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Oglivie's Syndrome- Acute Colonic Pseudo Obstruction (ACPO)

Authors

**Dr. Rupa Talukdar¹, Group Captain JC Sharma², Brig SPS Kochar³, Col D Arora⁴,
 Lt Col Urmila Prasad⁵, Dr Namrita Sandhu⁶**

¹Senior Gynaecologist –Cantoment General Hospital, Delhi Cantt , New Delhi -10010

²Associate Professor, ³Professor & HOD ⁴Professor, ⁵Assistant Professor, ⁶Senior Resident. Army College of Medical Sciences , Delhi Cantt , New Delhi-110010

Corresponding Authors

Dr Rupa Talukdar

MD, Senior Gynaecologist, Dept of Obst & Gynaecology, Cantoment General Hospital
 Delhi cantt. New Delhi, pin 110010

Email: *talukdar.rupa@yahoo.in*

CASE REPORT:

A 23 yrs old unbooked primigravida at 31 weeks of gestation with triplet pregnancy presented with anaemia and PIH., she had inadequate ANC care.. she was admitted on 14/07/13 at 2200 hrs for ANC care for triplet pregnancy, treatment of anemia and ,mild PIH. She was already on antihypertensive labetalol 200mg TDS and haematinics. . Her menstrual history was regular with cycle of 3-4/ 30 days, LMP WAS 20 /12/12 & EDD was on 27/09/13.she had conceived spontaneously . the re was significant past history. She was a home maker and belonged to low socioeconomic status and was literate. There was

no significant family history. On admission she was asymptomatic with mild itching on abdomen and limbs . no history /clinical feature suggestive of pre eclamsia, hypertesion, renal disease and hepatic diseases. There was no history of drug allergy or blood transfusion. On examination she was thin built , of average height and fairly nourished. There was moderate degree of pallor ,oedema, there was no clubbing, koilonychia, raised JVP , lymphadenopathy , thyromegaly andicterus,.. Her pulse was 84/min, regular, BP was 114/94 mm of Hg, and respiratory rate was 20/min regular. On abdominal examination the uterus was enlarged upto 36 weeks size with

palpable multiple foetal parts . three foetal hearts were audible and was confirmed by ultrasound examination. On admission her investigation reports were as Hb 7.8 Gm%, PCV 26.2, TRBC 3.46 milion/ml. TLC was 11,600/ml, platelet count was 3. lacs/ml , INR 1.00, PT – control 13 sec , test 13 sec. PTTK control 30 sec , pt 32 sec. . Her random sugar was 104 mg/dl, urea 16.2 mg/dl, s creatinine 0.7mg/dl, uric acid was 5.6 mg/dl, bilirubin –total 0.6mg/dl, direct 0.7mg/dl, total protein 6.3 mg/dl, albumin 3Gm/dl; ALT 179.5 iu/L, AST 368.2 She was put on haematinics, inj vit K ,inj betamethasone for surfactant induction. Her antihypertensive medication was stopped. She remained asymptomatic till 17/07/3 at 0600hrs when she had PROM on third day. She was taken up for emergency LSCS was performed under spinal anaesthesia and had three premature male baby was born . At operation she was transfused 1 unit of PRBC and 4 FPP. She was given postoperatively IV fluid, analgesics and antibiotics and kept under closed observation.

Postoperatively she had developed tahcypnoea, tachycardia 150/min and hypertension 160/90 mmof Hg, SPO2 fell to 70%, she was immediately resuscitated and evaluated for cause of tachypnoea and distension of abdomen with feature of mild intraperitoneal fluid.. There was no excessive vaginal bleeding urgently exploration of the abdomen was done under GA by opening the suture. There was moderate amount of f haemorrhagic fluid , no active bleeding or

loosened suture line was observed. Peritoneal lavage was done and she was kept in ICU on ventilator for management of severe PIH and Pulmonary oedema. With subsequent two days her condition improved and she was shifted out of ICU with antibiotics and analgesics. She became normotensive. She was on normal die had regular bowel movements and was recovering smoothly till she developed feature of sepsis with wound infection which was treated with local antiseptic dressing and systemic antibiotics.

On 28/07/13 (11th POD) she developed pain abdomen obstipation and nausea, bilious vomiting, and tachycardia. On evaluation clinical, ultra sound abdomen, plain x ray abdomen in erect posture, showed multiple fluid level in intestine with cecal dilatation was diagnosed as a case of acute large intestinal obstruction. Her laboratory investigation : was blood test – blood urea -13.8 mg/dl, s creatinine 0.7mg/dl, sodium 1136mg/dl, potassium 4 mEq/L, bilirubin -0.5mg/dl, albumen – 3 gm/dl, ALT-25.5 iu /dl, AST – 26.7 IU/dl. No organic cause of obstruction was detected. She was managed as acute colonic obstruction and was treated by conservative method of Ryle's tube nasogastric aspiration, injection pot chloride, IV fluid parenteral nutrition ,analgesic, antiemetics and antibiotics. She had recovered from intestinal obstruction gradually after 48 hours..

