

CASE REPORT

COMPRESSIVE SHOCK RESULTING FROM GASTRIC DISTENSION AFTER ARTERIAL SWITCH OPERATION: A CASE REPORT

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ABSTRACT

Compressive shock is an important cause of reversible cardiovascular compromise. Abdominal causes of compressive shock have been known to be difficult to diagnose. We report our experience in the management of compressive shock resulting from gastric distension. We consider gastric distension to be an unusual cause of compressive shock. Immediate relief of intra-abdominal tension is key in the management.

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INTRODUCTION

Shock is a state of acute circulatory failure leading to decreased organ perfusion, with inadequate delivery of oxygenated blood to tissues and resultant end-organ dysfunction. It may result from hypovolemic, distributive, cardiogenic or obstructive mechanisms¹. These mechanisms inform the classification of shock.

We present our challenges in the post-operative management of a newborn with transposition of great arteries managed by arterial switch operation that was complicated by compressive shock resulting from gastric distension.

CASE REPORT

Baby AS a first born male was born in a peripheral center at term via spontaneous vaginal delivery. Birth weight was 2.8 kilograms. Baby was noted to have cried at birth. Within 24 hours of birth, he was noted to have a bluish discoloration. Two-D echocardiography showed d-Transposition of great arteries hence patient was commenced on prostaglandin and referred to Apollo hospital, Delhi. Age of mother at birth was 26 years.

At presentation, baby was cyanosed, hypothermic, hypoxic and tachypneic. Echocardiography showed atrio-ventricular concordance and ventriculo-arterial discordance, patent foramen ovale with bidirectional shunt, muscular ventricular septal defect with left to right shunt. There was also a small patent ductus arteriosus with bidirectional shunt.

An emergency atrial septostomy was done for resuscitation, and patient was scheduled for arterial switch operation. Arterial switch operation was done on cardiopulmonary bypass with high aortic and bi-caval cannulation. Alpha stat technique was maintained.

Post operatively, patient was maintained on pressure controlled assisted mechanical ventilation and later on synchronized intermittent mandatory ventilation. Haemodynamic and oximetry parameters were normal

When feeding was started on second postoperative day, abdominal distension was noted, central venous pressure rose to 20cmH₂O, ventilator requirement increased. Blood pressure and urine output dropped. X-ray of chest and abdomen

showed a distended gastric shadow (figure

This episode was managed conservatively using diet restriction, nasogastric suctioning and intravenous fluids. Patient responded and parameters came to acceptable ranges.



Figure 1: X-ray of chest and abdomen showed a distended gastric shadow

Abdominal ultrasonography showed the pylorus with a single wall thickness of 0.33 cm, length of 1cm and the maximum pyloric lumen noted was 0.2cm.

Patient was commenced on atropine at a dose of 0.05 mg/kg/day in 8 divided doses administered through the nasogastric tube at 4 hourly intervals.

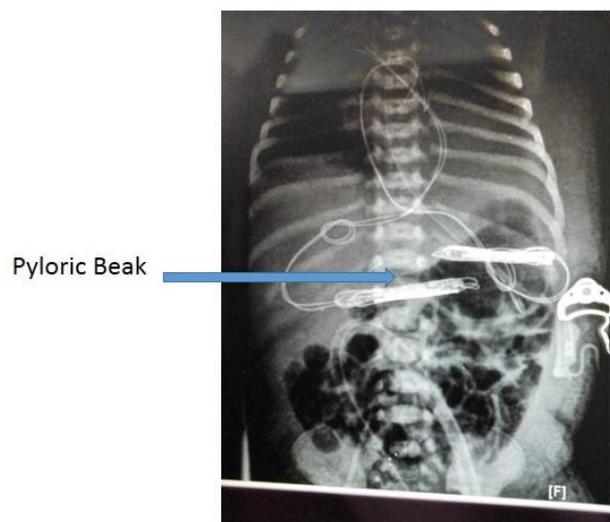
Feeding was introduced and gradually increased. Sonographic follow-up showed improvement in pyloric size and flow of gastric contents. Weight gain ensued.

