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Case report

Hemolytic anemia after percutaneous coronary intervention (PCI): A case report

Yu Zhang [a], An Fuxiang [a], Meizhu Yan [a,b], Yi Zhou [a], Hongjun Bian [a,*]

[a] Shandong Provincial Hospital Affiliated to Shandong First Medical University, Jinan, Shandong, 250021, PR China
[b] Jinqiu Hospital of Liaoning Province, Shenyang, Liaoning, 110016, PR China

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ABSTRACT

Background: In clinical practice, intravascular hemolysis is not common after interventional cardiovascular procedures. Although diagnostic and treatment techniques have developed, with the increasing importance placed on people's own health and the popularity of cardiovascular intervention, there have been occasional reports of hemolysis after different cardiovascular interventions, mainly including cardiac pacemaker implantation, atrial-fibrillation radiofrequency ablation, transcatheter aortic-valve implantation (TAVI), transcatheter mitral valve replacement (TMVR) and percutaneous repair of Gerbode defect and percutaneous coronary intervention (PCI) with Impella. However, so far, there have been no relevant reports on postoperative hemolysis after percutaneous coronary intervention (PCI).

Case report: This article reports a very rare case of a 42-year-old male who developed hemolysis after PCI. The patient had dark brown urine for two days. Blood test showed significant decreases in red blood cell (RBC) and hemoglobin (Hb). After blood transfusion of 2 units, dexamethasone treatment and repeat PCI, he gradually recovered with no symptoms of further episodes of hemolysis.

Conclusions: Due to the use of antiplatelet and anticoagulation drugs in PCI patients, gastrointestinal bleeding (GIB) is often believed to be the main cause of postoperative bleeding events. Identifying the etiology of anemia in patients after PCI is crucial for targeted treatment in the later stage. Based on the symptoms of dark brown urine and the levels of RBC, HB, reticulocyte and unconjugated bilirubin (UCB), we finally diagnosed the patient with hemolytic anemia (HA), rather than the traditional consciousness of GIB. This is an uncommon case of hemolysis after PCI. Although the association between PCI and HA is very rare, PCI is now a commonly used treatment for patients with acute coronary syndromes (ACS). Therefore, clinicians should recognize that in addition to GBI, HA may also occur after PCI. Early recognition of the cause of anemia and early treatment is one of the key steps to ensure the later life and health of PCI patients.

1. Introduction

In recent years, with the rapid advancement in the number, difficulty, and techniques of interventional cardiovascular procedures, such as cardiac pacemaker implantatio [1], atrial-fibrillation radiofrequency ablation [2], transcatheter aortic-valve implantation

* Corresponding author. Shandong Provincial Hospital Affiliated to Shandong First Medical University, 324 Jingwu Road, Jinan, Shandong, 250021, PR China.
E-mail address: bhj0227@126.com (H. Bian).

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Table 1
Some cases of hemolytic anemia complications after interventional cardiovascular procedures.

Reference	Year	Patients	Characteristics	Severity	Treatment	Outcome
Ref [2]	2023	A 75-year-old male	Recurrence of symptomatic and persistent AF, pulmonary vein isolation, a PFA procedure was performed to lead to a restoration of sinus rhythm	AKI, anemia and hemolysis	Maintain preserve diuresis	The creatinine level returned to baseline over 2 months
Ref [2]	2023	A 74-year-old male	A history of heart failure with preserved ejection fraction and persistent AF, PFA procedure was performed to lead to a restoration of sinus rhythm	Biological renal failure, hemolysis	Maintain preserve diuresis	The creatinine level returned to normal level over 20 days
Ref [3]	2020	An 84-year-old male	Severe AS and underwent an AAC combined with coronary artery bypass grafting	Require frequent blood transfusion	TAVI	No recurrence of hemolytic anaemia
Ref [4]	2019	Two octogenarian female	Severe MR related to a failed MVR using a complete semi-rigid ring (Edwards Physio 2 N°32 and N°26)	Acute renal failure with anuria associated with an acute mechanical hemolysis, require blood transfusion	Dialysis for a women, mplantate new Edwards Sapien 3 N°26 valve deeper for another women	No recurrence of hemolytic anaemia
Ref [5]	2016	A 69-year-old female	Acquire Gerbode defect after AVR, MVR and re-do AVR	Severe hemolytic anemia and AKI, require daily blood transfusion	Open surgical closure of the Gerbode, hemodialysis	No recurrence of hemolytic anaemia
Ref [7]	2019	An 82-year-old male	Impella-assisted LMS PCI in a patient with severe LV systolic dysfunction and AIHA	High bleeding risk(active hemolysis, thrombocytopenia, impaired renal function, use of steroids)	Protected PCI with Impella CP	No evidence of exacerbation of the patient's hemolysis

