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CASE REPORT

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Sigmoid volvulus after CABG surgery

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Abstract

We report a case of a geriatric patient who underwent CABG and developed sigmoid volvulus (SV) with recurrence. SV is a rarely reported complication of CABG. Timely diagnosis, management, and follow-up are strictly advised, particularly in geriatric patients. Immediate surgical measures are required to prevent volvulus-associated ischemia.

KEYWORDS

abdominal obstruction, sigmoid colon, sigmoid volvulus

1 | INTRODUCTION

Sigmoid volvulus (SV) is a rare but significant clinical condition fore-mostly described by Von Rokitansky in 1836, as a condition in which the sigmoid colon is twisted around its own base.[1] SV is the most common form of obstruction of the colon that is responsible for about 2%-50% cases of intestinal obstruction.[2] It occurs mainly in adults, most commonly in males within the age range of 40-80 years. Incidence in children is exceptionally rare with below 100 cases reported in literatures so far; however, SV-related morbidity in children is high.[3-5] Similarly, SV is considerably uncommon in pregnant women. In 2005 and 2015, 73 and 100 cases are reported worldwide, respectively.[6,7] From the etiological viewpoint, some anatomical traits, which increase the susceptibility to SV, include sigmoid colon redundancy that increases with advancement in age, dolichomensentery, and narrow base of the sigmoid mesentery.[8-10] SV is often presented with an acute onset and a typical tirade of abdominal pain, distention, and constipation. Other clinical features reported by patients include nausea, vomiting, rectal bleeding, anorexia, and diarrhea. Physical presentation reveals abdominal distention and tenderness, abnormal bowel sounds, empty rectum, and visible peristalsis, while gangrene or perforation and peritonitis are indicated by rectal melanotic stool or rebound tenderness and muscular defense.[11,12] Routine laboratory examinations for SV are normally not pathognomonic; rather, the presence of intestinal obstruction, bowel perforation, or ischemia needs to be established.[13] X-ray radiography of plain abdomen usually reveals dilated sigmoid colon and has aided 57%-90% of SV diagnosis.[5] But abdominal CT scan and MRI scan are by far more helpful in SV diagnosis, compared with X-Ray, as it can reveal whirled sigmoid mesentery.[14-16] Similarly, flexible endoscopy has been demonstrated to be of high diagnostic value as it can show the obstruction of the colon in a sphincter-like twist of the mucosa.[17,18] Another diagnostic approach is laparoscopy or autopsy, however, and accounts for a limited percent (10%-15%) of diagnosed cases.[19] Generally, mortality due to SV depends largely on the early diagnosis and treatment. Delayed diagnosis, which in some cases is unavoidable such as in pregnant women, children, and chronically ill elderly patients, leads to serious and fatal complications of gangrene, perforation, shock, wound infection, intra-abdominal abscess, and anastomotic leakage which often

result in emergency surgeries leading to increased mortality rate. Overall mortality rate of SV has been reported between 6% and 24%.[20] The mortality in the elderly patients with age above 70 years is much higher due to coexistence of serious complicating illnesses.[21]

2 | CASE PRESENTATION

A 78-year-old man was admitted to the emergency department, reporting generalized chest pain, particularly in left hemithorax. He stated that the pain started in the morning and later shifted to the left upper limb and lower jaw. Upon placing fist of the chest, he complained of the pain (Levine's sign—fist clenched to chest for the indication of ischemic chest pain) and emphasized that the pain is exacerbated by activity and reduced by rest and that the pain lasted for 15 minutes. He also reported shortness of breath during the daily activities but had no complaints regarding abdominal problems. His medical history includes hypertension, use of drug (opium), and cigarettes. Electrocardiography revealed ischemic heart disease, and therefore, 325 mg aspirin, 300 mg Plavix, 60 mg Enoxaparin, and 80 mg Atorvastatin and nitroglycerine were administered. Coronary artery angiography revealed three diseased vessels, involving coronary arteries. From echocardiography, the LVEF was estimated 40%. The patient's laboratory test results were as follows: Hb: 13, WBC: 10 500, PLT: 250 000, MCV: 90, blood urea: 30, creatinine: 0.5, Na: 137, and K: 3.8.

