Case report

Salvage treatment for severe upper gastrointestinal bleeding caused by Mallory-Weiss syndrome and myocardial depression after acute myocardial infarction

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ABSTRACT

Complications related to upper gastrointestinal bleeding (UGIB) after acute myocardial infarction (AMI) and hyperkalemia-induced myocardial depression (MD) are not rare. However, severe UGIB caused by Mallory-Weiss syndrome (MWS) after AMI in patients with hyperkalemia-induced MD after red blood cell (RBC) transfusion has not previously been reported. We report a case of a 44-year-old male patient, presenting with AMI of the anterior wall, who underwent percutaneous coronary intervention (PCI). The patient developed two episodes of hema-
temesis within 12 h post-operation, the second of which was massive (about 2000 ml). Emergency endoscopy revealed MWS, thus titanium clips were simultaneously placed to stop the bleeding. In addition, the patient received RBC transfusion. One hour later, his blood pressure (BP) suddenly dropped to 63/35 mmHg, with arterial blood gas analyses revealing hyperkalemia. He was treated with bicarbonate, insulin and calcium gluconate to correct hyperkalemia, which lowered serum potassium and returned his cardiac function to normal. This case shows a dilemma in treatment of upper gastrointestinal bleeding caused by MWS after coronary stent implan-
tation, suggesting the need for timely hemostatic endoscopic procedures when this happens. Clinicians are advised to monitor patients’ vital signs in order to prevent serious damage caused by transfusion-related com-
lications. This is because complex and uncommon complications may begin with such common symptoms, which can rapidly deteriorate to life-threatening situations if not timely treated.

1. Introduction

Restoring coronary blood flow, through percutaneous coronary intervention (PCI), is the gold standard for management of acute myocardial infarction (AMI) [1], while both antiplatelet and anti-
coagulation therapies are integral components in the treatment of pre-
and post-PCI. Gastrointestinal bleeding (GIB), a complication of PCI following AMI with a 2% incidence, has been well documented [2]. Mallory-Weiss syndrome (MWS) is a rare cause of upper gastrointestinal bleeding (UGIB) after AMI or PCI. However, patients with GIB following PCI have higher mortality rates than those with either GIB or post-PCI [3], due to the low efficacy of treatment therapies of UGIB after coronary stent implantation. Moreover, severe UGIB requires RBC transfusion to correct anemia. For decades, hyperkalemia, a life-threatening electrolyte imbalance, has been recognized as a complication of transfusion. Notably, transfusion-associated hyperkalemia can result in bradycardia and cardiac arrest, which may be fatal if not treated appropriately. In the present case, the patient developed hyperkalemia causing myocardial depression (MD) immediately after RBC transfusion, a phenomenon that had not been previously reported.

2. Case report

A 44-year-old male patient was admitted to our emergency depart-
ment with a half an hour abdominal pain, mainly epigastric, that was accompanied by bloating, nausea and vomiting. Vomiting was the stomach contents without hematemesis. He had no fever, cough, chest pain or tightness, although electrocardiogram (ECG) analysis revealed AMI of the anterior wall (Figure 1). Emergency laboratory analysis showed hemoglobin (Hb) level of 151 g/L, cardiac troponin I (cTnI) level of 0.2 ng/ml, creatine kinase MB (CK-MB) level of 8.7 ng/ml, and normal blood coagulation. He was administered with aspirin 300 mg and
ticagrelor 180 mg. Next, he was subjected to coronary angiography (CAG), via the right radial artery, which revealed total occlusion of the left anterior descending coronary artery (LAD) after the first diagonal branch (D1), as well as 80, 30 and 20% stenosis for the opening of the D1, middle of left circumflex coronary artery (LCX) and middle of right coronary artery (RCA), respectively (Figures 2A & 2B). He was intraoperatively administered with heparin (a total of 6500 u) then a stent (3.5 × 33 mm drug-eluting stent) implanted in the LAD (Figure 2C). After the surgery, the patient was transferred to the department of cardiac intensive medicine. Approximately 5 h post-operation, he vomited approximately 300 mL of bright red blood, after ingesting a morning meal and was administered with somatostatin as well as Yunnan Baiyao. Numerous hematemesis, about 2000 ml, reappeared around 12 o’clock. His BP decreased rapidly to 65/53 mmHg, while his heart rate (HR) increased to 125 beats/min with trance. At identical time, he showed signs of irritability cold moist limbs. He was subjected to emergency gastroscopy, and gastroscopy revealed tears in his cardia mucosa (Figure 3A). The tear was clamped using a titanium clip, which stopped active bleeding (Figure 3B). By now the analysis of arterial blood gas revealed potassium concentration of 4.9 mmol/L. After endoscopy, he was given a quick transfusion comprising 4 units of RBC suspension, due to massive blood loss caused by hematemesis. Consequently, his BP returned to normal. However, the BP suddenly dropped to 63/35 mmHg 1 h later, although there was no drainage fluid from the gastric tube. The patient failed to have any symptoms of chest pain and ECG monitoring didn’t reveal signs of myocardial reinfarction. Analysis of arterial blood gas revealed a PH of 7.32, potassium concentration of 7.2 mmol/L, as well as blood glucose and calcium concentrations of 14.60 and 1.0 mmol/L, respectively. Moreover, bedside ultrasound analyses revealed a left ventricular ejection fraction (LVEF) of 20%. Considering that hyperkalemia-induced myocardium depression, bicarbonate, insulin and calcium gluconate were administered in combination with correcting hyperkalemia, dopamine and norepinephrine were administered to maintain BP levels. Two hours later, potassium levels returned to normal, LVEF increased to 50% while his haemodynamics stabilized.

