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Case Report

Perils with Laparoscopic Surgery for Median Arcuate Ligament Syndrome

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Abstract

A 64 years, ASA 1 male patient with median arcuate ligament syndrome was posted for laparoscopic release of coeliac artery compression. The median arcuate ligament is a fibrous arch that unites the diaphragmatic crura on either side of the aortic hiatus. The ligament usually passes superior to the origin of the celiac artery near the first lumbar vertebra. . Infrequently, when this union occurs anterior to celiac axis it may cause hemodynamically significant stenosis compressing the vessel and nerves leading to median arcute ligament (MALS).The surgery of median arcuate ligament release for coeliac artery decompression involved working in the proximity of major vessels. Anticipation of major blood loss should be kept in mind that may be may be further compounded by the disease process in the vessels which make repair of vessels more difficult.Iatrogenic aortic injury happened in our case which was succesfully managed. Timely involvement of cardiovascular surgeons is important therefore the case has to be discussed with them prior proceeding for surgery. Hypothermia, hemorrhagic shock, coagulopathy, hypoxia, acidosis,pre-renal failure can be prevented with thorough preparation and timely intervention.

Keywords: Laparoscopy, massive blood transfusion protocol, median arcuate ligament

Introduction

The median arcuate ligament is a fibrous arch that unites the diaphragmatic crura on either side of the aortic hiatus. The ligament usually passes superior to the origin of the celiac artery near the first lumbar vertebra. Infrequently, when this union occurs anterior to celiac axis, it may cause hemodynamically significant stenosis compressing the vessel and nerves leading to median arcuate ligament. In the general population, 10%–24% of people may have indentation caused by an abnormally low ligament.[1]

Case Report

A 64-year-old, ASA, male patient with median arcuate ligament syndrome was posted for laparoscopic release of celiac artery compression. The presenting complaints were postprandial abdominal pain which was relieved by lying down in the prone position. He was a known tobacco chewer with no other comorbidities. The general and systemic examination was unremarkable except the heart rate – 50/min and blood pressure – 90/60 mmHg. All routine blood investigations were within normal limits. Electrocardiogram showed sinus bradycardia; however, two-dimensional echocardiography was normal with ejection fraction – 65%. Holter monitoring showed a minimum rate of 40/min; it revealed no arrhythmia. Computed tomography image showing median arcuate ligament syndrome in Figure1.

Preoperatively, tablet alprazolam 0.25 mg was given in the night before surgery and in the morning of surgery along with tablet pantoprazole 40 mg with sips of water. In the operation theater, standard monitors were attached and baseline readings of vitals were taken. After securing 16 G intravenous cannula, the patient was premedicated with fentanyl 100 µg and midazolam 1 mg. Intravenous induction was achieved with etomidate 32 mg followed by rocuronium 30 mg, and trachea was intubated with 8.5-mm endotracheal tube. Anesthesia was maintained with

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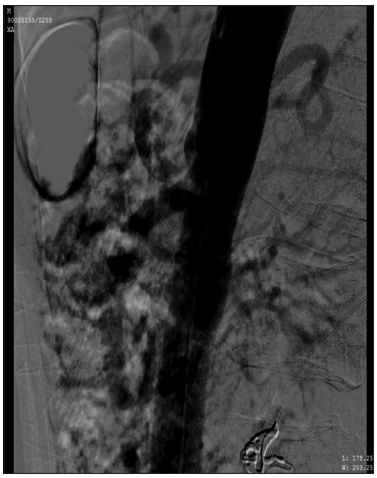

Figure 1: Computed tomography image showing median arcuate ligament syndrome

O_2: N_2O (40:60%) and isoflurane. Concentration of isoflurane was titrated to achieve a minimum alveolar concentration of 1. Triple lumen central venous line of 7 Fr was inserted in the right internal jugular vein, and the left radial artery was cannulated. Capnography and temperature monitoring were also established. Deep-vein thrombosis pump was applied, and forced air warming blanket was used to prevent hypothermia.

Pneumoperitoneum was created, and while attempting release of the median arcuate ligament, an iatrogenic nick was made in the aorta. While mechanically compressing the aorta, a midline incision was immediately placed. Promptly, cardiovascular surgeons were intimated. Meanwhile, hemodynamic stability was maintained with intravenous crystalloids, inotropes, blood, and blood products. Warm fluids and blood products were used. Aortic cross-clamping increases the left ventricular afterload. Thus, preparation for the cross-clamping including increasing depth of anesthesia and vasodilator therapy with nitroglycerin was kept ready.

Aortic cross-clamp was applied, and the primary nick was successfully repaired in 120 s, but unfortunately, while releasing the clamp, an accidental second nick was made in the diseased aorta at the clamp site. However, it was also successfully managed too.

Total estimated blood loss was about 3 l. Five units of packed cells, 5 units of fresh frozen plasma, and 5 units of platelet-rich plasma were used during the resuscitation. Urine output was measured vigilantly for every 15 min, and intraoperative arterial-blood gas (ABG) analysis was done at regular intervals. Successful release of arcuate ligament was done, and after achieving complete hemostasis, the abdomen was closed. Postoperatively, the patient was shifted to the Intensive Care Unit for elective ventilation and extubated on the next day.

Discussion

Median arcuate ligament syndrome (also known as Dunbar syndrome or celiac artery compression syndrome) was first described by Harjola in 1963.[1] The pathophysiology of the disease is external compression of the celiac artery by an abnormally low-lying ligament. During expiration, as the diaphragm moves caudally, the compression exaggerates. It is speculated that compression on celiac artery may result in impairment of intestinal perfusion, especially following enteral intake, and results in intestinal angina. Other investigators, on the other hand, implicate that the course is a neurological pathology of the celiac ganglion and consider this disease as a primary neurological disorder.[2]

Sustained compression of the celiac axis may lead to changes in vascular layers such as intimal hyperplasia, proliferation of elastic fibers in the media, and disorganization of the adventitia.[3] The surgery of median arcuate ligament release for celiac artery decompression involved working in the proximity of major vessels. Anticipation of major blood loss should be kept in mind that may be further compounded by the disease process in the vessels which makes repair of vessels more difficult. Timely involvement of cardiovascular surgeons is important; therefore, the case has to be discussed with them before proceeding for surgery.

Preoperative arrangement of adequate blood and blood product is necessary. Massive hemorrhage requires aggressive management, and the massive blood transfusion protocol followed in this case maintains adequate circulation and hemostasis.[4] Zealous interventions were done to avoid hypothermia, hypoxia, and acidosis. During aorta clamping, renal function shows a marked decrease in urine output, glomerular filtration rate, and renal plasma flow.[5] However, here, although clamp time was short during the period and after release of clamp, the renal perfusion and output are to be maintained.

The patient was kept on elective ventilation and extubated the next day after reviewing clinical status, hemogram ABG, and coagulation status, all of which were within normal limits.

These efforts were lifesaving and made the management of what could have been a catastrophe into a rewarding attempt that aided in successful revival and subsequent retreat of the patient to normal homeostasis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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