Such an acute surgical condition characterized by massive dilation of the colon in the absence of mechanical obstruction was named as Oglivie's syndrome- acute colonic pseudo

obstruction(ACPO) after Sir Heneage Oglivie who first described this condition in 1948.[1] It usually occurs in high-risk and seriously ill or postoperative patients. If untreated, massive distension can cause rupture or perforation of the caecum and faecal peritonitis, which are associated with a high mortality.[2]

INCIDENCE AND CLINICAL SIGNIFICANCE

The true incidence of ACPO is unknown. There are there are no predisposing factors to indicate which group of women are likely to develop this syndrome .this syndrome is relatively rare despite the rising caesarean section rate. There is no reliable national or international data which tells about it's frequency.most cases are mild and resolve spontaneously [3].the most common operative procedure associated with this syndrome is caesarean section .[5–10] It has also been described after vaginal delivery,[8]forceps delivery,[11]caesarean hysterectomy,[12] and during pregnancy with preterm labour, pre-eclampsia and multiple pregnancies.[13–15] No parity is immune and there is no correlation between ACPO and indication for caesarean section.

PATHOPHYSIOLOGY

The exact pathophysiology of the condition is not known.it most likely involves an imbalance between the sympathetic and parasympathetic colonic innervation. the sympathetic and

parasympathetic nervous system act synergistically to maintain balance to achieve normal bowel function.

Ogilvie originally attributed the syndrome to parasympathetic deprivation.[1] Bachulis and Smith, in a review of 35 patients, suggested a possible role of prostaglandin abnormality.[18] A later analysis of the largest series on the condition suggested a temporary neuropraxia of the sacral parasympathetic nerves S2–4 as the underlying pathology.[4] These nerves pass through the inferior hypogastric plexus in close proximity to the cervix, vagina and broad ligaments, before terminating to supply the left colon, and could be injured during surgery or trauma in this area.

PREDISPOSING CONDITIONS FOR ACPO:-

Obstetrics and gynaecology

- Caesarean section, Caesarean hysterectomy, Vaginal/forceps delivery
- Total abdominal hysterectomy and bilateral salpingo-oophorectomy
- Radical hysterectomy, Vaginal surgery, Pelvic abscess, Pregnancy,

Surgery and trauma

- Pelvic surgery/trauma, Renal transplant, Hip surgery, Femoral fracture
- Retroperitoneal injury, Mechanical ventilation, Medical, Myocardial infarction
- Cerebrovascular accident, Liver or renal failure, Diabetic autonomic neuropathy
- Pneumonia, Herpes zoster infection Drugs
- Opiates/narcotics, Phenothiazines, Sodium polystyrene sulphonate-sorbitol enema, Tricyclic antidepressants Neoplasia
- Retroperitoneal tumour, Leukaemia, Pelvic radiotherapy, Choriocarcinoma

These conditions can alter the autonomic regulation of the colon, either by excessive parasympathetic suppression or sympathetic stimulation, or both. This leads to an adynamic distal colon that is similar to Hirschsprung's disease, one difference being with normal ganglion cells observable on autopsy unlike the Hirschsprung's disease. [3] the obstruction in ACPO is functional or 'pseudo'. Recent data indicate that the volatile short-lived gas, nitric oxide (NO) which is one of the major inhibitory neurotransmitters released by enteric neurons, can be overproduced in some pathological conditions, causing gut dysmotility and dilatation.[19] Volume depletion occurs as bowel contents stagnate in the dilated loops of the pseudo-obstructed bowel causing sequestration of fluid. Also fluid and electrolyte is lost through vomiting and as transudate into the peritoneal cavity. The caecum which is at the proximal end of large bowel with largest diameter lumen when the valve remains competent, preventing reflux into the ileum, a closed-loop obstruction develops. Therefore, a small amount of pressure leads to an enormous increase in its size and wall tension, and hence it dilates more rapidly than the remainder of the colon. If left untreated, it is liable to rupture through the serosa between the taeniae due to direct mechanical stretching. Retrospective data suggest a critical threshold diameter of 12 cm for the caecum for rupture, when the intracaecal pressure rises to more than 26 cm H₂O.[4,20,21] At this pressure, there is also ischemia in the

bowel wall because of the prolonged compression of the vessels, leading to risk of ischaemic necrosis and perforation..[16]

