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PNEUMOPERITONEUM INDUCED BRADYCARDIA: EVALUATION OF ITS CAUSES, MANAGEMENT, PROPHYLAXIS AND ITS IMPLICATIONS

Surgery

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KEYWORDS

INTRODUCTION

Laparoscopy has become the standard of care for many procedures such as cholecystectomy, appendectomy, hernia repair and for the complex surgeries like colonic resection, nephrectomy, and the upcoming bariatric surgeries. It is becoming the standard of care owing to quicker post-operative recovery and less morbidity in the post operative period. It is now the gold standard procedure in management of symptomatic gall stone disease and achalasia cardia. All laparoscopic procedures require creation of pneumo-peritoneum, which is essential to separate the abdominal wall from the viscera, which improves visualization to the surgeon. Artificial pneumo peritoneum is created using carbon dioxide (CO_2) insufflation to the maximum pressure of 10-15 mmHg, maintaining the flow rate at 4-6 litres/min. This is maintained with the help of a constant gas flow of 200-400 ml/min. Additional benefit of carbon dioxide usage is that diathermy can be used, as it does not support combustion. However, this comes with its own set of complications. Particularly, the profound cardiovascular changes induced by the positive pressure pneumo peritoneum. Pneumo peritoneum is reportedly a predisposing factor for autonomic imbalance, resulting in a possible risk of cardiac arrhythmia. An ominous concern which has received less attention is bradycardia or worse, cardiac arrest. Systematic knowledge regarding this intrao perative bradycardia and its significance is lacking. Through the this article we tried to make assimilate the available information regarding the factors contributing, causes, early prediction, prophylaxis, management and implications of pneumo peritoneum induced bradycardia in order to have an insight into this important but under reported phenomenon.

1.METHODS

A thorough literature search was done on Medline, Science Citation Index, Current Contents, Embase, and PubMed databases for publications from 1985 to March 2019 in English language. Several case reports or retrospective cohort series were identified.

2.RESULT AND DISCUSSION

3.1 Cardiovascular changes due to pneumoperitoneum:

Pneumo peritoneum causes profound cardiovascular changes, which are intriguing because patho-physiologically, they resemble chronic heart failure. This similarity is confirmed by the model of chronic heart failure in animals, known as the inferior vena caval constriction model. The model simulates a similar condition to positive pressure pneumo peritoneum in laparoscopy, by artificially creating mechanical obstruction of inferior vena cava.[1] The studies reveal that there is a certain amount myocardial ischemia, but no apparent increase in the cardiovascular mortality due to laparoscopic procedures. In one study, 39% of patients had intra operative myocardial ischaemia when monitored, but the clinical observation of the anaesthetist was able to identify only 2% of myocardial ischemia.[2] However, there is very little information about this myocardial ischemia. One study showed that two of sixteen patients who were free from cardiac disease and underwent laparoscopic cholecystectomy had acute ST changes on the electrocardiogram.[3] Apparently, these were seen in low risk patients. Clearly there is a correlation with underlying baseline myocardial function and overall health status of the individual.

The main haemodynamic changes induced by CO2 pneumo peritoneum include a increase in systemic vascular resistance by 65%, an increase in pulmonary vascular resistance by 90%, and decrease in cardiac index by 20-59%.[4] Thus there is increase in after load and a decrease in cardiac output. These changes resemble chronic heart failure, but of course the inciting events are vastly different. However, pneumo peritoneum is not the only influence on the cardiovascular system in this situation. There are other factors in play such as posture and mechanical distention. Head-down tilt with pneumo-peritoneum increases the preload and pulmonary wedge pressure but normalises the after load. On the other hand, head-up tilt with pneumo peritoneum normalises the preload, but furthers the after load increase.[5] Mechanical distension of the abdomen stimulates the vagus, produces bradycardia or atrioventricular block such that some advocate the routine use of anticholinergic agents. Thus, there is a crucial interplay of numerous factors without a clear consensus, but what is clear is pneumo peritoneum has a substantial effect on the cardiovascular system.

3.2 Bradycardia due to pneumo peritoneum:

Bradycardia is defined as heart rate of less than 50 or 60 beats/min, without remark regarding the duration of the event. The reported incidence during laparoscopy is highly variable depending upon the study, but it ranges between 15–56%.[8,14,15] Pneumo peritoneum is known to cause bradycardia and even asystole, which may originate from increased abdominal pressure and CO_2 retention.[6] A recent analysis found that occurrence of bradycardia during laparoscopy is a early sign of cardiac rhythm abnormalities and subsequent arrest.[7] The maximum amount of cases happened in apparently “healthy” patients and were directly associated with pneumo peritoneum.

