The Cast Syndrome – Vascular Obstruction of the Duodenum

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SUMMARY

Vascular obstruction of the duodenum has been reported as a consequence of the application of a cast or the correction of a spinal curvature following severe burn or combat injuries and complicating treatment of a fractured femur with balanced traction. This syndrome rarely develops more than a week after the application of a cast.

An unusual case is reported in which the onset was delayed, but progressed rapidly to a fatal outcome. The pathogenesis, clinical features and treatment of this condition are reviewed.

A high index of suspicion in patients at risk is essential as early diagnosis and prompt treatment are imperative.


In the surgical literature, vascular compression of the duodenum by the superior mesenteric artery is commonly called the superior mesenteric artery syndrome, while in orthopaedic practice it most often follows the application of a body cast and is usually known as the cast syndrome.

The syndrome is thought to result from mechanical compression of the third part of the duodenum by the superior mesenteric arterial trunk. Should this syndrome go unrecognized, acute gastric dilatation associated with nausea and vomiting rapidly gives rise to severe hypokalaemic alkalosis, hypovolaemia and death.

Although Von Rokitansky1 recognized the syndrome of obstruction caused by mesenteric vascular compression of the duodenum as early as 1861, the first case following application of a cast was described by Willett2 in 1878.

The bulk of surgical literature related to this syndrome concerns the chronic and more benign form. A more acute presentation, as a complication of other conditions, may be more common than is generally recognized.

The term ‘cast syndrome’, first introduced by Dorph,3 may be a misnomer, as vascular compression of the duodenum has been reported after correction of a spinal curvature without the application of a cast.4 Other predisposing conditions which have been reported include burn injuries in excess of 30% of the body surface,5 confinement to bed in combat casualties6 and even treatment of a femoral fracture with balanced traction.7

Since successful treatment requires early diagnosis, a high index of suspicion is necessary.

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A case following application of a cast, but with delayed onset and rapid progression to a fatal outcome, is reported.

CASE REPORT

A 7-year-old White girl was referred for treatment of torticollis. Her parents had been aware of the deformity for many years, but had not thought it significant.

Examination revealed a contracture of the left sternomastoid muscle, with a hard fibrotic mass present in the muscle belly. No other systemic abnormalities were noted.

Radiographs of the cervical spine were normal. After operative release of the sternomastoid muscle from its inferior origin, passive correction could easily be obtained.

The immediate postoperative course was uneventful. As the patient proved unco-operative during attempts at manual stretching, a Minerva cast was applied 6 days postoperatively. The cast extended from the iliac crests and held the patient’s head in the corrected position. Four days after application of the cast the patient was fully ambulatory and comfortable. The sutures were removed and the patient was discharged from hospital.

Nine days after discharge and 13 days after application of the cast the child developed nausea, retching and vomiting. A general practitioner diagnosed gastro-enteritis. He was not unduly concerned, but requested permission to remove the cast as the child was having difficulty in vomiting. Despite removal of the cast the patient died 4 hours later.

The first signs of retching had apparently appeared only some 12 hours before death. Treatment had not been more aggressive because no one had considered the possibility of a cast syndrome in an ambulatory patient nearly 2 weeks after the cast had been applied.

Autopsy Report

At autopsy an acute dilatation of the stomach and proximal duodenum was revealed. The wall of the stomach and proximal duodenum was intensely haemorrhagic, thickened and oedematous; a perforation was present on the anterior aspect of the lesser curvature of the stomach. Dissection of the coeliac and mesenteric arteries revealed no obstructive lesion. The rest of the bowel was normal. A fibrinous exudate of short duration was present on the peritoneum. The lungs were normal, with no signs of inhalation. No other significant abnormalities were found.

Microscopical examination of multiple sections revealed ischaemic necrosis of the entire thickness of the wall of the stomach and proximal duodenum, in marked contrast with the normal appearance of the rest of the gut.
DISCUSSION

Anatomy

The horizontal third part of the duodenum normally originates on the right side of the fourth lumbar vertebral body and passes to the left from where it curls forward around the spinal column to the point where it becomes the fourth part at the level of the second lumbar vertebra. The fourth part is relatively fixed by the ligament of Treitz. The superior mesenteric artery originates from the aorta behind the neck of the pancreas at the level of the first lumbar vertebra. The axis of the artery is directed towards the right lower quadrant of the abdomen, crossing anterior to the duodenum. The third part of the duodenum thus passes between the aorta and vertebral column posteriorly and the root of the superior mesenteric artery anteriorly, and may therefore be caught between immovable structures behind and the artery in front (Fig. 1).

