Case Report
Acute myocardial infarction associated with blood transfusion: Case report and literature review
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A B S T R A C T
A 62-year old patient with a history of chronic anemia associated with malabsorption secondary to short gut syndrome, experienced acute chest pain the second hour after the transfusion of a crossmatch-compatible erythrocyte suspension. His electrocardiogram (ECG) revealed widespread ST-segment depressions and he had an elevated troponin level. Laboratory findings and physical examination did not indicate the presence of immunological or non-immunological blood transfusion reactions. Cardiac catheterization was performed and showed angiographically non-obstructive, atherosclerotic plaques and the absence of vasospasm or thrombus formation. Following antiischemic therapy his symptoms resolved completely. The ECG obtained 24 hours after the emergence of chest pain demonstrated normal sinus rhythm with no ST-T wave changes. We present a rare case of acute myocardial infarction induced following a blood transfusion. To the best of our knowledge, a few cases of acute myocardial infarction associated with blood transfusion have been formally recorded in the medical literature and the clinical experience regarding such cases is indeed quite limited. The present case is reviewed in the context of the relevant literature as a practical resource for clinical practice.

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1. Introduction
Acute coronary syndrome (ACS) is relatively rare in comparison to other complications of blood transfusion and it is difficult to explain how blood transfusion could have precipitated the myocardial infarction [1]. The majority of the reported cases involved acute myocardial infarction associated with blood transfusions performed due to gastrointestinal bleeding [2,3]. ACS following blood transfusion for the treatment of anemia in the absence of bleeding is very rare.

2. Case report
A 62-year old male patient presented to the general surgery department with weakness, fatigue and decreased effort capacity. The patient had a history of chronic anemia associated with malabsorption secondary to short gut syndrome and had undergone periodic blood transfusions. His initial laboratory parameters were reported as follows: Hb 10 g/dL (13–17 g/dL), Hct 30% (42–54%), MCV 95 fL (80–100 fL), WBC 4.7 × 10⁹/L, PLT 258 × 10⁹/L (150–450 × 10⁹/L), urea 26 mg/dL (15–45 mg/dL), creatinine 1.07 mg/dL (0.66–1.25 mg/dL), K 4.9 mmol/L (3.5–5.1 mmol/L) AST 40 U/L (0–35 U/L), ALT 59 U/L (0–45 U/L), total bilirubin 0.6 mg/dL (0.3–1.2 mg/dL), direct bilirubin 0.2 mg/dL (0.00–0.2 mg/dL), lactate dehydrogenase 268 U/L (0–248 U/L), fibrinogen 470 mg/dL (175–400 mg/dL), erythrocyte sedimentation rate 40 mm/h, ferrum 75 µg/dL (49–167) and ferritin 643 µg/L (10–300 µg/L). There was no evidence of bleeding. As the serum hemoglobin level was low blood transfusion was considered. 1 unit (250 mL) crossmatch-compatible type A Rh+ donor erythrocyte suspension was transfused over a period of two hours in

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the general surgery department. The patient reported chest pain approximately two hours after the transfusion. The arterial blood pressure and heart rate were 120/75 mmHg and 56 beats per minute, respectively. The axillary body temperature was 36.7 °C (98.06 °F); no increase in body temperature was observed during follow up. A physical examination was normal. Electrocardiography (ECG) revealed a 0.1 millivolt diffuse ST segment depression in all derivations with the exception of aVR (Fig. 1). The chest X-ray was unremarkable (Fig. 2). Oxygen saturation was 95% as measured by a pulse oximeter. Transthoracic echocardiography (TTE) revealed mild global hypokinesis of the left ventricle with an ejection fraction of 45%. The patient’s blood sample on admission was re-evaluated for blood group and typing and blood incompatibility was ruled out. The indirect antiglobulin test (Coombs test) was negative using a post-transfusion blood sample. Laboratory parameters were reported as follows: Hb 10 g/dL, Hct 30.9%, WBC 7.1 x 10^9/L, PLT 252 x 10^9/L, urea 26 mg/dL, creatinine 0.82 mg/dL, K 4.4, AST 78 U/L, ALT 49 U/L, total bilirubin 1.3 mg/dL, direct bilirubin 0.6 mg/dL, lactate dehydrogenase 338 U/L, fibrinogen 530 mg/dL and haptoglobin 160 mg/dL (30–200 mg/dL). Laboratory findings and physical examination did not indicate the presence of immunological or non-immunological blood transfusion reactions. Serum biomarkers of myocardial injury were elevated; Troponin I was 5.5 ng/dL (0–0.04 ng/dL). The patient was taken to the catheterization laboratory for coronary angiography following loading dose of clopidogrel (600 mg) and acetylsalicylic acid (300 mg), resulting in a diagnosis of acute myocardial infarction. Coronary angiography revealed non-obstructive, atherosclerotic plaques and the absence of vasospasm or thrombus formation (Fig. 3). Following the antiischemic therapy (100 mg acetyl salicylic acid, 75 mg clopidogrel, 50 mg metoprolol succinate and 60 mg isosorbide mononitrate once a day) his symptoms resolved completely. ECG readings obtained 24 h after the first symptoms demonstrated normal sinus rhythm with no ST-T wave changes (Fig. 4). The patient's hospitalization was uneventful and he was discharged from the hospital after five days with resolved symptoms. TTE obtained at two weeks post-discharge revealed normal LV size with recovery of systolic function (EF 58%).

3. Discussion

Acute coronary syndrome (ACS) is relatively rare in comparison to other complications of blood transfusion such as immune or non-immune hemolytic reactions. Only a few cases of acute myocardial infarction following a blood transfusion have been reported and therefore the clinical experience with such cases is limited [1–3]. The majority of the reported cases involved ACS associated with blood transfusions performed due to bleeding resulting from peptic ulcer or gastrointestinal malignancy and in most of these cases disseminated intravascular coagulation developed after the transfusion [2,3]. ACS following blood transfusion for the treatment of anemia in the absence of bleeding is very rare. The mechanism linking acute coronary syndrome to blood transfusion is poorly understood. However, potential mechanisms may include formation of rouleaux in the donor red blood cells blocking narrow coronary vessels, the rupture of atherosclerotic plaque present...
in the coronary artery after blood transfusion, post-transfusional disseminated intravascular coagulation induced intramyocardial coronary thrombosis, or the formation of coronary thrombus triggered by rapid increases in hematocrit and viscosity [3–7]. The present case exhibited no signs of critical lesion or thrombus formation in the epicardial coronary arteries. Therefore, we propose that microvascular coronary obstruction caused ACS in this patient. The basic features of management strategy in blood transfusion induced acute myocardial infarction should be no different from the management of classical acute myocardial infarction. However, to reduce the risk of excessive blood loss, antiplatelet and anticoagulant therapy should be carefully monitored in patients who develop ACS following a blood transfusion to treat bleeding.

A multidisciplinary approach is required, involving the cardiologist, hematologist, gastroenterologist and anesthesiologist, to determine the patient’s risk and devise an optimal treatment strategy in such cases. Emergency coronary angiography may be applicable in cases demonstrating hemodynamic instability, acute congestive heart failure, the development of life-threatening arrhythmia, or ST segment elevation. In cases where both increased troponin levels and ST-T segment changes are present, coronary angiography should be performed within 24 h [8].

In conclusion, myocardial infarction following blood transfusion is a very rare complication and may be life threatening. Therefore, the use of ECG and cardiac enzyme testing should be considered during follow up for the early detection of cardiac complication in patients who develop chest pain or shortness of breath following blood transfusion.

References