A retrospective cohort study done at Tel Aviv medical centre shed light on this phenomenon.[9] They reported that bradycardia was more prevalent in males, older patients and smokers, as well as patients with cardiovascular and renal co-morbidities and high ASA score patients. It was less commonly seen in patients with diabetes mellitus and those who treated with beta, alpha and calcium channel blockers. The strongest risk factor for bradycardia among drugs was treatment with beta-blockers. The triggers for bradycardia varied from flawed documentation of CO2 insufflation, opioid administration, deflation of pneumo-peritoneum to injection of phenylephrine and beta-blockers. However, most bradycardia events were related to CO2 insufflation and traction on pelvic structures and spontaneously resolved without the need for pharmacological treatment.[8]

3.3 Factors influencing bradycardia:

There are various factors influencing this phenomenon. The factors can be broadly divided into two categories, patient and instrumental factors. The patient related factors include, age, beta blocker use, preoperative haemoglobin, gender, ASA and dys lipidaemia. The only risk factor with a statistical significance was age. The incidence is higher among young age group. The instrumental factors include flow rate, peak Intra-Abdominal Pressure (IAP), posture during the surgery. The ones studied in detail are discussed below:

2.1.1.Flow Rate

The development of bradycardia has been shown to occur due to high flow rate of CO_2 insufflation during laparoscopic gynaecological

procedures.[10] The pressure limit was 12mm of Hg but the flow rate was 20L/min or 50L/min. Gas embolism was ruled out as $ETCO_2$ and SpO_2 were within normal limits. The next logical question is how the abdominal stretching can occur even if the upper limit of pressure is normal. This can be explained by the insufflator performance during laparoscopy.[11] Insufflator works effectively when a balance is established between pressure, resistance and flow of the equipment. Thus, gas flow from insufflator to the abdomen of the patient follows the Hagen-Poiseuille's law, theoretically[11]:

$$V = \frac{\pi \times \Delta P \times r^4}{8 \times \eta \times l}$$

where V = gas flow rate, π = Pi, r = radius, 8 = constant, η = viscosity, l = length, and ΔP = pressure difference.

The Hagen-Poiseuille's law states, gas flow rate is directly proportional change in pressure, and critically dependent on the smallest radius or diameter of equipment[11,12] Thus, when gas will pass through a high resistance channel, such as the luer lock connector, pressure will rise to maintain a high flow rate. Consequently, to keep up the velocity the IAP will rise rapidly as if the insufflator is regulated to keep a high flow rate (about 20 L/min) from the starting. Also, insufflators which support high flow rate usually have over-pressure insufflation principle. This means that the pressure put at the beginning of the insufflation is much greater than pre-set value. This pressure is then decreased during further insufflation intermittently, until IAP reaches pre-set value of 12 mmHg.[10] Therefore, even if the pre-set pressure is capped at 12 mmHg, peak value of IAP will surpass the capped pressure in an instant to keep up the fixed flow rate. Whereas a gradual abdominal insufflation with a flow rate of 1 L/min and peak IAP of 12mm Hg, was associated with no cardiovascular abnormality even in elderly ASA III patients.[13]

2.1.2.Upper Limit of IAP

If the intra-abdominal pressure (IAP) is maintained below 12-15 mmHg during insufflation, there are minimal pathophysiological changes because of pneumoperitoneum.[6] The various changes in the cardiovascular parameters with increasing IAP is shown in the illustration below[16]:

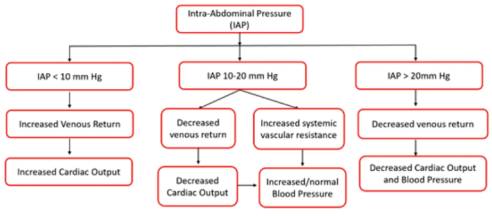

Fig 1: Various changes in the cardiovascular parameters with increasing IAP

2.1.3.Posture of the patient during the procedure

The common postures are head up or head down. A study which evaluated the hemodynamic changes associated with head down position and prolonged pneumoperitoneum during totally endoscopic robot-assisted radical prostatectomy, revealed that the heart rate in all patients was very low and there was no variation of heart rate during positioning to head down or prolonged pneumoperitoneum. However, there was a significant increase in the base line heart rate during deflation.[17]

