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Case Report Open Access

Autonomic Hyperreflexia (AHR) in a Patient Undergoing Laparoscopic Cholecystectomy: A Case Report

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Abstract

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 life expectancy of patients with traumatic paraplegia in the age group of 20-40 yrs is about 25-45 yrs.

The survival of patients with spinal cord injuries (SCI) is increasing, these patients could present to the anaesthesiologist for a wide variety of surgeries. 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. Our case presents a unique opportunity to discuss the implications of pneumo-peritoneum which is an essential aspect of most intra-abdominal surgeries.

Laparoscopic surgeries typically need gas insufflation at pressures of 15-20 mmHg at 2-4 litre/min. Insufflation pressures can cause a rise in intra-abdominal pressures leading to high SVR and increased myocardial contractility. CO2 absorption 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 haemorrhage.

Keywords: Autonomic hyperreflexia; Autonomic dysreflexia in laparoscopic surgery; Anesthesia implications in autonomic hyperreflexia

Case

A 51-years 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.

Patient's ultrasound and CT scan revealed cholelithiasis, finally as HIDA scan 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. Anesthesia induced with versed 2 mg, Fentanyl 150 mcg, Lidocaine 100 mg and Propofol 200 mg.

We deferred from using any muscle relaxant to avoid unwanted respiratory complications in the immediate post-operative period. Infusion of remifentanil 0.25 mcg/kg/min was started immediately after intubation.

Oxygen and Sevoflurane 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 15 mmHg. Surgery proceeded smoothly and gall bladder was extracted within 45 mins.

At emergence patient's blood pressure was seen to rise to 220/120 with no associated bradycardia. BP during emergence was controlled with sodium nitroprusside infusion and labetalol l 10 mg IV push which produced smooth extubating at BP of 150/70.

Case Discussion

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, surgical or diagnostic procedures and pain.

The sympathetic afferent fibers enter the spinal cord and form reflex connections at the segmental level with autonomic efferent fibers. 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 ch chains of sympathetic ganglia (Figure 1).

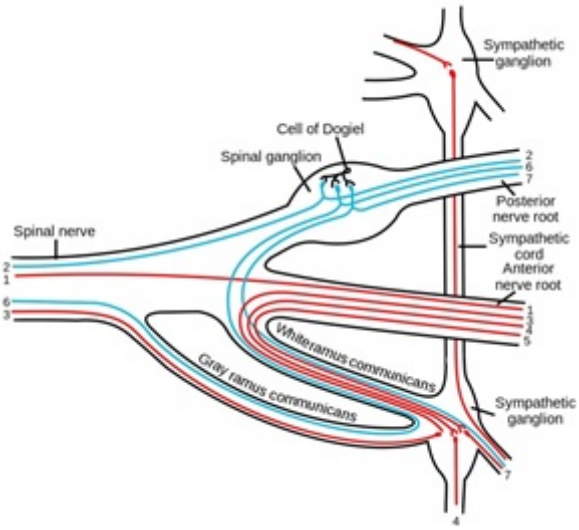

Figure 1: Sympathetic nervous system anatomy at the spinal cord level. (1) Somatic efferent; (2) Somatic afferent; (3-5) Sympathetic efferent; 6 and 7 Sympathetic afferent.

This image is from the 20th US edition of Gray's Anatomy of the Human Body and is in the public domain.

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 supra spinal 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 grey 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 generalized sympathetic hyperactivity below the lesion.

Injury above T5 disrupts the descending input to the sympathetic preganglionic neurons that control the splanchnic bed (Figure 2). This also results in an abnormal interplay between the parasympathetic and sympathetic nervous system [1,2].

