Case Report

Acute Pseudo-Obstruction of the Colon (Ogilvie’s Syndrome) a Recurring Diagnostic Dilemma

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Acute colonic pseudo-obstruction (Ogilvie’s Syndrome) is a rare clinical disease characterized by either total or segmental distension of the colon caused by paralysis without mechanical obstruction. The condition was first described in 1948 by Ogilvie when he postulated it as a condition caused by imbalance between the sympathetic and parasympathetic innervations of the colon. Up till now its etiology and pathophysiology still remains poorly understood. Though formerly associated primarily with the post operative state, this entity is increasingly being recognized in association with a wide variety of major medical problems. Clinically, the syndrome manifests itself as an acute abdominal distension, the caecum being the site of greatest dilatation, and the situation could be rapidly progressive and if left untreated it may cause caecal necrosis and perforation which could be fatal. Treatment remains largely empirical. The choice involves the time honored bowel rest of nasogastric tube decompression with supportive care; while a wide variety of pharmacologic approaches have been advocated. However, surgical intervention may be required if the required improvement is not achieved so as to avoid bowel perforation with its attendant mortality. The aim of this article is to draw the attention of clinicians to this rare disease; especially when colonic obstruction is suspected one should always consider the possibility of Ogilvie’s Syndrome as one of the differential diagnosis.

Keywords: Acute, Pseudo-Obstruction, Colon, Syndrome

INTRODUCTION

Ogilvie in 1948 (Ogilvie, 1948) first reported two patients with acute colonic distension highly suggestive of mechanical obstruction but in whom no distal obstruction was found. Ogilvie’s syndrome as it is otherwise now known is an infrequent pathology which has been the subject of numerous medical debates in the past two decades and whose etiology and pathophysiology remains poorly understood (Jetmore et al., 1992; Shi et al., 2011).

The actual incidence of this syndrome is unknown because spontaneous recovery may occur in some cases without it being reported. It has been thought to be of an imbalance between the sympathetic and parasympathetic innervations of the colon.

Though formerly associated primarily with the post operative states (Tenofsky et al., 2000; Norwood et al., 2005), the entity is now being increasingly recognized in association with a wide variety of major medical problems notably chronic renal failure, respiratory or cardiovascular illnesses (Kadesky et al., 1995; Edelman et al., 2010).

The causes of the syndrome as seen from the foregoing are multi-factorial (Schjoldager et al., 2001; Silverberg et al., 2001); however the three most common associations are trauma especially retroperitoneal,
serious infections, cardiac disease especially myocardial infarction and congestive heart failure.

It presents clinically with progressive soft but gross acute abdominal distension with colicky pain. It is characterized by massive total or segmental colonic dilatation in the absence of a mechanical cause. There might be visible and audible peristalsis as in mechanical obstruction, but others have no abdominal complaint whatsoever apart from the distension. The right colon is more usually affected. The caecum is the site of greatest dilatation and tenderness around the caecal area should raise suspicion of perforation (Soreide et al., 1977; Kuhn et al., 2003).

Diagnosis is established by the combination of clinical history (Ohkubo et al., 2012), physical examination and radiological findings (Choi et al., 2008). The plain abdominal X-ray usually shows gas-filled colon without fluid levels as a rule. A high index of awareness is required to diagnose this condition clinically.

The management is along the lines of general measures to reduce colonic distension (Fausel and Goff, 1985). Drugs that improve colon motility, endoscopic colonic decompression and surgery are all available modalities of treatment.

This report is therefore presented in a patient with acute colonic pseudo-obstruction (Ogilvie’s Syndrome) post emergency caesarean section managed surgically after failure of the usual bowel rest and failed colonoscopic decompression. It is intended as a wake-up call for all clinicians.

CASE REPORT

A thirty four (34) year old female patient thirty eight (38) weeks pregnant and in labour was admitted to the Antenatal ward of our hospital. Initial assessment of her labour showed a big baby with breech presentation and polyhydraminous.

Approximately thirty (30) minutes after admission, labour was not progressive as expected, the baby had foetal distress and she was therefore delivered by emergency lower segment Caesarean section. She had the usual injection of Syntocinon and methargin intra and post operatively after delivery.

About eighteen (18) hours post Caesarean section she started complaining of severe right sided abdominal pain without vomiting. A surgical consultation was requested when she failed to settle on simple administration of analgesics.

Clinical examination at this time showed a fairly anxious patient, BP = 110/60 mm Hg, Pulse = 80/min, Temperature 37.9 °C.

Abdominal examination showed gross distension, mild tenderness in the right upper quadrant of the abdomen with visible peristalsis along the region of the transverse colon. Abdomen was tympanitic with exaggerated bowel sounds. Rectal examination revealed empty rectum.

Significant in her past medical history was the fact that she had an umbilical hernia repair eight (8) years earlier.

Abdominal X-ray showed gross gaseous colonic distension with multiple fluid levels of the small bowel. Electrolytes and Urea values were within normal limits.

A provisional diagnosis of early intestinal obstruction probably secondary to adhesions from previous operation was made.

Abdominal ultrasound showed fluid and gaseous distension of the ascending and transverse colon with vigorous peristalsis.

Urgent gastrografin enema was done which showed normal passage of contrast into the caecum, with collapse of the left side of the colon and massive distension of the mid transverse, ascending colon down to the caecum. The caecal diameter was about twelve (12) cm radiologically.

She was treated conservatively with nasogastric tube decompression, nil by mouth and intravenous fluids since there was no evidence of mechanical obstruction.

