Autonomic Hyper-reflexia in a Patient Undergoing Laparoscopic Cholecystectomy

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Introduction

Although the phenomenon of autonomic hyperreflexia (AHR) in patients undergoing bladder surgeries is well documented, there is very scarce literature regarding its presentation in other intra-abdominal surgeries. The survival of patients with spinal cord injuries (SCI) is increasing, these patients could present to the anesthesiologist for a wide variety of surgeries.

Our case presents an unique opportunity to discuss the implications of pneumo-peritoneum which is an essential aspect of most intra-abdominal surgeries. Laparoscopic surgeries typically needs gas insufflation at pressures of 15-20mmHg at 2-4litre/min. Insufflation pressures can cause a rise in intra abdominal pressures leading to high SVR and increased myocardial contractility. CO2 absorption in itself can cause rise in catecholamines which in turn induces hypertension and tachycardia. AHR although transient in nature could adversely affect these patients with poor myocardial and cardiovascular reserve leading to myocardial infarction, atrial fibrillation, LV failure, seizures and intra-cerebral hemorrhage.

Case

A 51 year old male with past medical history of traumatic paraplegia at the level of T4-T5 presented with 2-3 weeks of fever, chills and loss of appetite. He had absent sensation below T5 level and was unable to feel pain in his abdomen. His past surgical history also included bilateral traumatic fracture hip with osteomyelitis for which patient had undergone surgery with flap rotation 30 years ago. Patient’s ultrasound scan revealed cholelithiasis, thickened gall bladder wall of 4mm. CT abdomen was done which also showed gallstones. Patient was treated with IV antibiotics for 5 days during which time he underwent MRI scan of the hips to rule out osteomyelitis of the hip. A HIDA scan finally revealed obstructed cystic duct and decision was made to proceed with gall bladder removal. In the operating room patient was placed on standard ASA monitors and a right radial arterial line was inserted prior to induction of anesthesia. Infusion of sodium nitroprusside was connected to IV line for immediate use and syringes of NTG for IV push were prepared. We induced anesthesia with Versed 2mg, Fentanyl 150mcg, Lidocaine 100mg and Propofol 200mg. We deferred from using any muscle relaxant to avoid unwanted respiratory complications in the immediate post operative period. Infusion of remifentanil 0.25mcg/kg/min was started immediately after intubation. Oxygen and Sevofluorane anesthesia at MAC of 1.5-2.0 was titrated continuously to prevent any stimulatory blood pressure surges. Abdomen gas insufflation pressures were kept at 15mmHg. Surgery proceeded smoothly and gall bladder was extracted within 45min. At emergence patient’s blood pressure was seen to rise up to 220/120 with no associated bradycardia. BP
during emergence was controlled with sodium nitroprusside infusion and labetalol 10mg IV push which produced smooth extubation at BP of 150/70.

Case discussion

The typical presentation of cholelithiasis with pain abdomen in the right upper quadrant and radiation to the shoulder was absent and this warrants a high index of suspicion on behalf of the surgeon to reach a diagnosis.

Autonomic dysreflexia (AD) also described as autonomic Hyperreflexia (AHR) or mass reflex is characterized by a widespread reflex sympathetic discharge in patients with spinal cord lesions above the level of T6. It presents as a life threatening medical emergency with elevated blood pressures. AHR can be triggered by a variety of stimuli below the level of the lesion. These could be bladder and bowel distension, urinary tract infection, cystoscopy, detrusor-sphincter dyssynergia, scrotal compression, epididymitis, gastric ulcers, hemorrhoids, gastro-colic irritation, menstruation, pregnancy, labor and delivery, pressure ulcers, burns, insect bites, constrictive clothing or shoes, fractures, trauma, surgical or diagnostic procedures and pain.

The sympathetic afferent fibres enter the spinal cord and form reflex connections at the segmental level with autonomic efferent fibres. The afferent impulses ascend in the spino-thalamic and posterior columns to the brain stem, ventrolateral nucleus of the thalamus and the cerebral hemispheres. The efferent sympathetic neurons arise from the anterolateral spinal cord from T1 to L3 and synapse with the post-ganglionic neurons in the paravertebral chains of sympathetic ganglia.