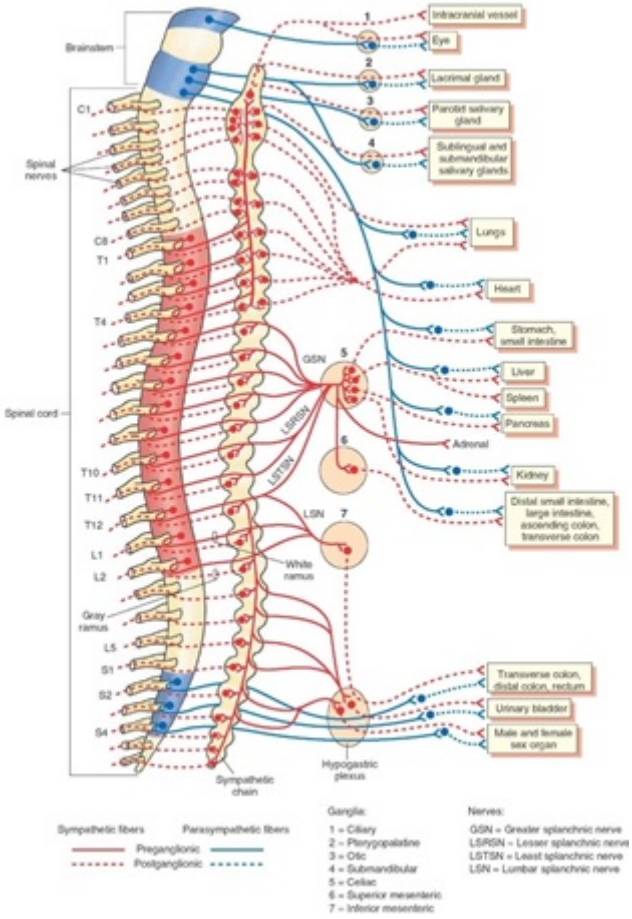

Figure 2: ANS anatomy-Parasympathetic pathways represented by blue and the sympathetic pathways in red and the interrupted red lines indicate post-ganglionic rami to the cranial and spinal nerves.

This image is from the 20th US edition of Gray's Anatomy of the Human Body and is in the public domain.

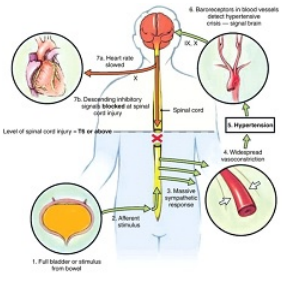

Figure 3: This image is from Faust's anaesthesiology 4E shown the increased arterial stiffness and higher resting rate.

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integration and sympathetic activity becomes reflexive in nature (Figure 3). 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 grey 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 generalized sympathetic hyperactivity below the lesion. Injury above T5 disrupts the descending input to the sympathetic preganglionic neurons that control the splanchnic bed. This also results in an abnormal interplay between the parasympathetic and sympathetic nervous system [3].

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) [4]. 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 [2]. Studies have shown that AD induces a similar attenuation of β-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 β-AR desensitization and impaired inotropic reserve acting as a predecessor of cardiomyopathy [5].

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

In contrast to the above there are findings suggestive that the maintenance anaesthesia requirements in SCI patients are much less for 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 anaesthetics. Isoflurane's action in the spinal cord indirectly inhibited brain cortical activity induced by electrical stimulation of the reticular formation [6].

Combinations of sevoflurane with other adjuncts (opioids or N_2O) have been shown to be a better choice because they may enhance the actions of inhalation anaesthetics 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 [7].

In our case we also used the opioid remifentanil which acted in synergy with Sevoflurane for maintenance anaesthesia. 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%-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 [8]. Spinal and epidural anaesthesia have shown to inhibit anterior pituitary responses including cortisol in patients who underwent pelvic or lower limb surgery. Huang et al. [9] also found that the hypothalamus-pituitary adrenal axis was impaired in patients with spinal cord injuries. Plasma cortisol concentrations have been shown to be increased significantly over baseline values at 1 hour after the end of surgery in the cord injured patients which proved that the strongest stress response occurs during emergence from anaesthesia.

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. Power spectral analysis consists of breaking down variability into its component sinusoidal waves by means of fast Fourier transformation. Information derived from applying Fourier transformation on biomedical signal variability is indirectly used to assess ANS activity. Some studies on heart rate variability (HRV) in detection of AHR during sacral root stimulation have been reported to be accurate. This could be a useful screening tool and monitor due to its non-invasive character [10].

Summary

The perioperative period is very prone to somatic and visceral stimuli of surgical origins. The major triggers for AHR were found to be bladder spasm, gastric or colonic distension, pelvic compression, urinary retention with overfull bladder and insertion of bladder catheter. Management of AHR includes sitting or reverse Trendelenburg which pools the blood in lower extremities and decreases the preload, removal of precipitating cause which could be constrictive tourniquet or urinary drainage catheter, distension from faecal impaction or intra-peritoneal stimulation. Pharmacologic means of reducing the blood pressures include of use of rapid acting anti-hypertensive like sodium nitroprusside, NTG, hydralazine, labetalol (in the absence of bradycardia), nifedipine [11]. There have also been case reports in the successful use of magnesium sulphate for patients in labour and delivery [12]. Intraoperative maintenance of deep levels of volatile anesthesia in conjunction with opioids has been proven to be useful. Use of antihypertensive at emergence could also be advocated because findings have shown that it markers of stress like cortisol are seen to rise during this period. HRV as a screening tool in monitoring these patients in ambulatory settings could be useful for patients planning elective surgeries. In conclusion SCI patients need careful monitoring for active prevention and early management of AHR during the perioperative period and anesthesiologist's role in its awareness is vital.

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