The patient underwent CABG surgery, and four vascular grafts from the saphenous vein were anastomosed between Lima to LAD and SVG to OM and SVG to D1 and SVG to PDA. During the surgery, due to the opening of the left pleural space, a chest tube was inserted into the left hemithorax. Heparin was used during the operation. After the operation, the patient was transferred to the ICU. Heparin was switched with enoxaparin. 24 hours after surgery, the patient was extubated, and oral nutrition was started. The milk of magnesia was prescribed due to the constipation. In ICU, the patient had the mean blood pressure of 130/74 and pulse rate of 90. The oxygen saturation was 98%. The urinary output within 24 hours after the operation was 2200 cc. He was afebrile, and laboratory tests 24 hours after surgery were as follows: Hb: 11, WBC = 11 000, MCV: 92, PLT: 215 000, blood urea: 32, creatinine: 0.6, Na: 140, and K: 3.9. Second day of postoperative follow-up, patient defecated while his only complaint was irritation of the urethra due to the presence of a urinary catheter. Phenazopyridine was prescribed, and the burning sensation was relieved. Following the day, he complained of severe hypogastric pain. He also developed fever (38°C) accompanied with nausea and vomiting. Physical examination revealed soft abdomen, but severe hypogastric tenderness was seen. Rebound tenderness was absent but the abdomen had mild distension. Laboratory test results obtained were Hb: 10.35, WBC: 16 000, PLT: 94 000, Cr: 0.6, Na: 135, and K: 3.8. Portable abdominopelvic X-ray was taken in a supine position. Colon distension was evident, and sigmoid volvulus was suspected (Figure 1A).

For this reason, an abdominopelvic CT scan with oral and IV contrast was taken. The scout view of the abdominopelvic CT scan clearly indicated the sigmoid volvulus. In the axial view, there was a complete whirl sign that indicates the twisting of sigmoid mesocolon (Figure 1B).

After performing the abdominal and pelvic CT scan, severe rebound tenderness was evident. His blood pressure, pulse rate, and temperature were 135/75, 110, and 38.5°C, respectively. Results of requested laboratory tests were as follows: Hb: 10.3, WBC: 16.2, RBC: 3.4, MCV: 85.9, MCH: 30.3, MCHC: 35.3, amylase: 655, blood sugar: 116, ALP: 229, blood urea: 66, Cr: 1.2, Na: 141, K: 3.6, AST: 427, ALT: 460, LDH: 1536, bilirubin total: 1.3, and bilirubin direct: 0.5. As per patient's clinical examination and the abdominal and pelvic CT scan, sigmoid volvulus was diagnosed. Initially, fluid therapy was started under the guidance of CVP and appropriate urine output was obtained. Antibiotics including ceftriaxone and metronidazole at the dosage of 1 g state and bid and 500 mg state and Q6h were prescribed, respectively. He underwent laparotomy where the dilated colon was pulled out of the abdomen. There was no evidence of gangrene in the small intestine and the