Notes: AF atrial fibrillation, PFA pulsed field ablation, AS aortic stenosis, AAC apico-aortic conduit, TAVI transcatheter aortic valve implantation, MR mitral regurgitation, MVR mitral valve replacement, AVR aortic valve replacement, AKI acute kidney injury, LMS left main stem, LV left ventricular, AIHA autoimmune hemolytic anemia.

(TAVI) [3], transcatheter mitral valve replacement (TMVR) [4], percutaneous repair of Gerbode defect [5] and percutaneous coronary intervention (PCI) with Impella [6,7], acute physical hemolysis has been reported after the above treatments (Table 1). This may be result from the increased fragility of red blood cells (RBC) (an underlying asymptomatic condition) following pulsed field ablation (PFA), mechanical damage of RBCs caused by the change in blood flow direction due to turbulence and shear stress by the stenosis, as well as the high rotatory shear forces of the impeller during the process of passing through the stent. Although there have been a lot of reported studies of complications, diagnosis and therapeutic algorithm related to PCI (Table 2), there have not yet been generally reported of cases of immune hemolysis after PCI [8,9].

For non-ST-segment elevation myocardial infarction (NSTEMI), a time-to-angiography depending on the risk stratification. Which based on pain characteristics, severity of pain, clinical findings, ECG changes, biochemical markers and risk scores like the Thrombolysis in Myocardial Infarction (TIMI) risk score, and the Global Registry of Acute Coronary Events (GRACE 2.0) risk to define the risk of each patient. Immediate invasive strategy (<2h) in very high-risk patients, routine early invasive strategy in high-risk patients (<24h), selective invasive strategy in intermediate/low-risk patients [10].

1.1. Case presentation

A 42-year-old male with a history of thoracalgia for 10-h, dysphoria and unconsciousness for 5-min was sent to the department of Emergency Medicine of Liaocheng People's Hospital Affiliated to Shandong First Medical University. The electrocardiogram (ECG) monitor showed that the patient had ventricular fibrillation with no spontaneous breathing and heartbeat. After once electric defibrillation and about 4 minutes of cardiopulmonary resuscitation (CPR), the patient's sinus rhythm was recovered. At the same time, the patient was given tracheal intubation and assisted breathing with a ventilator. His past medical history only include coronary atherosclerotic heart disease (CHD) for 2 years. At present, his high sensitive troponin I (hs-TnI) level was 0.031 mg/L (normal value 0.01–0.023 mg/L), Pro-B-type natriuretic peptide (Pro-BNP) level was 200.0 pg/mL (normal value $\leq$ 125pg/mL) and lactate dehydrogenase (LDH) level was 283 U/L (normal value $\leq$ 250 U/L). The level of international normalized ratio (INR), activated partial thromboplastin time (APTT), anti-thrombin and fibrinogen were normal. Haptoglobin was not tested in this patient, for the reason that the clinical demand is too low to perform the laboratory test in the hospital. The ECG showed a significant depression in the ST-segment. As the patient did not show any symptoms of gastrointestinal and urinary system infections, relevant laboratory tests did not complete to rule out infections. HIV infection, Covid-19, HBV and HCV were ruled out by laboratory tests.

On the first day after admission, with oral tracheal intubation and sedation, coronary angiography showed that the proximal segment of the left anterior descending artery (LAD) was occluded, the proximal segment of right coronary artery (RCA) had a stenosis of about 90 % and its middle segment was occluded. After accurate positioning, a 2.0 $\times$ 15 mm cutting balloon was used to dilate the proximal segment of the LAD at 10 atm (atm) pressure. Coronary angiography showed that the lesion was a heavy thrombotic burden lesion. At this time, 10 mg Eptifibatide was administered intracoronary. Then, an Exrosssal 3.5 $\times$ 36 mm stent was implanted in the

Table 2
Complications, diagnosis and therapeutic algorithm related to PCI.

