



MECHANICAL THROMBECTOMY OF SMA- A CASE REPORT

Surgery

Dr Paulia Devi T	Ms, Assistant Professor, Institute Of General Surgery, Madras Medical College , Chennai, Tamil Nadu, India
Dr Manivannan	Ms, Associate Professor, Institute Of General Surgery, Medical College, Chennai, Tamil Nadu, India
Dr Sindhu Niruba*	Resident , Institute Of General Surgery, Madras Medical College, Chennai, Tamil Nadu , India *Corresponding Author

ABSTRACT

Mesenteric Ischemia occurs most commonly due to occlusion of superior mesenteric vessels. Treatment options vary depending on the time of presentation and the extent of disease. Here presenting a interesting case of SMA thrombosis in a 65 years female for which Mechanical Thrombectomy was done. 65 years female presented with Complete thrombosis of Superior mesenteric artery with patchy gangrene of a segment of jejunum. We proceeded with Mechanical thrombectomy of SMA with resection of the segment of jejunum with Jejunostomy. Patient started on Heparin postoperatively and also given both enteral and parenteral nutrition. She was discharged on postoperative day 30. Acute thromboembolic occlusion of the superior mesenteric artery is a condition with a serious prognosis. Despite considerable advances in medical diagnosis and treatment, mesenteric vascular occlusion still has a poor prognosis with a inhospitable mortality rate of 59 to 93%. Although the prognosis of mesenteric ischemia is poor, a prompt diagnosis, aggressive surgical treatment and supportive intensive care could improve the outcome of this condition.

KEYWORDS

CASE REPORT

65 years old female came with complaints of abdominal pain for 3 days and vomiting for 1 day. She is a known case of systemic hypertension and CVA with left hemiparesis (recovered) on antiplatelets and statins. On examination her BP was 200/100 mmHg and PR was 88/min. Per abdomen examination showed mildly distended abdomen with diffuse tenderness in all quadrants and bowel sounds was absent. Her TC was 21,500/microlitre. CECT abdomen showed circumferential mixed plaque involving thoracic and abdominal aorta with 30% stenosis of thoracic aorta with 40% stenosis of abdominal aorta with mixed plaque noted at SMA origin with normal bowel wall enhancement noted with splenic infarct. Vascular surgeon opinion obtained and she found to have left side tibial disease with Monophasic flow in Anterior tibial artery, posterior tibial artery and dorsalis pedis artery and they advised CT angiogram. CT angiogram showed multiple plaque in arch of aorta, thoracic aorta, suprarenal abdominal aorta causing luminal narrowing with complete thrombosis of SMA 5.5 cm distal to its origin with splenic infarct noted in inferior pole with relatively reduced enhancement of small bowel loops. We proceeded with emergency laparotomy.

Intraoperatively she had patchy gangrene of jejunum of length 30 to 40cm which is approximately 30cm from DJ flexure with multiple areas of impending perforation noted. We did resection of gangrenous segment of jejunum with proximal jejunostomy with Distal mucous fistula.

Intraoperatively vascular surgeon opinion obtained. They proceeded with mechanical thrombectomy. SMA pulse not felt. SMA control taken, proximal ostial part of SMA pulse palpable and distally no pulse. Systemic heparinization given. Arteriotomy done and 4F Fogarty catheter passed into proximal SMA and upto 10cm thrombus extracted. 3F Fogarty catheter passed into distal SMA and retrieving upto 8cm of distal thrombus from distal branches. Arteriotomy closure done with 6/0 prolene. Good flow present. Congested bowel loops started to become pinkish after thrombectomy. Heparin infusion started after 6 hrs at a rate of 750 IU per hour. Patient was closely monitored postoperatively and started on sips of water on POD#5 and Soft solid diet on POD#7. Stoma was healthy and functioning.

Total parenteral nutrition also given every three days. Strict monitoring of electrolytes was done. Stoma output was approximately 400 ml per day and strict Input and output chart maintained. Vascular review obtained regularly and bridging of Heparin with acitrom done. Patient was discharged on POD#30 with strict post op advice of maintaining of Input and output, adequate salt and fluids intake, high protein diet. Unfortunately she died of short bowel syndrome a month

later.

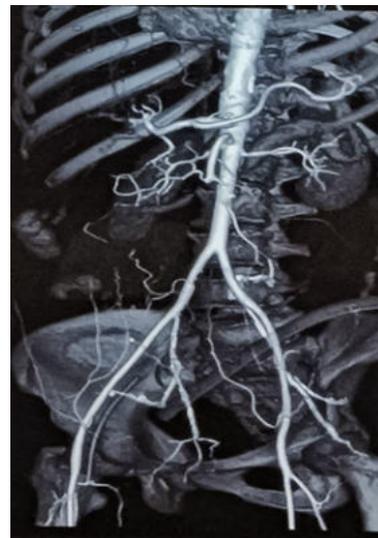


Figure 1 : CT ANGIOGRAM showing complete thrombosis of SMA 5.5cm distal to its origin.



Figure 2: Intraoperatively patchy gangrene of a segment of jejunum around 30cm from DJ flexure noted.