AHR develops after the phase of spinal shock when autonomic activity returns. SCI leaves the sympathetic activity below the lesion functionally separated from the inhibitory effects of the supraspinal regulatory centres and therefore results in loss of sympathetic integration and sympathetic activity becomes reflexive in nature. The reflex becomes highly excitable and results in widespread reaction. There is peripheral adrenergic receptor super-sensitivity as well as a reorganization of spinal pathways controlling sympathetic pre-ganglionic neurons. There is sprouting of afferent component of the spinal reflex. Thus after a stimulus is evoked below the injury intact peripheral sensory nerves transmit impulses via the spinothalamic and posterior columns to stimulate sympathetic neurons in the intermediolateral gray matter of the spinal cord, there the impulses are not capable of crossing and cannot stimulate the inhibitory action of the supraspinal centres giving rise to generalised sympathetic hyperactivity below the lesion. Injury above T5 disrupts the descending input to the sympathetic pre-ganglionic neurons that control the splanchnic bed. This also results in an abnormal interplay between the parasympathetic and sympathetic nervous system.

It has been shown that increased arterial stiffness and higher resting heart rate are more prevalent in paraplegic patients than tetraplegics which places them a higher risk of early coronary artery disease (CAD). There is significant association between the number of daily AD events and markers of systolic function, structural indices, diastolic function, and left ventricular mechanics implying that a greater daily incidence of AD is associated with impaired cardiac function in humans with spinal cord injury. Studies have shown that AD induces a similar attenuation of $Î²$-AR responsiveness to isoproterenol as that which occurs in primary hypertension, suggesting a lack of contractile reserve. The increase in sympathetic firing during AD results in aberrant spikes of
circulating catecholamines, which ultimately lead to $\beta^2$-AR desensitization and impaired inotropic reserve acting as a predecessor of cardiomyopathy.

Studies have suggested that maintaining deep levels of anesthesia with typical MAC of 1.3-2.0 are necessary to avoid AHR. With deep levels of anesthesia these patients are prone to hypotension with a decrease in mean arterial pressures (MAP) below 70mmHg despite fluid therapy.

In contrast to the above there are findings suggestive that the maintenance anesthesia requirements in SCI patients are much less for a BIS of 40-50. It is suggested that blockade of ascending somatosensory transmission either by neuraxial blockade or spinal cord injury (SCI) increases susceptibility to anesthetics. Isoflurane’s action in the spinal cord indirectly inhibited brain cortical activity induced by electrical stimulation of the reticular formation.

Combinations of sevoflurane with other adjuncts (opioids or N2O) have been shown to be a better choice because they may enhance the actions of inhalation anesthetics in producing immobility or in preventing autonomic responses in the face of noxious stimulation. Studies have demonstrated that target-controlled concentrations of 1 ng/mL and 3 ng/mL remifentanil reduced the end-tidal concentrations of sevoflurane required to prevent AHR by 16% and 29% in the presence of 50% N2O, respectively.

In our case we also used the opioid remifentanil which acted in synergy with Sevoflarane for maintenance anesthesia. Further our strategy to use no muscle relaxation proved effective because intra-abdominal pressures were easily maintained due to muscle paralysis below the level of T5.

Studies have reported that chronic spinal cord transection reduces the sevoflurane concentration by 20% to 39% required to maintain BIS between 40 and 50 and blunted the sympathoadrenal and cortisol responses when the surgery was performed below the level of the lesion. Spinal and epidural anesthesia have shown to inhibit anterior pituitary responses including cortisol in patients who underwent pelvic or lower limb surgery. In the cord-injured patients the strongest stress response occurs during emergence from anesthesia.

Power spectral analysis is new method using analysis of biomedical signal variability to assess autonomic function. Heart rate (R-R interval) or arterial pressure variability is analysed using power spectral analysis. Some studies on heart rate variability (HRV) in detection of AHR during sacral root stimulation have been reported to be accurate.

SUMMARY

The perioperative period is very prone to somatic and visceral stimuli of surgical or other origins. Management of AHR includes removal of precipitating cause which could be constrictive tourniquet, urinary drainage catheter, distension from fecal impaction or intra-peritoneal stimulation. Pharmacologic means of reducing the blood pressures include of use rapid acting antihypertensives like sodium nitroprusside, nitroglycerine, hydralazine, nifedipine. There have also been case reports in the successful use of magnesium sulfate for patients in labor and delivery. Intraoperative maintenance of deep levels of volatile anesthesia in conjunction with opioids and use of antihypertensives at emergence are advocated. In conclusion SCI patients need strategies for active prevention and early intervention during the perioperative period and anesthesiologists role in its awareness is vital.