Complications		Diagnosis	Therapeutic algorithm
Coronary complications	Dissection	Chest pain with associated ST segment change, if dissection results in abrupt vessel closure, haemodynamic collapse may be observed	Stop ablation to prevent further damage. Place or retain coronary wire in the true lumen. If significant dissection, appropriate stent placement.
	Perforation	Distal contrast injection to confirm	Immediate balloon tamponade. Haemodynamic support, fluid resuscitation, consider blood or autotransfusion. Emergency pericardiocentesis if tamponade evident. Placement of covered stent for large vessel perforation. Distal embolisation with fat, coil, thrombin or autologous clotted blood for guidewire exit perforation.
	No-reflow	Consider distal contrast injection with microcatheter, aspiration catheter or over-the-wire balloon to confirm diagnosis, followed by distal	Check activated clotting time and administer intracoronary nitrates. Delivery of intracoronary adenosine, verapamil or sodium nitroprusside. Blood pressure optimization.
	Air embolisation	Haemodynamic collapse	100 % oxygen for ventilating, inotropic support,adenosine or sodium nitroprusside to increase the capillary bed space, and wiring and aspiration of the embolised vessel to disperse and extract the air, respectively.
	Abrupt vessel closure	Distal contrast injection to confirm	Ensure coronary wire in side branch to reduce risk of occlusion and guide rewiring. Adequate proximal optimization. Consider use of hydrophilic coronary wire to rewire side branch.
	Equipment entrapment or loss	Distal contrast injection to confirm	Deployment with serial insertion and dilatation of small to larger balloons. Crushing stent against vessel wall with a balloon and second stent to cover the lesion. Retrieval with advancement and dilatation of balloon distal to stent, wire braiding or use of a snare.
	Hypotension	Confirm hypotension by ensureing guide catheter is not too deep on target vessel, not interacting with aortic valve or contains thrombus, and rule out hematoma (mainly for femoral access)	Rule out perforation. Further etiological assessment. Correct reversible causes quickly. Hemodynamic support and ancillary imaging.
Noncardiac complications	Allergies	Haemodynamic embarrassment	High-flow oxygen and anaesthetic support. Intravenous crystalloid fluid challenge, epinephrine, hydrocortisone, chlorphenamine.
	Vascular access complication	Anticoagulation review, emergency CT scan and invasive angiography if necessary	Large bore venous access, blood transfusion and haemodynamic support.
	Thromboembolic complications	Haemodynamic embarrassment	High-flow oxygen. Consider microvascular vasodilatation with adenosine, sodium nitroprusside or verapamil. Consider wiring and aspiration of embolised vessel.

Table 3
Pro-BNP and hs-TnI data.

Indicators	Pre-surgery	Immediate after surgery	Day 1	Day 2	Day 3	Day 4
hs-TnI(mg/L)	0.031	11	12	8.4	3.1	1.3
Pro-BNP(pg/mL)	200	–	866	–	–	–