3.4 Cardiac Arrest due to pneumoperitoneum

Asystole is a known complication of pneumoperitoneum. A case series by **Gautam et al** from elucidated four such cases of non-fatal intra-operative cardiac arrests during laparoscopic cholecystectomy. The bradycardia was precipitated even at low flow rate of 1L/min and IAP below 15 mmHg.[18] There was no baseline cardiac dysfunction or any other co-morbidity. The age group varied between 28 to 68 years. Various factors have been identified for causing cardiac arrest during laparoscopy. Most discernible is the vagal stimulation due to peritoneal stretch, Halothane induced myocardial sensitisation, Diminished venous return owing to the position of the patient, external pressure inferior vena-cava, acute loss of blood and effect of anaesthetic medications.[19,20] One of the four patients was diabetic. Diabetes is associated with less incidence of bradycardia[9] but there is profound autonomic dysfunction probably leading to arrest. They are at increased cardiovascular lability during anesthesia.[21] An objective measure to indicate cardiac autonomic dysfunction in diabetics is Dipyridamole myocardial single photon emission computed tomography (SPECT) scan. A peak to basal Heart rate ratio of less than 1.2 indicates dysfinction.[22] Vagus-mediated reflex bradycardia and sinus arrest has been observed due to a variety of surgical stimuli[23] including laparoscopy.[24,25]

3.5 Prediction, Management and Prevention of Bradycardia

There is a 4.7% overall risk for the development of bradycardia (with or without progression to asystole) in simple laparoscopic cholecystectomies while creating pneumoperitoneum.[26] The most perplexing fact about this phenomenon is that the cardiovascular changes are never expected and are hard to predict. The patients do not display any special sign, symptom or biochemical abnormality which can help us anticipate such an event or do any kind of special monitoring. There are no underlying common patient denominators that might explain the bradycardia. Thus the anaesthetist and the surgeon should be well aware of the management strategies which include: 1) decreasing the CO2 pressure in the; 2) halting the anaesthetic drug ; 3) shifting patient to supine position, with or without 4) atropine.[27]

It is said that prior planning prevents poor performance, thus if the occurrence of bradycardia cannot be predicted and management may not be prompt enough to salvage the patients in time, we should focus on prophylaxis. Two studies are of interest here, the first study, which was done on adults undergoing laparoscopic urological surgeries for prophylactic effect of intravenous atropine sulfate on cardiac arrhythmias (sinus bradycardia) during anesthesia showed that bradycardia significantly reduced.[14] In the other study, by Annila and colleagues that evaluated intravenous atropine sulfate and glycopyrrolate on cardiac arrhythmias for adenoidectomy in children, bradycardia was more common in the placebo group than in the atropine group.[28]

3.6 Implications of this phenomenon

Bradycardia is a common phenomenon during laparoscopy, and also a harbinger of doom, in the form of a clinical marker which heralds a potential cardiac arrest. Predicting which bradycardic episodes will go on to cardiac arrest currently remains unclear.

The basic root cause of bradycardia is disturbed autonomic balance. There are attempts to measure the autonomic function of the individual to predict the response to pneumoperitoneum. One such study used, corrected QT (QTc) interval to predict the deadly cardiac arrythmias.[29,30] Few studies showed that pneumoperitoneum with the head-up position did not increase the QTc interval.[29,31,32] Egawa et al. observed pneumoperitoneum induced QTc interval prolongation in patients older than 65 years. There is increased QTc interval after the application of pneumoperitoneum in young adults as well. The risk of torsades de pointes is increased in patients with increased QTc interval (>440 ms). The risk was also increased in patients with relative prolongation of the QTc interval from the normal value.[33,34] This QTc interval prolongation occurred at 90 minutes of pneumoperitoneum application with an intra-abdominal pressure of 12 mmHg.

3.CONCLUSION:

Insufflation with high flow rate can increase IAP momentarily to cause unexpected cardiovascular changes, such as hypotension, bradycardia and even cardiac arrest may occur. Therefore, it is of utmost importance to maintain IAP below 12-15 mmHg as well as keeping slow insufflation rate. There is multi-factorial origin of bradycardia and cardiac arrest, it is associated with the nature of the surgical technique, preoperative condition of patient , inadequate risk assessment, and even intra-operative errors. The surgeon as well as the anaesthetist needs to be aware of the possible complications during laparoscopy.

Therefore, peri-insufflation bradycardia constitutes a 'red flag' event that should be a significant warning for surgeons and anaesthetists.

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