Figure 3: 4F Fogarty catheter passed into the proximal SMA and 10cm of thrombus extracted.



Figure 4: 3F Fogarty catheter passed into the distal SMA and retrieving upto 8cm of distal thrombus.



Figure 5: Resected segment of jejunum showing patchy gangrenous segments.



Figure 6: Retrieved thrombus from the SMA.

DISCUSSION

Intestinal wall is supplied by superior and inferior mesenteric arteries which ramify into mesenteric arcades as well as collaterals from proximal celiac, distal iliac and pudendal circulation. This pattern of circulation makes the intestine to tolerate slow progressive loss of bowel supply from the artery as in chronic occlusion. In contrast to chronic progressive hypoperfusion, acute compromise of any major vessel can lead to infarction of several meters of intestine. In vast majority of cases acute obstruction is caused by thrombosis or embolism. The most important risk factor for thrombosis is severe atherosclerosis. Less common causes include systemic vasculitides conditions such as thromboangitis obliterans and polyarteritis nodosa. Obstructive emboli most commonly originate from aortic atheroma or cardiac mural thrombus. Mesenteric venous thrombosis occurs due to inherited or acquired hypercoagulable state such as factor five Leiden disorder. Other causes include portal hypertension, portal pyemia, sickle cell disease and women taking oral contraceptives. Irrespective whether the occlusion is arterial or venous hemorrhagic infarction occurs. The mucosa is extremely sensitive to ischemic injury because of high metabolic activity. The intestine and mesentery become swollen and edematous especially with venous occlusion. Blood stained fluid exudes into the peritoneal cavity and the bowel lumen. The changes develop rapidly and irreversible injury ranging in severity from mucosal necrosis and sloughing to full thickness infarction. The clinical features include sudden onset of severe abdominal pain in a patient with arterial fibrillation or atherosclerosis. The pain is typically in the central abdomen and is out of proportion to all physical findings. Persistent vomiting and defecation occur early with subsequent passage of altered blood. Abdominal tenderness may be mild initially, with rigidity being a late feature. Shock with features of both hypovolemia and sepsis occur. Investigation will show a profound neutrophil leukocytosis, severe metabolic acidosis and raised blood lactate. CECT will show reduced or absent bowel wall enhancement and presence of free fluid in the abdomen with pneumatosis intestinalis. Treatment of mesenteric venous thrombosis includes anticoagulants and close monitoring. For arterial ischemia, early cases treated with immediate laparotomy with thrombectomy or embolectomy or revascularization of SMA by vascular bypass followed by post operative anticoagulants. For late diagnosis, mortality is high. In young all the affected bowel should be resected. In elderly inform the situation may be incurable. If the demarcation between viable and non viable bowel is uncertain plan relook laparotomy. After extensive enterectomy, patient requires parental nutrition. In selected cases consideration is given for small bowel transplantation.

CONCLUSION

Acute thromboembolic occlusion of the superior mesenteric artery is a condition with a serious prognosis. Despite considerable advances in medical diagnosis and treatment, mesenteric vascular occlusion still has a poor prognosis with a inhospitable mortality rate of 59 to 93%. Although the prognosis of mesenteric ischemia is poor, a prompt diagnosis, aggressive surgical treatment and supportive intensive care could improve the outcome of this condition.

REFERENCES

1. Klar E, Rahmaniyan PB, Buckner A, Hauenstein K, Jauch KW, Luther B. Acute mesenteric ischemia: A vascular emergency Dtsch Arztebl Int. 2012; 109(14): 249-56. Doi: 10.3238/arztebl.2012.0249. Epub 2012 Apr 6. PMID:22536301; PMCID: PMC3336145.
2. Jagielski M, Piatkowski J, Jackowski M. Challenges encountered during the treatment of acute mesenteric ischemia. Gastroenterol Res Pract. 2020;31: 2020:5316849.
3. Nagaraja R, Rao P, Kumar V, Yadav A, Kapoor S, Varma V, et al. Acute mesenteric ischemia-an Indian perspective. Indian J Surg. 2015;77(Suppl3):84349
4. Bala M, Kashuk J, Moore EE, Kluger Y, Biffl W, Gomes CA, et al. Acute mesenteric ischemia: Guidelines of the World Society of Emergency surgery. World J Emerg Surg. 2017;12:38. Doi: 10.1186/s13017-017-0150-5. PMID:28794797; PMCID: PMC5545843.
5. Treskes N, Persoon AM, van Zanten ARH. Diagnostic accuracy of novel serological biomarkers to detect acute mesenteric ischemia: A systematic review and meta-analysis. Intern Emerg Med. 2017;12(6):821-36. Doi: 10.1007/s11739-017-1668-y. Epub 2017 May 6. PMID: 28478489; PMCID: PMC5559578.
6. Martin J, Depietro R, Bartoli A, Markarian T, De Maria L, Di Bisceglie M, et al. Acute mesenteric ischemia: Which predictive factors of delayed diagnosis at emergency unit? Eur J Trauma Emerg Surg. 2023;49(5):1999-2008. Epub 2022 Sep 21.