Notes: hs-TnI high sensitive troponin I, Pro-BNP Pro-B-type natriuretic peptid

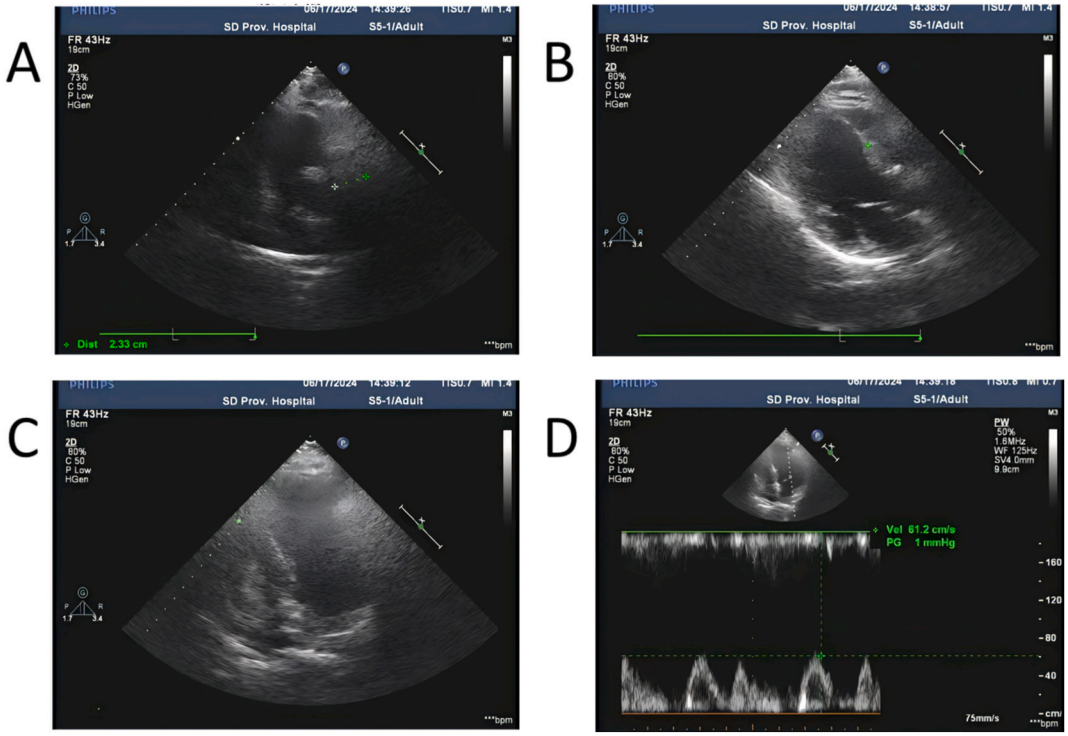

Fig. 1. The echocardiographic imaging data before the repeat PCI shows old myocardial infarction in left ventricular anterior wall. The diameter of main pulmonary artery is 2.33 cm (A). The long axis view of the left heart shows severe akinesia in middle and lower segments of the ventricular septum, the anterior wall of the left ventricle, and the ventricular wall of the apical segment. The thickness of them become thinner slightly and the echo of them enhance (B). The short axis view of the heart base (C). Color doppler flow imaging (CDFI) shows a small amount of regurgitation signal across mitral valve (D).

proximal segment of the LAD. Later, a 4.0 × 15mm hyperbaric balloon was delivered to the stent to perform post-dilated with 12atm. There was no residual stenosis, dissection or/and tear. TIMI III flow in the proximal segment of the LAD had been improved compared to the patient's previous baseline state. The procedure went smoothly without any signs of vascular entrapment, stent strut malapposition and tissue prolapse. Then, intra-aortic balloon pump (IABP) was used for two days as auxiliary device for LV unloading. The hs-Tnl value measured immediately after the intervention was 11.0 mg/L, and 100 mg of aspirin once a day and 90 mg of Ticagrelor twice a day were given by nasal feeding. Simultaneously, as the patient had recently received a stent treatment, he had to continue to take antiplatelet drugs and statins. On the 5th day after coronary angioplasty, the levels of hs-TnI and Pro-BNP decreased significantly (Table 3).

Based on the patient's medical history, symptoms, laboratory examination and other auxiliary examination results, the diagnosis is considered as [1] Coronary atherosclerotic heart disease [2]; NSTEMI.

On the 9th postoperative day, the patient began to get out of bed and move around. However, he lost consciousness again and fainted on the 11th day. Based on the patient's medical history, the cause of his re-collapse was first considered as Adams-Stokes syndrome or malignant arrhythmia. Fortunately, he regained consciousness soon. Under the current situation, immediately after he lost consciousness again, his red blood cell (RBC) level was 0.56×10^{12}/L (normal value 4.3–5.8 $\times 10^{12}$/L), Hemoglobin (Hb) level was 61.0 g/L (normal value 130–175 g/L), platelet (PLT) level was 654×10^{9}/L (normal value 125-350 $\times 10^{9}$/L). White blood cell (WBC) level was 32.24×10^{9}/L (normal value 3.5–9.5 $\times 10^{9}$/L). The patient was asked disease history in detail, and found that he had dark brown urine 2 days before. His ABO blood group is type B and Rh blood group is positive blood (B+). After received blood transfusion of 2 units of white depleted suspended red blood cells (O+), his RBC and Hb had improved compared to the basic state. Under the current situation, the reticulocyte level was 328.0 (normal value 23.0–70.1 $\times 10^{9}$/L), unconjugated bilirubin (UCB) level was 27.67

Table 4
Blood test data.