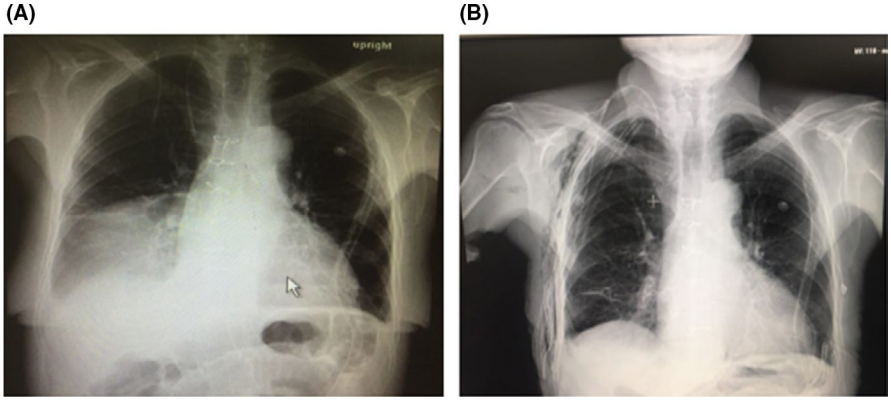

FIGURE 1 A-B, Colon distension and sigmoid volvulus were suspected

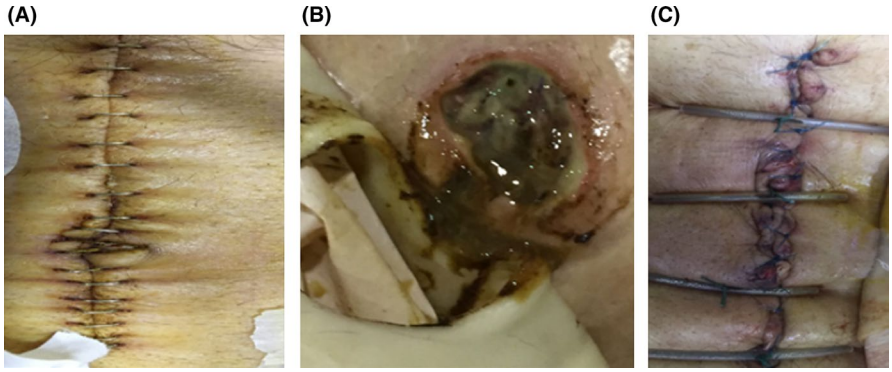

FIGURE 2 A-C, The colostomy sutured with silk and vicryl sutures

colon, but considering the inappropriate general condition of the patient and his bowels, sigmoid resection and primary anastomosis were avoided. Therefore, the sigmoid colon was resected, and Hartmann colostomy was embedded (Figure 2A). After the surgery, he was transferred to the ICU and intubated. The patient experienced a very stable condition in the ICU such that he had about 1600 cc urine within 7 hours after surgery. Vital sign and laboratory tests showed blood pressure: 104/70, HR: 81, O2 saturation: 98%, Tem: 37.2, PH: 7.43, PCO_2: 44.4, HCO_2: 29.5, BEb: 4.4, Hb: 10.2, WBC: 10.3, PLT: 113, PTT: 39, INR: 1.5, PT: 15, Cr: 1.1, Na: 140, and K: 3.5. 80mg of enoxaparin was administered daily, and the KCl 15% was started by dosage of 10cc/lit. Three hours after extubation, ABG was requested, which was completely normal. Oral food intake was resumed. Next day, patient underwent rapid atrial fibrillation and echocardiography did not reveal clot either four of the heart chambers and LVEF was estimated to be 40%. Amiodarone was prescribed, and the AF rhythm was controlled. Chest X-ray was taken (Figure 2B). In CXR, significant pleural effusion was evident in the right hemithorax; therefore, the chest tub No.28 was inserted in the right hemithorax. Approximately 1200 cc of serous fluid was alternately evacuated such that both hemithoraxes were cleared in the control chest X-ray, 12 hours after the insertion (Figure 2C). The patient was completely stable in the ICU.

Two days later, he suffered from respiratory distress and the accessory respiratory muscle activity was seen. The patient complained of severe shortness of breath. RR: 34, BP: 14/8, O2 sat: 85%, PR: 135, Tem: 38.4; Hb: 10.2, WBC: 24 000, PTT: 28, PT: 13, Platelet: 154, Cr: 0.9, Na: 135, K: 4; PH: 7.49, PCO_2: 20, HCO_2: 18.2, and BEb: −3.8.

An ECG was obtained which, in addition to the presence of RBBB, presented S1Q3T3, and pulmonary CT-angiography was conducted to find pulmonary embolus. Lung embolism was evident in the vascular branches of the lower segments of both lungs. Based on this, enoxaparin was discontinued, and heparin was started with a dosage of 1000 IU/h. PTT levels were kept between 45 and 90. The patient was treated provided oxygen via Venturi mask 60%. Laboratory results obtained were as follows: Hb: 10.4, WBC: 25 000, platelet: 70 000, PTT: 85, INR: 1.5, PT: 14, PH: 7.46, PCO_2: 27.6, HCO_2: 19.7, BEb:-3, BP: 13/75, PR: 100, O_2sat: 94%, Tem: 38, total protein: 2.4, and albumin: 1.3.

Due to the severe reduction in platelet count after the onset of heparin infusion, the possibility of heparin-induced thrombocytopenia was strongly suspected. Platelet PF4 antibody, PBS, and platelet serotonin release tests were requested. Dabigatran treatment raised platelet levels; however, considering the increase in WBC, the sepsis work-up was done. Blood and urine culture revealed no infection. The general condition of the patient did not allow reanesthetization for fascia repair, and low levels of protein and albumin in the patient also indicated a severe malnutrition. At this point, it was decided that the skin to be repaired with nylon thread in order to avoid incisional hernia, while fascia repair was decided to be performed later following the improvement in

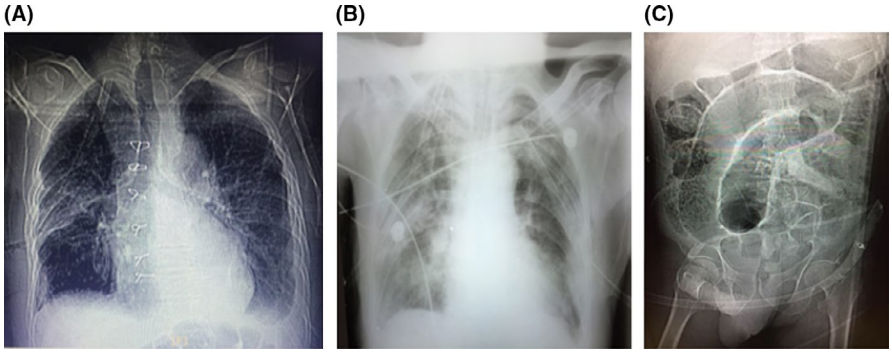

FIGURE 3 A-C, The lumen of the small intestine of the skin sutures

his condition. The patient's laboratory report was as follows: Plt: 192 000, WBC: 12.5, Hb: 8, BP: 122/88, HR: 97, O_2sat: 100%, RR: 17, and Tem: 37.5.

