Cardiovascular manifestations of perioperative acute urinary bladder over-distension

Sir,

Bladder over-distension may lead to undesirable autonomic manifestations such as vomiting, bradycardia, hypotension, hypertension, cardiac dysrhythmias, or even asystole.[1] We report two clinically diverse cardiovascular presentations of acute bladder over-distension.

Case 1: A 64-year-old male, with no associated comorbidities, was scheduled for transurethral resection of the prostate (TURP). Spinal anaesthesia was administered (2 ml heavy bupivacaine 0.5% plus fentanyl 10 μg). T8 level of sensory blockade was achieved. The intraoperative period was uneventful. In the recovery room, the patient was stable, heart rate (HR) 84/min, blood pressure (BP) 126/80 mmHg, SpO_2 99% and sensory block at T8 level. One hour later, the patient became restless, anxious, and diaphoretic. HR was 44/min, BP 90/60 mmHg, SpO_2 98%. Atropine 0.6 mg intravenous (IV) was administered. Abdominal examination revealed a grossly distended bladder (up to umbilicus). The tri-way urinary catheter lumen draining the bladder was blocked (blood clots) while bladder irrigation with saline continued, resulting in acute bladder over-distension. Bladder irrigation was discontinued. Patency of the draining catheter was established, and 650 ml of urine/saline was drained. Level of block had regressed to T10. The patient experienced relief and became haemodynamically stable.

Case 2: A 45-year-old, female was scheduled for staging laparotomy for ovarian malignancy under general anaesthesia. Pre-anaesthetic evaluation was unremarkable. Standard anaesthetic technique was used. Intra-operatively, patient's vitals remained stable and but her urine output was only 30 ml at 1.5 h. Fluid administered was adequate, and the bladder was not distended. A fluid challenge (500 ml Ringer's lactate) was administered followed by furosemide (10 mg) IV. Otherwise, patient had received crystalloid (3000 ml), colloid (500 ml) and blood (one unit). Patient had lost one litre of blood intraoperatively. Surgery lasted 3.5 h. The patient remained haemodynamically stable (HR: 62-76/min, BP: 110/76-126/82 mmHg).

While abdomen closure was in progress, HR increased from 62/min to 157/min, BP 158/114 mmHg, irregular pulse and multiple atrial ectopics. Per-abdomen examination revealed an over distended bladder due to blocked catheter. Following a saline flush, 500 ml urine was drained. Within seconds of bladder decompression, HR decreased from 150 to 60/min and BP was 117/65 mmHg. The patient made an uneventful recovery.

Urinary retention(UR) is common after anaesthesia and surgery with a reported incidence between 5% and 70%.[2] The risk factors for UR include anaesthetic (excessive fluid administration, opioids, anticholinergics, neuraxial blockade); surgical (hernia repair, rectal and urologic surgery, duration of surgery >2 h) and patient factors (previous history of UR, underlying neurologic dysfunction, age >60 years, pre-existing contracted bladder).[3]

Clot retention (catheter blockage) manifests itself with suprapubic distension, severe lower abdominal discomfort, decreased or no exit of fluid and leakage of fluid/urine around the catheter. Symptoms of pain and lower abdominal discomfort may be masked by regional anaesthesia where bladder filling perception is abolished,[2] as in case 1. However, decreased fluid exit and presence of leakage around the catheter should alert to presence of catheter obstruction in a post-TURP patient. Close monitoring for colour and consistency of bladder returns, patency of inflow/outflow tubing and rate of irrigation can prevent postoperative bladder distension.

In case 2, furosemide was administered as urine output was less than optimal despite adequate fluid replacement, no response to a fluid challenge and after confirmation of a non-distended bladder by the surgeon during laparotomy. A blocked catheter resulted in bladder over-distension and consequent haemodynamic manifestations. Prior to administration of a diuretic, it is imperative that the patency of the urinary catheter (kinking/obstruction) and its correct position in the bladder be ascertained.

Normal adult bladder capacity ranges between 400 and 600 ml.[3] The first need to void is experienced at a bladder volume of 150 ml and the urge to void at 300 ml.[3] Dullness of the bladder to the level of the umbilicus provides a rough estimate of at least 550 ml of urine, but it can vary as much as 1000 ml with dullness extending above the umbilicus.[4] Spontaneous voiding of urine does not occur until after segmental sensory analgesia has regressed to S3.

Urinary bladder distension can cause cardiac dysrhythmias, most likely due to autonomic disturbances.[5,6] Eggers and Baker[5] reported multifocal ventricular tachycardia attributed to a neurogenic reflex due to bladder over-distension. Yamaguchi *et al*.[6] described the appearance of bigeminy on distension of the bladder in a conscious 47 years old diabetic. The parasympathetic response to acute over-distension is possibly due to a vaso-vagal reflex (afferent impulse enters the spinal cord through the pelvic nerves, ascends via sacro-bulbar connection close to the vagal nuclei and the resultant vaso-vagal reflex).[7] A vesicovascular response to urinary bladder distension is mediated by sympathetic vasoconstrictor neurons that contribute to increased sympathetic outflow.[8]

Acute bladder over-distension is an important, but often unrecognised medical complication. Potentially harmful UR should be suspected in the presence of severe pain, restlessness, confusion, chills, bradycardia, hypotension or hypertension, cardiac dysrhythmias or vomiting.Failure to recognise acute over-distension of the bladder can lead to serious cardiovascular morbidity.

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