Indicators	Pre − surgery	Day1	Day2	Day3	Day4	Day5	Day9	Day11	Day12	Day13	Day14	Day15	Day20	Day23	Day28	Day39	Day67	Day88	Day109	4monthlater
RBC(10ˆ12/L)	5.03	4.63	4.72	4.72	4.83	4.86	3.44	0.56/1.82	2.38	2.89	2.43	2.21	2.05	2.62	3.32	4.08	4.71	5.15	4.91	4.85
Hb(g/L)	146	138	141	142	144	144	105	61/56	74	91	80	74	65	84	108	131	144	155	145	139
PLT(10ˆ9/L)	427	401	360	299	374	420	618	654/562	644	690	627	704	560	455	393	380	358	315	302	321
WBCs(10ˆ9/L)	20.47	25.11	19.22	19.32	14.45	11.11	25.18	32.24/26.09	30.94	35.95	26.10	21.79	10.95	10.67	11.60	16.52	12.22	14.83	10.07	8.42

Notes: RBC red blood cell, Hb hemoglobin, PLT platelet, WBC white blood cell, Day 1 1st after surgery, Day 109 two weeks after Dexamethasone drug discontinuance, 4 month later of the first PCI, before the repeat PCI.

(normal value 1.5–18μmol/L) and the indexes of anemia were higher than normal subjects. A peripheral blood smear be performed showing different volume of RBCs and abnormal RBC morphologies. His glucose hemolysis test, sucrose hemolysis test were positive and cluster of differentiation (CD) 55 and CD59, anti-nuclear antibody (ANA), serum free light chain, lupus anticoagulant (LAC), antiphospholipid antibody (APA) and systemic vasculitis antibody were negative, except β2-Glycoprotein 1-igG was 31.80 (normal value $\leq$ 20) and anti-glomerular basement membrane antibody (GBM) was 37.90 Human (normal value $\leq$ 20). The patient also received ham test and Coombs test. Unfortunately, due to the extensive destruction of RBCs, the number of collected RBCs after washing is extremely small, and it cannot be prepared into a qualified RBC suspension, so subsequent experiments cannot be conducted.

The patient was discharged after receiving 10 mg Dexamethasone via intravenous drip once a day for 5 days. One month later, his RBC and Hb has improved to 4.42 $\times$ 10^{12}/L (normal value 4.3–5.8 $\times$ 10^{12}/L) and 137.0 g/L (normal value 130–175 g/L) with oral administration dexamethasone of 3.75 mg. Dexamethasone was gradually reduced depending on the patient's condition. His RBC and Hb improved to normal level after two weeks of Dexamethasone drug discontinuance (Day 109 after surgery). Three months later, his 6-min walk test showed that there were no ST-T changes in the ECG, no arrhythmia before and after walking 482.46 m. The patient was scheduled to undergo a repeat PCI 2 weeks later. At that time, the transthoracic echocardiography showed old myocardial infarction in left ventricular anterior wall, and the left ventricular ejection fraction (LVEF) is 45 % (Fig. 1). Before the repeat PCI, his RBC and Hb has improved to 4.85 $\times$ 10^{12}/L (normal value 4.3–5.8 $\times$ 10^{12}/L) and 139 g/L (normal value 130–175 g/L) with no oral administration dexamethasone (Table 4). After repeat PCI, the patient had no symptoms of further episodes of hemolysis.

2. Discussion

The highlight of this case is the rare and life-threatening hemolytic anemia (HA) phenomenon after PCI. It is important for clinicians to be aware of this complication, as PCI is now a common treatment for patients with acute coronary syndromes (ACS) [11].

The common reason of anemia after PCI may be GIB. Because of the use of aspirin, dual antiplatelet therapy (DAPT) (clopidogrel, ticagrelor, or prasugrel), parenteral anticoagulation (unfractionated heparin, low-molecular-weight heparin, bivalirudin, and fondaparinux), proton pump inhibitors are recommended to prevent bleeding caused by the use of these antiplatelet and anticoagulation drugs in patients [12]. However, this case shows an rare cause of anemia after PCI, which is hemolytic anemia rather than common GIB.