![Fig. 1. Line diagram of the anatomical relationships between the third part of the duodenum and the superior mesenteric artery.](image)

Mansberger et al. used biplanar angiography to demonstrate that the angle of the superior mesenteric artery to the aorta in normal individuals varies between 45° and 60°. They reported this angle to be 10°, 12° and 22° in 3 patients with vascular compression of the duodenum. Similar findings were subsequently reported by Wayne et al. in 2 additional cases. Therefore the anatomical arrangement in this area predisposes to partial or complete anteroposterior obstruction of the duodenum when any condition that narrows the angle of origin of the superior mesenteric artery is present.

Pathogenesis

A number of theories explaining the decrease in the angle of origin of the superior mesenteric artery have evolved.

The duodenum crosses the lumbar spine at the summit of the normal lordotic curve, and it is suggested that the application of a cast increases normal lumbar lordosis, thus decreasing the angle between the spine and the superior mesenteric artery. Traction on the spine during correction of scoliosis similarly decreases the angle of origin of the artery. Eight cases have recently been reported after correction of a spinal curvature, but without the application of a cast.

The wide angle of origin of the superior mesenteric artery is normally kept open by a generous fat pad. Loss of body fat changes the support of the superior mesenteric artery from a bulky band to a linear structure. The resultant narrowing of the angle of origin of the artery by this mechanism has been suggested as the pathogenesis in emaciated bedridden combat casualties or after extensive burn injuries.

Diagnosis

Vascular compression of the duodenum, like any obstructive gastrointestinal condition, will be heralded by persistent nausea and vomiting. Tenderness in the epigastrium is found early in the course of obstruction before severe distension develops, and serves to differentiate the cast syndrome from postoperative ileus without obstruction.

Careful monitoring of gastrointestinal function is essential in patients in whom one of the predisposing conditions is present. Plain radiographs of the abdomen may demonstrate gastric and proximal duodenal distension with little gas in the remainder of the bowel. Contrast media studies should confirm the diagnosis. A sharp cutoff pattern on the oblique views and/or vigorous retrograde peristalsis observable under fluoroscopy may be present. Relief of the radiological signs of obstruction by moving the patient on to his side or on to his abdomen confirms the diagnosis. The left lateral or left oblique position is reported to be the most successful in facilitating passage of barium. This information is valuable in subsequent therapy.

Time of Onset

Vascular obstruction of the duodenum usually arises within the first few days after application of a cast or correction of a spinal curvature. However, of the 13 previously reported cases which occurred after cast application, more than a week elapsed before the first symptom appeared, in 4 instances. This is in sharp contrast with cases arising in debilitated patients with loss of body fat as a possible significant aetiological factor. In combat casualties the condition is seen only after 8-12 weeks. In severe burn injuries the syndrome developed early in
15 patients, the mean interval before diagnosis being 16 days after the burn. However, in 4 cases the interval was 63 days.

Treatment

When the syndrome is recognized early, treatment should be conservative with nasogastric suction, intravenous fluid and careful monitoring of fluid intake and output. If symptoms persist, immediate removal of the cast, traction or brace may be necessary. Cutting a window in the cast is not effective. Positioning the patient in the left lateral or prone position is often helpful. Non-operative treatment has proved successful in a large percentage of emaciated or burned patients or patients injured in combat. If these measures do not relieve the problem, laparotomy is indicated, with duodenojejunostomy being the procedure of choice. However, division of the ligament of Treitz, allowing mobilization of the duodenum, has proved successful in some cases.

Vascular compression of the duodenum is a recognizable complication of cast application that may appear late. Many cases of partial obstruction have hitherto probably gone undetected.

REFERENCES


Drug Kinetics in Protein Energy Malnutrition

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SUMMARY

Potential problems of drug metabolism in human protein energy malnutrition (PEM) are reviewed in the light of the pathophysiological changes seen in PEM. Data derived from human studies and animal experiments are discussed, and a plea is made for further research in this area.


People suffering from protein energy malnutrition (PEM) have physiological derangements which may exert important effects on the absorption and metabolism of drugs administered to them. Knowledge of altered pharmacokinetics in patients with PEM is necessary in view of the high incidence of this disorder among underdeveloped populations. The present article reviews some of the important problems which drug therapy of malnourished people may pose.

Magnitude of the Problem

No precise definition of PEM exists. The most appropriate definition is perhaps that provided by the World Health Organization: 'a range of pathological conditions arising from coincident lack, in varying proportions, of protein and calories, occurring most frequently in infants and young children and commonly associated with infections'.

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