CLINICAL FEATURES AFTER CAESAREAN SECTION

The women usually presents with ACPO on 2–12 days post caesarean section. Signs and symptoms mimic those of mechanical large bowel obstruction. Most women (80%) have abdominal pain.[3] a dull cramps pain with no specific localisation, typical of hollow viscus distension is observed. Bowel movements cease or only a small amount of faecal fluid or flatus is passed. Nausea can be present but vomiting is usually a late symptom. The early features are tachycardia and a leucocytosis in the absence of any sepsis or signs of peritonism. The temperature is usually normal (pyrexia indicates sepsis, bowel necrosis and/or perforation) and progressive abdominal distension is the most typical finding (90–100%). Bowel sounds may vary – they can be hyperactive or normal (40%), high-pitched, tinkling or absent (60%)[3] – depending on the duration. The woman becomes acutely ill with dehydration, oliguria and features of electrolyte imbalance. Renal and liver function abnormalities may develop. Localized tenderness in the right iliac fossa over the caecum indicates impending rupture.[16] Laboratory findings are not diagnostic.

Differential diagnosis includes mechanical bowel obstruction, volvulus, bowel perforation, peritonitis and intra- or retroperitoneal bleeding .

The most useful diagnostic test is plain abdominal x-ray showing a typical picture of large bowel dilatation, especially the caecum which tails off at the splenic flexure or rectosigmoid. Mechanical obstruction can be ruled out by using a water-soluble radiographic contrast enema. In ACPO there will be free flow of the contrast medium to the caecum.[16] Pneumoperitoneum with gas under diaphragm confirms bowel perforation.

MANAGEMENT

Management of the condition is mainly conservative, general measures, drug treatment, endoscopic decompression and surgery. It should involve a multidisciplinary team of an obstetrician/gynaecologist, anaesthetist, gastroenterologist, surgeon, radiologist, midwife/nurse and theatre personnel.

CONSERVATIVE THERAPY

When caecal dilatation is less than 10 cm in diameter, initial treatment consists of a conservative approach, with the woman kept nil by mouth, Ryles tube nasogastric decompression and correction of fluid and electrolyte imbalance. Optimal body positioning and mobilisation out of bed are often advised.[20] However, most women with ACPO typically may not be ambulatory. In some women a central venous line may be warranted. In addition to administration of intravenous fluid, this facilitates monitoring of the woman's haemodynamic status. Fluid replacement is usually in the form of normal saline or Ringer's

solution, with colloids reserved for those women in severe hypovolaemic shock. Drug therapy should be reviewed: any medication that can adversely affect colonic motility — for example, narcotic analgesics and anti-cholinergic drugs — should be stopped, changed or avoided. Assessment of hourly urine output and fluid balance are monitored. Blood culture is indicated if sepsis is suspected. Due to depleted blood volume in the first week after a caesarean section these women are at an increased risk of venous thromboembolism (VTE); they should, therefore, be commenced on VTE prophylaxis. If the condition occurs during pregnancy, the fetal condition should be monitored carefully and antenatal corticosteroids administered if the gestation is less than 34 weeks. ACPO by itself is not an indication for delivery by caesarean section. Rieger et al and Imai et al. describe two cases during pregnancy that were successfully managed by conservative therapy. [14,,22] Both women underwent caesarean sections later for obstetric indications but without any recurrence of the condition after the operation. Conservative therapy can be tried for 24–48 hours before pharmacological or endoscopic intervention. The reported success of conservative management alone is variable, with rates from 20–92%. [3]

PHARMACOLOGICAL THERAPY

A variety of pharmacological agents, for example, erythromycin, metoclopramide and cisapride (which has now been withdrawn), have been tried for active reversal of ACPO with inconsistent

results.[3] The only consistently positive results have been with neostigmine.[23-29] In particular, a recent prospective, double-blind, placebo-controlled trial has shown promising results: 50% of patients had an immediate clinical response within a median time of four minutes (range 3–30 minutes) in the treatment group compared with no response in the placebo group ($P = 0.001$); and 73% had a sustained response three hours later in the treatment group compared with 20% in the placebo group ($P = 0.04$). However, this study is too small to be significant for the risk of colonic perforation and mortality.