Considering the patient's cardiopulmonary condition, it was preferable to maintain hemoglobin levels above 10; hence, a unit of packed cell was transfused to the patient and the hemoglobin reached 9.8. Ferrous sulfate and folic

TABLE 1 displays laboratory values of patient, at different intervals

	Normal values	Patient's values
Before CABG	MCV: 80-100 fL MCH: 28-32 pg MCHC: 32-36 g/dL Amylase: 0-130 U/L Blood sugar: ALP: 36-92 U/L AST: 0-35 U/L ALT: 0-35 U/L LDH: ≤130 mg/dL Bilirubin total: 0.3-1.2 mg/dL Bilirubin direct: 0-0.3 mg/dL Blood pressure: 120/80 HR: 72 beats/min Oxygen saturation–95% or greater pH -7.38-7.44 PCO2 - 35-45 mm Hg Bicarbonate (HCO3)—23-28 meq/L BDb: Partial thromboplastin time (PTT): 25-35 s INR: Prothrombin time: 11-13 s Rate of respiration (RR): 12-20 breath/min PR: Total protein: 6.0-7.8 g/dL Albumin: 3.5-5.5 g/dL	Hb: 13 WBC: 10 500 PLT: 250 000 MCV: 90 Blood urea: 30 Creatinine: 0.5 Na: 137 K: 3.8
After CABG		Hb: 11 BC: 11 000 MCV: 92 PLT: 215 000 Blood urea: 32 Creatinine: 0.6 Na: 140 K: 3.9
Three days after CABG, at the time of abdominal symptoms		Hb: 10.35 WBC: 16 000 PLT:94 000 Cr: 0.6 Na: 135 K: 3.8.
After diagnosis of SV via X-ray and CT scans		Hb: 10.3 WBC: 16.2 RBC: 3.4 MCV: 85.9 MCH: 30.3 MCHC: 35.3 Amylase: 655 Blood sugar: 116 ALP: 229 Blood urea: 66 Cr: 1.2 Na: 141 K: 3.6 AST: 427 ALT: 460 LDH: 1536 Bilirubin total: 1.3, Bilirubin direct: 0.5
After SV surgery		Blood pressure: 104/70 HR: 81 O2 saturation: 98% Tem: 37.2 PH: 7.43 PCO_2: 44.4 HCO_3: 29.5 BEb: 4.4 Hb: 10.2 WBC: 10.3 PLT: 113 PTT: 39 INR: 1.5 PT: 15 Cr: 1.1 Na: 140 K: 3.5
After respiratory distress		RR: 34 BP: 14/8 O2 sat: 85%, PR: 135 Tem: 38.4 Hb: 10.2 WBC: 24 000 PTT: 28 PT: 13 Platelet: 154 Cr: 0.9 Na: 135 K: 4 PH: 7.49 PCO_2: 20 HCO_3: 18.2 BEb: −3.8.

(Continues)

TABLE 1 (Continued)

(Continues)

TABLE 1 (Continued)

Normal values	Patient's values
After detection of lung embolism and treatment	Hb: 10.4 WBC: 25 000 Platelet: 70 000 PTT: 85 INR: 1.5 PT: 14 PH: 7.46 PCO_2: 27.6 HCO_3: 19.7 BEb: -3 BP: 13/75 PR: 100 O_2sat: 94% Tem: 38 Total protein: 2.4 albumin: 1.3
After diet-based treatment	Platelet: 192 000 WBC: 12.5 Hb: 8 BP: 122/88 HR: 97 O_2sat: 100% RR: 17 Tem: 37.5 PTT: 44 PT: 17 PLT: 276 WBC: 14.6 Hb: 9.3 INR: 2 Cr: 0.4 Na: 135 K: 3.7.
After second onset of respiratory distress and hypotension	WBC: 14.1 Hb: 9 PTT: 38 PT: 15 INR: 1.6 Cr: 0.5 Na: 134 K: 3.4.
After treatment	PH: 7.12 PCO_2: 86.3 HCO_3: 28.3 BEb: -2.3
Before death	PH: 7.03 PCO_2: 110.4 HCO_3: 29.3 BEb: −3.4

acid tablets were also prescribed for the patient. Two days later, at the ICU, the lumen of the small intestine came out of the skin and it strangulated between the sutures (Figure 3A). Patient tests were as follows: PTT: 44, PT: 17, PLT: 276, WBC: 14.6, Hb: 9.3, INR: 2, Cr: 0.4, Na: 135, and K: 3.7.