The following morning her clinical condition was reviewed, but the progress made was not satisfactory. Abdominal x-ray was repeated which was reported as revealing a narrow segment of the colon immediately below the splenic flexure extending over and area of five (5) cm with smooth tapering raising the suspicion of a mechanical obstruction probably inflammatory or extrinsic in origin.

A colonoscopic examination was requested but failed to access beyond twenty five (25) cm of the colon. This patient continued to experience severe colicky abdominal pain, with progressive abdominal distension even though the abdomen remained soft. The bowel sounds still remained hyperactive and the patient was in absolute constipation.

A decision to carry out an exploratory laparotomy was then taken.

At laparotomy, no mechanical obstruction of the colon was found but there was gross distension of the whole right colon up to the mid-transverse colon (figure 1). The caecum was grossly distended with early signs of necrosis (figure 2).

An intra operative diagnosis of Ogilvie’s Syndrome (Acute Colonic Pseudo-Obstruction) was made.

Appendicectomy was carried out and colonic decompression done through the appendicular stump. A tube caecostomy was performed through the appendicular stump.

Her post operative period was uneventful. The caecostomy tube was removed on the eleventh (11th) post operative day and she was discharged home on the 12th post operative day.

She has since been discharged from follow up from the surgical out-patient clinic.
COMMENTS AND CONCLUSION

Acute colonic pseudo-obstruction (Ogilvie’s Syndrome) (Ogilvie, 1948) is an infrequent disease entity, the etiology of which is still poorly understood. A high index of awareness is required to diagnose (Ohkubo et al., 2012) this condition and the exclusion of a mechanical obstruction is important for further treatment of the disease.

Formerly associated primarily with a variety of post operative states (Clarke et al., 1997), the entity has increasingly been recognized with a wide variety of major medical problems.

The condition has been reported in a wide variety of post operative states following cases of caesarean sections, spinal surgery, various Orthopedic procedures and other procedures not related to the bowel (Norwood et al., 2005; Kadesky et al., 1995).

Many medical situations or conditions such as chronic respiratory diseases, chronic renal failure, electrolytes imbalance, systemic infections, drugs or even neurologic disease have all being associated with this syndrome. However in this case there was no evidence of electrolyte imbalance.

In chronic renal failure it has been suggested that the metabolic disturbances lead to an altered milieu interior of the neuromuscular system while in chronic respiratory disease the labored respiration leads to an excessive amount of air being sucked into the gastrointestinal tract with subsequent intestinal distension. The sympathomimetic drugs given to relieve bronchospasm may inhibit colonic motility and it is a combination of this inhibition of colonic motility and excessive sucking in of air that leads to the gross distension of the large bowel.

The pertinent point to bear in mind is the fact that this syndrome presents spontaneously as an acute abdomen (Kuhn et al., 2003) with colicky abdominal pain, distension with exaggerated bowel sounds and constipation and every effort must be made to exclude a mechanical aetiology.

The syndrome as it is known could affect the whole colon, but invariably segmental colonic involvement is the usual finding particularly on the right side of the colon with associated extensive caecal dilatation (Spira and Wolff, 1976) resulting sometimes in caecal perforation with its attendant mortality (Marinella, 1997; Kukora and Dent, 1977). The mortality rate can be as high as forty percent (40%) when perforation occurs.

Relevant ancillary investigations after adequate history and physical examination must include imaging studies such as plain abdominal x-ray and water soluble imaging enema to rule out mechanical obstruction (Choi et al., 2008; Stewart et al., 1984; Koruth et al., 1985).

After arriving at the diagnosis, conservative management (Fausel and Goff, 1985; Sloyer et al., 1988) in the form of nasogastric tube decompression, nil by mouth, intravenous fluids, electrolytes correction and sometimes passage of rectal tube for decompression provided there is no associated caecal perforation (Marinella, 1997). Colonoscopy both for diagnosis as well as for decompression could be instituted (Quigley, 2000; Vivatongs et al., 1982; Gosche et al., 1989).

Recently a wide variety of pharmacologic approaches have been advocated (De Giorgio et al., 2001; Breccia et al., 2001). Few have been subjected to, or have survived, the rigors of a properly controlled trial. However, neostigmine is a notable exception and has been shown to be effective in Ogilvie’s Syndrome (Paran et al., 2000; Trevisani et al., 2000; Amaro and Rogers, 2000; Loftus et al., 2002).

The search for new colokinetic agents continues, and among these agents, the potential of 5-hydroxytryptamine–4 receptor agonists and motilin receptor agonists is ongoing, even though some authors feel that recurrence of the condition after medical treatment is 20-50% and intra hospital mortality is thirty percent (30%).

Caecal necrosis and subsequent perforation is a significant threat in this syndrome especially if the diagnosis is made late and the caecal diameter is more
than twelve (12) cm. In this kind of situation, surgical decompression in form of either a caecostomy (open or laparoscopic) or even a right haemicolectomy might be the only option to save the patient’s life (Lobato et al., 1998; Duh and Way, 1993).

Treatment should be instituted as soon as possible before complications set in. The syndrome is an uncommon but serious condition and it is very important to make an early diagnosis as the condition can progress quickly to caecal necrosis as seen in this case and possible perforation with its attendant mortality.

The conclusion therefore is that when colonic obstruction is suspected, one should always consider the possibility of the occurrence of Ogilvie’s Syndrome when mechanical obstruction has been ruled out or excluded.

REFERENCES