Hemolysis is a process in which RBCs are destroyed and prematurely removed from the circulatory system. HA refers to the anemia phenomenon when hemolysis exceeds the compensatory capacity of the bone marrow hematopoiesis. Hemolysis occurs when RBCs are destroyed intravascularly, extravascularly in the reticuloendothelial system, or both. The possible pathophysiological mechanisms underlying hemolytic anemia in patients with ACS and/or PCI are numerous. The primary extravascular mechanism is sequestration and phagocytosis. Which occurs when RBCs with poor ability to inable to change shape enough to pass through the spleen. In this case, the patient has acute hemolytic anemia, with no history of hemolytic anemia before, which makes this mechanisms occurs improbably. The intravascular mechanisms include direct cellular destruction, fragmentation, and oxidation. Direct cellular destruction is caused by toxins, trauma or lysis. Fragmentation hemolysis occurs when extrinsic factors produce shearing and rupture of RBCs. Oxidative hemolysis occurs when the protective mechanisms of the cells are overwhelmed. PCI use indovascular devices, which may cause shearing and rupture of RBCs theoretically, but in reality the trauma is so small to observed. In immune-mediated hemolytic anemia, antibodies bind with the RBCs, resulting in phagocytosis or complement-mediated destruction. The extrinsic nonimmune causes include microangiopathic hemolytic anemia (MAHA), infections, direct trauma, and drug-induced hemolysis, among others. The patient has a history of thoracalgia for 10-h, dysphoria and unconsciousness for 5-min, accepted one electric defibrillation, about 4 minutes of CPR, PCI, and IABP was used for two days. The combination of these treatments above may induce direct cellular destruction, fragmentation, and oxidation or immune-mediated hemolysis [13,14].

Hemolysis produces free hemoglobin. If the degree of hemolysis is mild, the released free hemoglobin will be bound by circulating haptoglobin. However, if the degree of hemolysis is severe, the haptoglobin reserve will be exhausted. Excess free hemoglobin can be filtered out from the glomerulus, and hemoglobinuria occurs when the free hemoglobin exceeds the reabsorption capacity of the proximal convoluted tubules.

The patient in this case is a male, middle aged. Firstly, for the patient, the interventional therapy was successful, without perforations, dissections, hemodynamic collapse, reflow, entrapped equipment or other complications7. Secondly, the cause of hemolysis after other interventional cardiovascular procedures is mostly mechanical injury caused by physical factors. It is reported that 49 of 1123 (4.4 %) patient experienced hemolysis after using IABP [15], which was defined as peak plasma free hemoglobin 50 mg/dL occurring at least once during the device run and sustained for at least 2 days. The patient used IABP for two days as adjunct devices for LV unloading, hemolysis developing after one week of using IABP. Thirdly, patients with hemolysis may present with acute anemia, jaundice, hematuria, dyspnea, fatigue, tachycardia, and possibly hypotension. Laboratory test results that confirm hemolysis include reticulocytosis, increased lactate dehydrogenase, increased unconjugated bilirubin, and decreased haptoglobin levels8. Blood smear examination and direct antiglobulin test (DAT) is the cornerstone of acquired formal diagnosis, but may be affected by various drawbacks [13]. The differential diagnosis should rule out possible hemoglobinopathies, membranopathies, enzymopathies and extrinsic nonimmune causes [16]. The patiend had a symptom of dark brown urine and abnormal levels of RBC, Hb, reticulocyte and unconjugated bilirubin (UCB). In this case, the pathogenesis of hemolysis after PCI is not clear, but it is speculated to be physical or immune-mediated (drug-induced immune and secondary autoimmune [14]) haemolysis, or both. The potential causes of hemolytic anemia in ACS patients undergoing PCI are numerous [13,17] (Table 5).

Several hemolytic markers can be used to guide the differential diagnosis of hemolysis and monitor the therapeutic effect of

Table 5
The potential causes of hemolytic anemia in ACS patients undergoing PCI.

	Class/type	Diseases	Mechanism
Immune-mediated	Alloimmune	Transfusion reactions	Trapping, phagocytosis, complement
	Autoimmune hemolytic anemia	Warm autoimmune, cold agglutinin disease, paroxysmal cold hemoglobinuria, paroxysmal nocturnal hemoglobinuria	Trapping, phagocytosis, complement
Extrinsic nonimmune causes	Microangiopathy	Thrombotic thrombocytopenic purpura	Fragmentation
		Disseminated intravascular coagulation	Fragmentation
		Hemolytic uremic syndrome	Fragmentation
	Drug induced	Drug-induced thrombotic microangiopathy, drug-induced immune hemolytic anemia	Direct, toxin, phagocytosis, fragmentation, oxidation
	Infection	Malaria, Rickettsia, haemophilus influenzae, human immunodeficiency virus	Direct, toxin, phagocytosis, fragmentation, oxidation
	Systemic disease	Malignant hypertension, systemic lupus erythematosus, scleroderma, liver disease, vasculitides, hypersplenism	Trapping, fragmentation
	Trauma	Endovascular devices, aortic stenosis, extracorporeal membrane oxygenation, arteriovenous malformation	Fragmentation, direct