DISCUSSION

Compartment syndrome is defined as an increased pressure in a closed anatomic space, which threatens the viability of enclosed and surrounding tissue. It may occur in the cranium, thorax, abdomen or limbs. The abdominal compartment has unique effects because it is geographically situated 'up-stream' from the extremities and 'down-stream' from the chest. Therefore, it may influence the physiology

1).

On recommencement of oral feeds, the compressive features returned and X-ray of chest and abdomen done at this time showed a beak at the pyloric antrum (figure 2).



and pathophysiology of each of these other compartments².

The clinical manifestations of abdominal compartment syndrome in a newborn following arterial switch operation may be mistaken to be compressive shock resulting from pericardial tamponade complicating the surgery.

Intra-abdominal hypertension (IAH) has been defined as sustained repeated pathological elevation of intra-abdominal pressure ≥ 12 mmHg, while abdominal compartment syndrome (ACS) is defined as sustained intra-abdominal pressure >20 (with or without abdominal perfusion pressure <60) that is associated with new organ dysfunction/failure.⁽³⁾ These definitions were ratified at the 2004 International ACS Consensus Definitions Conference.

Abdominal causes of obstructive shock have been reported in literature^(4,5). These cases were difficult to diagnose, with one of them made post mortem.⁽⁵⁾ Clinical

diagnosis of obstructive shock may be difficult in the absence of invasive pressure monitoring because elevated CVP with systemic arterial hypotension is characteristic.

It is difficult to make a case definition of abdominal compartment syndrome in our patient because we did not measure intra-abdominal pressure. However, the extent of physiological derangement defines shock.

The Pathophysiology is that the increased abdominal pressure consequent upon gastric distension causes a restriction of venous return via the inferior vena cava. Since the inferior vena cava accounts for two-thirds of venous return to the heart, cardiac output will ultimately be compromised hence the shock state. The drop in cardiac output can also be attributed to a diastolic dysfunction resulting from compressive forces on the heart. This explains the elevated CVP and poor tolerance of intra-venous fluid administration. The elevated intra-abdominal pressure will also splint the diaphragm accounting for the elevated ventilatory requirements.

Our patient had an acute gastric outlet obstruction resulting from restrictive pylorus. The factors that may account for the restrictive pylorus in our patient may include mucosal oedema resulting from generalized fluid retention following cardiopulmonary bypass or mucosal hypertrophy complicating prolonged Prostaglandin infusion. These factors may have acted in synergy and in concert with a borderline pyloric hypertrophy or spasm. The history of prolonged infusion of Prostaglandin E1 raises the suspicion of hypertrophic pyloric stenosis since an association has been reported in literature.⁽⁶⁾ This suspicion is also supported by the finding of pyloric beak on plain abdominal X-ray.

The deterioration to cardio-respiratory embarrassment was precipitous. This is as a result of mechanical ventilation which can account for gaseous distension of the stomach.

Prostaglandin E1 (PGE) is used in ductal-dependant congenital heart disease to maintain the patency of ductus arteriosus. Hypertrophic pyloric stenosis (HPS) resulting from gastric mucosal proliferation is a rare complication of prolonged PGE infusion.⁽⁶⁾ In our patient, the pyloric stenosis and the post-operative mechanical ventilation resulted in accumulation of stomach gas resulting in significant cardiorespiratory embarrassment. Strict criteria for ultrasound diagnosis of hypertrophic pyloric stenosis, pyloric length of 16mm and single wall thickness of 4 mm,⁽⁷⁾ was not met, but it is our believe that this is probably due to early intervention.

The clinical and radiological features of transient Hypertrophic Pyloric Stenosis following PGE infusion usually relieves after stopping PGE infusion.⁽⁶⁾ It should be kept in mind that Hypertrophic Pyloric stenosis due to PGE infusion can be transient and pyloromyotomy should be kept for patients with persistent findings. However, conservative management with Atropine may be justified especially in cases with cardio-respiratory embarrassment.

CONCLUSION

Severe intra-abdominal hypertension may result in cardio-respiratory compromise. Progression may be rapid in patients at risk. This may present with features of compressive shock. Diagnosis may be difficult. Immediate relieve of abdominal tension is the treatment.

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