The patient was transferred to the operating room immediately, and the abdominal wall was opened. The strangulated bowel was released, and the fascia was also repaired with looped nylon threads and tension suture (Figure 3B).

After transferring to the ICU, the patient suffered from severe hypotension such that the patient's blood pressure reached 70/40. With normal serum infusion and discontinuation of midazolam and morphine, blood pressure rose to 100/60. The third night he suffered from respiratory distress, where the rate of respiration increased, such that he was intubated. The laboratory test results were as follow: WBC: 14.1, Hb: 9, PTT: 38, PT: 15, INR: 1.6, Cr: 0.5, Na: 134, K: 3.4, PH: 7.12, PCO_2: 86.3, HCO_3: 28.3, BEb: −2.3. Unfortunately, two hours after the intubation, the blood pressure decreased, severely. There was no evidence of electrocardiographic changes in the ECG, BP: 50/40, PR: 119, and CVP: 5. Due to severe hypertension in the patient, norepinephrine was administered at a dose of 20 μg/min plus dopamine 5 μg/min. Despite prescribing vasopressors, the patient's blood pressure did not exceed 70/40. The patient's PCO_2 also increased with an increase in RR and PEEP in the ventilator set up, PH: 7.05, PCO_2: 109.4, HCO_3: 30.4, and BEb: −1.9. Portable chest X-ray showed (Figure 3C) the evidence of pulmonary edema. The ratio of PaO2/ FIO2 was <100, indicating severe ARDS. The suspected cause of ARDS was septic shock due to leukocytosis and severe nonresponder hypotension that was resistant to vasopressor and serum therapy. 30 cc/kg of ringer lactate serum was prescribed, and the tidal volume was adjusted to 6 cc/kg. And 100 mg hydrocortisone was also IV infused.

The patient's latest ABG, despite the treatment, was as follows: PH: 7.03, PCO_2: 110.4, HCO_3: 29.3, and BEb: −3.4. Finally, patient went into cardiac arrest, and in spite of the therapeutic measures, he did not respond to a cardiopulmonary resuscitation and died (Table 1).

3 | DISCUSSION

Management of SV is complicated and controversial, but typically it focuses on relieving the obstruction and preventing reoccurrence. Since SV is prevalent in advanced age groups who might present chronical illness, treatment options and outcomes are subjected to the presence or absence of certain adverse events like perforation and peritonitis. Treatment often commences with an appropriate level of resuscitation and patient stabilization to avoid severe anesthetic risk, followed by surgical procedures. Although many cases of SV have been managed successfully with flexible endoscopic decompression where feasible, surgical intervention is still strongly advocated due to the high rate of reoccurrence accounting for 40%-60% following endoscopy.

Our case presented the coexistence of two different pathological conditions (ischemic heart disease and sigmoid

volvulus). Studies have also shown that the presence of cardiovascular and respiratory diseases, hypotension, and leukocytosis are some of the common causes of mortality in elderly patients with SV.[22] A clinical case has been reported where atrial fibrillation and cecal volvulus were found together in a geriatric patient.[23] It is therefore suggested to use broad diagnostic approach for SV, particularly in geriatric patients.

4 | CONCLUSION

Cardiovascular disease can concomitantly exist with sigmoid volvulus, where advanced age is associated with the mortality in both the cases. It often presents delicate and complicated situation which would continue to require timely intervention to minimize mortality and morbidity. However, effective concurrent management of SV in the presence of critical medical condition(s) remains a difficult challenge at the instance of which SV continues to pose considerable threat to life, particularly in geriatric patients. In light of this case study, SV can be considered as one of the complications, associated with cardiovascular surgery in geriatric patients.

CONFLICT OF INTEREST

The authors deny any conflict of interest in any terms or by any means during the study. All the fees provided by research center fund and deployed accordingly.

AUTHORS CONTRIBUTIONS

Dr Ali Pooria: conceptualized and designed the study, drafted the initial manuscript, and reviewed and revised the manuscript. Dr Morteza Azadbakht: designed the data collection instruments, collected data, carried out the initial analyses, and reviewed and revised the manuscript. Dr Parham Khoshdani-farahani and Dr Afsoun Pourya: coordinated and supervised data collection, and critically reviewed the manuscript for important intellectual content. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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