hemolysis. They mainly include reticulocyte (an indicator of marrow compensatory response), lactate dehydrogenase (a marker of intravascular hemolysis), haptoglobin, and unconjugated hyperbilirubinemia13. Hb defines the clinical severity of hemolysis, while thrombocytopenia suggests the possibility of thrombotic microangiopathy or Evans' syndrome. In addition to hemolysis, an increase in reticulocytes, lactate dehydrogenase and bilirubin, as well as a decrease in haptoglobin can also be observed in other conditions, which may confuse clinical diagnosis. A comprehensive clinical evaluation and laboratory examination could help to correctly diagnose and treat hemolytic diseases of different etiologies. If conventional laboratory tests are unable to detect the underlying cause of hemolysis, genetic testing may be considered [18].

Early clinical identification of the etiology of hemolysis after PCI will aid in the development of individualized treatment strategies and clinical management. Prevention is the key to the treatment of mechanical hemolytic anemia. Perioperative care should focus on etiological treatment, major organ protection and blood management [19]. Prior to the interventional therapy, some patients may need medications to control the infection or autoimmune disease to avoid mechanical hemolytic anemia. Besides, major organ protection should focus on the heart, kidney, liver, and the hematological and coagulation systems, since major postoperative complications and circulatory supporting devices applications are related to these organ dysfunctions. In addition to RBC, other blood products such as plasma and PLT should also be considered to correct coagulopathy. Tests such as Eosin-5-Maleimide binding assay, haemoglobin electrophoresis, G6PD/PK levels, and iron assessment should be performed in patients to identify a predisposition to haemolysis2. For immune-mediated HA, disease management involves corticosteroids [20], (potentially in combination with other medications such as azathioprine and cyclosporine), or targeting the underlying clonal B-cell proliferation or the classical complement activation pathway. The disease has proinflammatory properties and prothrombotic, and anticoagulant therapy should be initiated at the time of diagnosis. Additional therapies include RBC transfusion to support blood oxygen content. Future therapies may include therapeutic plasma exchange, anti-CD20 monoclonal antibodies, and complement inhibitors. For the patient, he finally recovered after blood transfusion of 2 units, dexamethasone treatment, and repeat PCI with no symptoms of further episodes of hemolysis.

3. Conclusions

GIB is often observed as major bleeding events after PCI due to the use of antiplatelet and anticoagulation in patients. But this case is special and does not belong to conventional GIB. So it is of great significance to recognize the reason of anemia after PCI to implement targeted treatment. In this case, we ruled out other diseases by auxiliary tests, and finally diagnosed as HA. This is a rare case of hemolysis after PCI. Although the association between PCI and HA is uncommon, PCI is now a commonly treatment in patients with ACS. It is important for clinicians to recognize this life-threatening complication as early as possible. Early diagnosis and therapy of HA is one of the key factors in ensuring the long-term prognosis of PCI patients.

