenous levothyroxine unavailable
Day 4	Oral levothyroxine (250µg) + low-dose corticosteroids administered	Marked hemodynamic improvement by the afternoon
Day 5	Weaned off vasopressors	Hemodynamic stabilization progressing
Day 6	Off all support • Laboratory parameters improving • Renal function improving (eGFR ↑ 8 → 99 mL/min/1.73 m^2)	Continued monitoring and supportive care
Day 9	ECG: Normal QT interval, resolved ST-segment depression	Kidney and liver function normalized
Day 10	Discharged with stable vital signs • Levothyroxine (50µg/day) initiated	Outpatient follow-up planned
1-Month Follow-up	No dyspnea or fatigue • Normal thyroid laboratory results • Coronary CT: Mild LAD/LCx stenosis	Left ventricular ejection fraction 50% • Good exercise tolerance

BP: blood pressure; ECG: electrocardiography; QT: QT interval; TSH: thyroid-stimulating hormone; FT4: free thyroxine; NSTEMI: non–ST-elevation myocardial infarction; Killip IV: Killip class IV; AKI: acute kidney injury; UTI: urinary tract infection; ICU: intensive care unit; PCI: percutaneous coronary intervention; ROSC: return of spontaneous circulation; LAD: left anterior descending artery; LCx: left circumflex artery; eGFR: estimated glomerular filtration rate; LV: left ventricle; EF: ejection fraction

Discussion

Several underlying mechanisms characterize the relationship between hypothyroidism and ACS.[6] A vicious cycle between MI and hypothyroid crises has been proposed, which persists until both conditions are thoroughly treated to break the cycle.[3,6,7] The relationship between hypothyroid crisis and acute MI is a critical area of clinical investigation. Hypothyroidism, particularly in its severe form (myxedema crisis), can exacerbate cardiovascular risk factors such as dyslipidemia, endothelial dysfunction, and impaired cardiac contractility, thereby increasing susceptibility to acute MI.[5–7] Conversely, acute MI may precipitate or worsen thyroid dysfunction due to systemic stress and inflammatory responses.[5,6] Further research is needed to elucidate the bidirectional pathophysiological mechanisms and optimize therapeutic strategies for patients with concurrent hypothyroid crisis and acute MI.

Notably, coronary vasospasm, driven by elevated catecholamine levels, and metabolic dysfunction due to reduced triiodothyronine/thyroxine (T3/T4) levels, which result in compromised myocardial contractility, are significant contributors.[8] A recent study reported that 25% of individuals with acute MI present with subclinical hypothyroidism.[8] Important considerations include the potential for

a myxedema crisis, which could simulate STEMI or NSTEMI even when the coronary arteries are unobstructed.[9] ECG indicators, such as bradycardia, T-wave inversions, and QTc prolongation, may appear before confirming hormone levels.[10] The use of diagnostic scoring is expected to reduce delays in therapy, which can prevent refractory shock, arrhythmias, and invasive treatment.[11,12] Immediate initiation of thyroid replacement therapy, whether administered orally or intravenously, is essential and can be life-saving.[13,14]

Conclusion

This case highlights the myxedema crisis as a reversible cause of refractory cardiogenic shock and acute kidney injury. Thyroid function tests should be routine in ACS with atypical features. Early levothyroxine and multidisciplinary care are critical for survival.

Declarations:

Ethical Approval

Informed consent was obtained from the patient's family, and they gave their written consent to use the patient's personal data for the publication of this case report and any accompanying images. Any identifiable information in images has been removed or masked.

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Conflict of Interest

The authors have no relevant financial or non-financial interests to disclose.

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