NEOSTIGMINE

Treatment consists of an intravenous injection of 2.5 mg neostigmine administered over a period of 3–5 minutes. The response is noted after 30 minutes and again after three hours. Immediate clinical response is defined as the passage of flatus with a reduction of abdominal distension on physical examination within 30 minutes. A sustained response is measured by a reduction in abdominal girth and colonic diameter on abdominal x-rays three hours after the injection. The rationale for using neostigmine is to restore the 'normal' autonomic balance by inhibiting acetylcholinesterase activity to facilitate impulse transmission across myoneural junctions in the bowel wall. Side effects include excessive salivation, vomiting, bronchoconstriction, symptomatic bradycardia, hypotension and syncope.[26] Hence, such treatment must be

accompanied by close monitoring of cardiorespiratory status and atropine must be available as an antidote for these muscarinic adverse effects. Prior to neostigmine therapy, a mechanical bowel obstruction must be ruled out. A diagnosis of acute colonic pseudo-obstruction was made and 2 mg of intravenous neostigmine was administered, with resolution of the patient's condition allowing for subsequent Emergency Department discharge. This report discusses the utilization of neostigmine, an acetylcholinesterase inhibitor, for patients with colonic pseudo-obstruction.

In addition, women with the following are not considered suitable for medical treatment with cholinergic agents :[27]

- baseline pulse rate of less than 60 beats per minute
- systolic blood pressure of < 90 mm Hg
- clinical and radiological signs of bowel perforation
- active bronchospasm requiring treatment
- prior treatment with prokinetic drugs, for example metoclopramide, less than 24 hours before evacuation
- active gastrointestinal bleeding
- serum creatinine level of > 260 $\mu\text{mol/l}$
- documented hypersensitivity to neostigmine.

Other acetylcholinesterase inhibitors (for example, pyridostigmine and physostigmine) are not widely used: intravenous formulations of pyridostigmine are not always available and physostigmine easily

crosses the blood–brain barrier. A selective peripheral opioid receptor antagonist has been shown to show rapid return of flatus and bowel movements, earlier hospital discharge with no increased pain. New colokinetic agents, including 5-HT(4) receptor agonists and motilides (motilin receptor agonists), may represent other useful medical therapeutic options to treat ACPO.

ENDOSCOPIC THERAPY

Colonoscopic decompression used for those who do not respond to, or who relapse after neostigmine, although its efficacy has not been established in randomised clinical trials.[20] Among the series with more than 20 cases, the reported success rate varied from 61–78%.[20] However, because this procedure is usually carried out as an emergency on an unprepared bowel, it can be technically difficult to ensure patency of the suction channel of the scope. It can fail because of refractory caecal dilatation and can precipitate bowel perforation (in up to 3% of patients). Condition may recur in up to 40% of patients after initial decompression. Other treatments, for example caecostomy, have been used to provide colonic venting. However, the optimal caecostomy technique has not been established and it has its own complications, for example: pressure necrosis from the external bumper of the tube, development of profuse granulation tissue, cellulitis of the abdominal wall and sepsis.

OPEN SURGERY

Open surgery is mandatory when perforation or ischaemia are suspected. It usually involves bowel resection with temporary diversion, requiring a second stage closure. Stoma care forms an important part of care after resection surgery. Surgical management carries greater morbidity. Potential complications include abscess formation, ileus and bleeding.

AFTERCARE

Although ACPO is rare in obstetric practice it has serious consequences in terms of maternal morbidity and mortality. abdominal distension in the early puerperium, particularly after a caesarean section or vaginal delivery, should always be carefully evaluated to rule out this condition. These young and apparently healthy mothers are psychologically traumatised by this unexpected complication after childbirth. They require adequate explanation of the condition, on repeated occasions if required, by a senior obstetrician and surgeon. Emotional support is vital, as is proper training for stoma care if a laparotomy with surgical resection is required. The woman and her family should be allowed enough time to settle down and reflect, and a trained stress counsellor may be helpful.

SUMMARY

ACPO is a well documented condition, which presents with features of large bowel obstruction without a mechanical cause. Obstetricians should

be aware that this rare complication usually occurs after caesarean section and can lead to serious maternal morbidity and mortality, mainly due to caecal perforation and/or ischaemia. Careful assessment of the gastrointestinal status of a woman who has had a caesarean delivery is essential. Plain abdominal x-ray is the most useful diagnostic test. ACPO should initially be treated conservatively with correction of metabolic, electrolyte and fluid imbalance. Most women respond to pharmacological therapy. When neostigmine is unsuccessful or contraindicated, a decompressive procedure, typically by colonoscopy, should be employed. Open surgery is mandatory if caecal rupture or necrosis is suspected. An early diagnosis and timely intervention is essential to avoid caecal rupture and its associated high maternal mortality rate. Proper explanation, adequate emotional support and appropriate follow-up by a senior obstetrician are vital in the management of the condition.

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