CRediT authorship contribution statement

Yu Zhang: Writing – original draft, Data curation, Conceptualization. **An Fuxiang:** Writing – original draft, Data curation. **Meizhu Yan:** Writing – original draft. **Yi Zhou:** Writing – review & editing, Supervision, Conceptualization. **Hongjun Bian:** Writing – review & editing, Supervision, Funding acquisition, Data curation, Conceptualization.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] J. Zuo, J. Xie, W. Yi, J. Yang, J. Zhang, J. Li, D. Yi, Results of transcatheter closure of perimembranous ventricular septal defect, Am. J. Cardiol. 106 (7) (2010) 1034–1037.
[2] S. Venier, N. Vaxelaire, P. Jacon, A. Carabelli, A. Desbiolles, F. Garban, P. Defaye, Severe acute kidney injury related to haemolysis after pulsed field ablation for atrial fibrillation, Europace 26 (1) (2023).
[3] K. Kim, N. Ehara, T. Koyama, Y. Furukawa, Successful transcatheter aortic valve implantation in a patient after an apico-aortic conduit for severe aortic stenosis complicated by haemolytic anaemia: a case report, Eur Heart J Case Rep 4 (6) (2020) 1–6.
[4] M. Jonveaux, R. Gallet, G. Mouillet, P. Lim, E. Teiger, J. Ternacle, Acute haemolysis after transcatheter mitral valve implantation, Eur. Heart J. 40 (5) (2019) 488–490.
[5] P.J. Ngu, R.W. Harper, A. Nasis, Percutaneous repair of acquired Gerbode defect complicated by hemolysis and acute kidney injury, Int. J. Cardiol. 204 (2016) 37–39.
[6] J. Apel, R. Paul, S. Klaus, T. Siess, H. Reul, Assessment of hemolysis related quantities in a microaxial blood pump by computational fluid dynamics, Artif. Organs 25 (5) (2001) 341–347.
[7] K. Konstantinou, T.R. Keeble, P.A. Kelly, O. Alsanjari, L.C. Napp, G.V. Karamasis, J.R. Davies, Protected percutaneous coronary intervention with Impella CP in a patient with left main disease, severe left ventricular systolic dysfunction and established hemolysis, Cardiovasc. Diagn. Ther. 9 (2) (2019) 194–199.
[8] S.R. Kandan, T.W. Johnson, Management of percutaneous coronary intervention complications, Heart 105 (1) (2019) 75–86.
[9] L. Azzalini, D. Karmpaliotis, R. Santiago, K. Mashayekhi, C. Di Mario, S. Rinfret, W.J. Nicholson, M. Carlino, M. Yamane, E. Tsuchikane, E.S. Brilakis, Contemporary issues in chronic total occlusion percutaneous coronary intervention, JACC Cardiovasc. Interv. 15 (1) (2022) 1–21.
[10] A. Mitsis, F. Gragnano, Myocardial infarction with and without ST-segment elevation: a contemporary reappraisal of similarities and differences, Curr. Cardiol. Rev. 17 (4) (2021) e230421189013.
[11] J. Atwood, Management of acute coronary syndrome, Emerg Med Clin North Am 40 (4) (2022) 693–706.
[12] M.W. Chin, G. Yong, M.K. Bulsara, J. Rankin, G.M. Forbes, Predictive and protective factors associated with upper gastrointestinal bleeding after percutaneous coronary intervention: a case-control study, Am. J. Gastroenterol. 102 (11) (2007) 2411–2416.
[13] J. Phillips, A.C. Henderson, Hemolytic anemia: evaluation and differential diagnosis, Am. Fam. Physician 98 (6) (2018) 354–361.
[14] Q.A. Hill, R. Stamps, E. Massey, J.D. Grainger, D. Provan, A. Hill, Guidelines on the management of drug-induced immune and secondary autoimmune, haemolytic anaemia, Br. J. Haematol. 177 (2) (2017) 208–220.
[15] E.W. Grandin, J.I. Nunez, B. Willar, K. Kennedy, P. Rycus, J.E. Tonna, N.K. Kapur, S. Shaefi, A.R. Garan, Mechanical left ventricular unloading in patients undergoing venoarterial extracorporeal membrane oxygenation, J. Am. Coll. Cardiol. 79 (13) (2022) 1239–1250.
[16] W. Barcellini, B. Fattizzo, Clinical applications of hemolytic markers in the differential diagnosis and management of hemolytic anemia, Dis. Markers 2015 (2015) 635670.
[17] C.J. Scheckel, R.S. Go, Autoimmune hemolytic anemia: diagnosis and differential diagnosis, Hematol Oncol Clin North Am. 36 (2) (2022) 315–324.
[18] M. Jamwal, P. Sharma, R. Das, Laboratory approach to hemolytic anemia, Indian J. Pediatr. 87 (1) (2020) 66–74.
[19] J. Wang, H. Zhang, H. Fan, K. Chen, Y. Zhang, K. Song, H. Ao, C. Yu, Intractable mechanical hemolytic anemia complicating mitral valve surgery: a case series study, BMC Cardiovasc. Disord. 20 (1) (2020) 104.
[20] S. Berentsen, B. Fattizzo, W. Barcellini, The choice of new treatments in autoimmune hemolytic anemia: how to pick from the basket? Front. Immunol. 14 (2023) 1180509.