Informed Consent: Written informed consent was obtained from patient who participated in this case report.

3. Discussion

We described a salvage treatment process for a patient diagnosed with UGIB, caused by MWS, following PCI and MD due to RBC transfusion-associated hyperkalemia. Each of these complications is not rare, whether UGIB after AMI or cardiac dysfunction following transfusion-associated hyperkalemia. However, their rapid and simultaneous occurrence significantly exacerbates the corresponding incidence of inhospital mortality [4].

Occurrence of UGIB, after PCI, in patients with AMI has been associated with stress. However, this was not the primary cause of UGIB in the patient under the present study. MWS, also known as cardial mucosal tear syndrome, is characterized by longitudinal mucosal lacerations in the gastroesophageal junction that result in massive bleeding of the
upper gastrointestinal tract. Notably, MWS has been associated with a sudden increase in intra-abdominal or intragastric pressure, which is transmitted to the gastroesophageal junction, due to retching, vomiting and straining. In fact, this condition is strongly correlated with alcohol consumption. In the present study, the patient had consumed small amounts of beer and physical emetic prior to onset, which may have been the underlying cause of the acute increase in intragastric pressure during MWS development. Upon admission to the hospital and diagnosis of AMI, he underwent PCI. It is possible that intraoperative application of contrast agent and stress may have caused postoperative vomiting, thereby exacerbating MWS.

Perioperative anticoagulant and antiplatelet drug administration of PCI is an integral component, which has also been associated with increased risk of bleeding. Therefore, patients undergoing PCI present a unique challenge during management of their UGIB. To this end, clinicians need to strike a balance between preventing bleeding and avoiding thrombosis. Previous studies have demonstrated that proton pump inhibitor therapy can reduce the future risk of UGIB [4, 5, 6]. For patients with UGIB, following PCI, who show no improvement after conventional medication, such as use of acid-inhibitory drugs or hemostatic agents, or with hemodynamic instability, there is need to consider MWS diagnosis and early endoscopy to confirm the diagnosis and endoscopic hemostasis with titanium clips [7].

In patients with AMI, low hemoglobin level has been associated with mortality. Notably, each 1 g/dL decrease in hemoglobin below 14 g/dL implies a 20% increased risk of death [8]. RBC transfusion has been widely used as a rapid and effective method for management of blood-loss anemia in patients. However, application of RBC transfusion during AMI as well as its optimal thresholds remain controversial owing to a lack of uniform international guidelines. Previous studies have demonstrated that a restrictive strategy for RBC transfusion is generally beneficial for patients with UGIB [9, 10]. However, those trials excluded AMI patients. Therefore, decisions for RBC transfusion in patients with AMI and severe UGIB should be based on individualized characteristics and risk. In the present case, the patient vomited approximately 2000 mL of bright red blood, and was accompanied by hemodynamic insufficiency, thus he was subjected to a quick transfusion comprising 4 units of RBC suspension. However, the patient developed transfusion-associated hyperkalemia with resultant MD.

Hyperkalemia is a rare but potentially life-threatening complication of RBC transfusion. Notably, transfusion-induced hyperkalemia is commonly associated with the volume, route, and rate of RBC transfusion as well as the transfused unit itself, including storage time, hemolysis and irradiated blood [11, 12]. Previous studies have demonstrated that most patients manifesting transfusion-related hyperkalemic cardiac arrest were hyperglycemic, hypocalcemic, hypothermic, and acidic, which may further exacerbate potassium cardiotoxicity [11]. This is consistent with the symptoms manifested by the patient in the present study.

Fortunately, the patient received electrocardiogram monitoring, which enabled us to detect changes in his condition and administer timely treatment to prevent cardiac arrest, aside from MD.

This patient with complicated conditions, it is difficult to sum up in one sentence. Firstly, the reasons for massive bleeding of the upper gastrointestinal tract after AMI or PCI can be thought about as MWS, especially if the patient had retched, vomited, and strained. An early endoscopic procedure should be performed to go further to exclude other factors that may cause massive bleeding of the upper gastrointestinal tract and clarify the diagnosis. Secondly, UGIB requires different treatment options according to the cause and the amount of bleeding. If patients present with acute haemorrhage, we believe that an immediate blood transfusion is necessary. But blood transfusion is associated with several adverse effects to patients. Therefore, monitoring the patient’s electrolyte levels and vital signs during and after transfusion is imperative to effectively preventing serious transfusion-related complications.

Critical care medicine’s concern is mainly with the pathophysiological evolution of the disease and the integrity of treatment. It applies advanced diagnosis and monitoring technology to assess the condition continuously and dynamically, and effective treatment are attended to promptly. As the case shows, we believe that the good outcome in our patient is due in large part to the strictly monitoring and prompt treatment.

Declarations

Author contribution statement

All authors listed have significantly contributed to the investigation, development and writing of this article.

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Data availability statement

No data was used for the research described in the article.

Declaration of interest’s statement

The authors declare no conflict of interest.
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