Life-threatening severe sepsis following stapled hemorrhoidopexy

Sir,

A 59-year-old hypertensive, non-diabetic male was admitted for stapled hemorrhoidopexy (SH) following a long history

of rectal bleeding and associated prolapsing piles. He was receiving tablet metoprolol 50 mg and tablet amlodipine 5 mg since 2 years. On examination, he had 4th degree piles with a large external component. All pre-operative investigations were within the normal limit. Prophylactic antibiotics in the form of ceftriaxone and gentamycin were administered 30 min prior to surgery. A standard SH was performed under spinal anesthesia, but due to difficulty in closure some part of the hemorrhoids were also excised. During the intraoperative period, the patient felt uneasiness and some discomfort, hence was sedated with midazolam 1mg, but his mean arterial pressure (MAP) fell below 65 mm of Hg. He remained hypotensive for 10-12 min which was managed by administration of Ringer lactate 500 ml and tetrastarch 500 ml. This resulted in stabilisation of mean arterial pressure (MAP) above 70 mm Hg. A total amount of fluid administered during the intraoperative period was 1300 ml. Patient was shifted to the postoperative ward after completion of surgery. After 2 h of surgery, the patient complained of pain at the operative site. Intranasal butorphanol nasal spray 1 mg was administered to relieve the pain. Rest of the post-operative period on that day was uneventful. The patient did not pass urine until the morning of the next day. He also felt generalized weakness and breathlessness. At around 3 pm of the next post-operative day, his BP started falling to a MAP below 60 mm Hg despite administering 1L of 0.9% normal saline and hence he was shifted to intensive care unit within half an hour of developing hypotension. He did not pass urine until 4 pm inspite of being adequately hydrated and his BP at that time was 70 mm of Hg systolic. A central line was inserted and central venous pressure (CVP) guided fluid therapy started. We targeted a CVP of around 10 mm of Hg. Arterial blood gas (ABG) analysis was carried out and oxygen was administered through venturi mask at 6 L/min. Monitoring of non-invasive BP, pulse rate, heart rate, SpO2, temperature, continuous CVP, Urine output and abdominal girth was done. Patient's vitals at that time were: Temperature-102°F, BP 74 mm of Hg (systolic), pulse rate 134 bpm, respiratory rate 32/min, SpO₂ 78% on 6 L/min O, through mask, decreased air entry in both lung; fine crepts present, abdominal distension present, but no tenderness, and bowel sounds were absent. Urinary catheterization was performed. Electrocardiogram revealed sinus tachycardia; cardiac markers were sent and were within the normal limits

At the same time, infusions of noradrenaline and dopamine were started. All routine investigations with coagulation profile and serum procalcitonin levels were also sent for early recognition of sepsis ABG report revealed metabolic acidosis with low pO₂ (pO₂51.9 mm of Hg, pCO₂37.8 mm of Hg, pH 7.27,HCO₃17.5 mmol/L, BE – 8.7 mmol/L). Non-invasive, biphasic positive airway pressure ventilation started through mask. Blood cultures and fungal staining were sent. Injection meropenem 2 g intravenous (IV) was administered over 1 h followed by 1 g in 100 ml NS over 1 h twice a day, injection teicoplanin 400 mg IV stat then 400 mg once daily on alternate day (blood urea – 69 mg/dl, serum creatinine – 3.7 mg/dl) and injection metronidazole 500 mg IV three times daily were also started. After few hours of starting vasopressors (maintaining MAP about 60-70 mm Hg) patient passed urine about 50 ml in 1 h. Sequential organ failure assessment Score was 14.

As the patient's coagulation profile was deranged, so 6 units of fresh frozen plasma were transfused over 48 h. Chest physiotherapy, nebulization and general nursing care was instituted aggressively. Patient's hemodynamic parameters, urine output and ABG gradually improved, so we tapered the dose of noradrenaline and dopamine gradually. Intermittent oxygen inhalation was provided through Hudson mask. After 4 days of treatment, gradually the patient improved and was able to maintain his vitals without any support. He was shifted to room care and was discharged after nine days without any permanent sequelae.

Over the years, a number of cases of overwhelming sepsis have occurred as a consequence of the surgical treatment of hemorrhoids, in some cases proving fatal. From 2000 to the present, 29 articles reporting complications in 40 patients were identified.^[1] Thirty-five patients underwent laparotomy with fecal diversion and further patients were treated by low anterior resection. A specific complication was rectal perforation with peritonitis. Factors that led to life-threatening sepsis were identified in 30 patients. Despite surgical treatment and resuscitation, there were four deaths.[1] Three patients succumbed to sepsis in the immediate post-operative period, one on the post-operative day 4 after SH, second on the post-operative day 6 after SH and the third on the post-operative day 10 after SH. [2] SH seems to be elective and easy procedure, but devastating complications can occur within 24 h. SH has resulted in potential serious morbidity and even mortality in the immediate postoperative period.[2]

The cause of severe sepsis and retroperitoneal sepsis associated with both surgical and non-surgical treatment of hemorrhoids remains uncertain. [3] It has been postulated that stapling itself may allow bacterial entry into the perirectal region. Full-thickness wall stapling may also allow the organism to reach the peri-rectal space. [4] Two cases have been reported in which patients developed severe,

life-threatening complications. Incomplete rings (doughnuts) of excised rectal tissue were noted in both patients. [2,5] This could be a causative factor in our patient too, because difficulty in closure may have resulted in excision of some part of rectal tissue. There is no standard treatment for sepsis after SH and several different approaches have been used. [4] In our case, we recognized this life-threatening problem in an earlier phase and managed conservatively with non-invasive ventilation, appropriate antibiotics and general supportive and nursing care. Therefore, the presence of unexpectedly severe pain, respiratory distress, hypotension and urinary difficulties appear to be useful alarms for the presence of life-threatening perineal sepsis following treatment for hemorrhoids.

Our case report depicts that although SH is a simple, elective procedure for an experienced surgeon, but life-threatening complications can occur within 24 h of surgery. As anesthetists and intensivists, we should be aware of this complication and it's alarming signs and symptoms.

Apurva Agarwal, Bikram Kumar Gupta, Shaily Agarwal¹, Ajay Bhagoliwal², Kiran Pandey¹

Departments of Anaesthesiology and Critical Care Medicine, ¹Obstetrics and Gynaecology, and ²Social & Preventive Medicine, G.S.V.M. Medical College, Kanpur, Uttar Pradesh, India

Address for correspondence: Dr. Bikram Kumar Gupta, Room No. 39, New Married Hostel, L.L.R. Hospital, G.S.V.M. Medical College, Kanpur, Uttar Pradesh, India. E-mail: bikramgupta03@gmail.com

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