Cardiac arrest despite optimal preloading of patient using ultrasonography-guided inferior vena cava indices under subarachnoid neuraxial blockade: A report of two cases

ABSTRACT
Spinal anesthesia (SA) is utilized as an effective means of anesthesia and has an impressive safety record but it is not devoid of complications, and sometimes, the complications are as fatal as cardiac arrest. Although many factors are involved in etiology of cardiac arrest under SA, the vagal responses to the decreased preload are the most common culprits. We report two cases of cardiac arrest under SA; which happened despite our patient being adequately preloaded utilizing the ultrasonography-guided targeted volume therapy. The patients were successfully resuscitated with no neurological deficit.

Key words: Cardiac arrest; spinal anesthesia; hypotension; vasopressors; noradrenaline' vagal tone

Introduction
In 1898, August Bier was the first to publish a report on successful spinal anesthesia (SA). SA is an effective means of anesthesia for lower limb and abdominal surgeries, but complications do occur. Hemodynamic complications such as hypotension and bradycardia are commonly seen as well as rare instances of cardiac arrest during SA are also reported.

There are many factors which are involved in etiology of cardiac arrests under SA; but, predominantly, the vagal responses to the decreased preload are the major offenders. We report two cases of cardiac arrest under SA despite patients being adequately preloaded using ultrasonography (USG)-guided volume targeted therapy based on the inferior vena cava (IVC) diameter and collapsibility index (IVC).

Case Report
Case 1
A, 59-year-old female of 55 kg, a known case of right-sided ovarian cyst, presented for elective total abdominal hysterectomy. The patient was accepted in American Society of Anesthesiologist (ASA) Grade I under combined spinal-epidural anesthesia technique. Baseline vital parameters recorded in operation theater were within the normal limits.

A transabdominal USG was performed on the patient to assess her volume status. The baseline IVC diameter was 9 mm and IVC-CI (Dmax-Dmin/Dmax × 100) was 73.4%. The IVC diameter in optiminally preloaded patient should be > 10 mm and IVC

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Bhatnagar V, Dwivedi D, Chakraborty S, Ray A. Cardiac arrest despite optimal preloading of patient using ultrasonography-guided inferior vena cava indices under subarachnoid neuraxial blockade: A report of two cases. Saudi J Anaesth 2018;12:478-81.
Grade II. Vitals were within normal limits. Preoperatively, a transabdominal USG was performed. The basal IVC diameter was 8.35 mm and IVC-CI was 62.8%. A preload of 500 ml RL was administered. A repeat measurement of the IVC indices showed that IVC diameter had increased to 10.3 mm and IVC-CI was 38.6%. Using 26G Quincke needle, 2.6 ml of hyperbaric bupivacaine was administered intrathecally in L3–L4 interspace in left lateral position. Level of sensory block achieved was at T8 which was confirmed by the pinprick method. Oxymask™ was applied and oxygen at the rate of 4L/min was started with continuous ETCO2 monitoring.

Around 25 min post-SA when the ovarian cyst weighing approximately 2.5 kg was being handled and removed, the systolic blood pressure became 70 mm Hg, patient stopped responding, and developed bradycardia (HR 28/min) resulting in asystole within few seconds. Pulses were not palpable. Immediately, cardiopulmonary resuscitation (CPR) was initiated and endotracheal intubation was performed; ventilation started with 100% oxygen. Postintubation and chest compression end-tidal CO2 was seen rising to 40 mmHg (monitored with the sidestream capnometer), and return of spontaneous circulation (ROSC) was noted within 2 min. Noradrenaline infusion (0.05 μg/kg/min) was initiated. Subsequently, vitals became stable. Postresuscitation fluid status of the patient was assessed. The IVC diameter was >10 mm, and the IVC distensibility index (Dmax-Dmin/ Dmin × 100) was <18% which was normal. Urine output was monitored. Arterial blood gas (ABG) was normal. Surgery proceeded, and postoperatively, the patient was mechanically ventilated in ICU. Echocardiography revealed 60% ejection fraction (EF) with no regional wall motion abnormality (RWMA).

The patient became hemodynamically stable, responded well, and was successfully extubated 6h postsurgery. Noradrenaline infusion was tapered over 24 h in postoperative period. There was no neurological deficit.

Case 2
A 67-year-old male, weighing 68 kg, was posted for L4-L5 lumbar laminectomy under SA and was accepted in ASA CI <50% [Figures 1 and 2]. A preload of 500 ml ringer lactate solution (RL) was administered. Postpreloading, IVC diameter was 12 mm and the IVC CI reduced to 33.7%. The patient was deemed as optimally preloaded. An 18G epidural catheter was placed followed by SA with 2.6 ml of bupivacaine (heavy) in L3–L4 interspace. Sensory level of the block was checked with the help of needle prick method and the level of sensory block was at T8. Oxymask™ was applied and oxygen at the rate of 4L/min was started with continuous ETCO2 monitoring.

The patient was turned prone after 20 min for the surgery on the spine when he became unresponsive and pulses became nonpalpable. The patient was immediately turned supine and CPR was started. The patient was intubated and ventilated with 100% oxygen. There was ROSC within 2 min (EtCO2 increased to 34 mmHg) which was measured with sidestream capnometer. A transabdominal USG was performed and the IVC diameter was found to be 7.78 mm and IVC distensibility index was 52.5%. Fluid resuscitation with 1200 ml of crystalloid was performed. The IVC indices were measured periodically. Once the IVC diameter was >10 mm and IVC distensibility index was <18%, the fluid resuscitation was stopped. Vital parameters became stable with HR – 88/min, SpO2 – 99%, and noninvasive blood pressure – 90/52 mmHg, respectively. Pupils were normally reacting to light. Echocardiography suggested 56% EF with no RWMA. The patient was extubated after an hour. A 12-lead electrocardiogram and ABG analysis were normal. Surgery was postponed for a later date and the patient was shifted to ICU for monitoring.

Discussion

After more than a century since SA being introduced by August Bier, the technique is still an effective and well-accepted means of anesthesia. Common complications

Figure 1: Ultrasonographic Doppler film showing subcostal view of inferior vena cava in both inspiration (1a) and expiration (1b). Inferior vena cava collapsibility index calculated was 46.25% during fluid preloading

Figure 2: Ultrasonographic film showing subcostal view of inferior vena cava in both inspiration (2a) and expiration (2b). Inferior vena cava collapsibility index calculated was 15.44% which denotes adequate fluid status
include hypotension, bradycardia, urinary retention, nausea, and vomiting. Certain rare but fatal complications include paraplegia, transverse myelitis, chronic arachnoiditis, and meningitis. Cardiac arrest under SA is rare with the incidence of around 6.4 ± 1.2/10,000 SA. Pollard identified vagal responses to the decreased preload as the primary etiology and specified that patients with high vagal tone are particularly at risk.

Various noninvasive tools such as USG, echocardiography, and Doppler-derived parameters have been used in guiding fluid therapy during resuscitation in the critically ill patients and the patients presenting at the emergency unit. These include both the static and dynamic parameters such as IVC diameter, IVC collapsibility/distensibility index, left ventricle end-diastolic area, left ventricular outflow tract velocity time integral (LVOT VTI), and LVOT VTI variation with passive leg raising. In our first case, assessment of volume status and preloading was done under USG guidance. However, few studies exist which define their role in predicting fluid responsiveness under SA thereby limiting the hypotension. In our first case, assessment of volume status and preloading was done under USG guidance. However, bradycardia followed by cardiac arrest occurred intraoperatively under subarachnoid block, which could be due to unopposed vagal response and handling of huge ovarian cyst which might have resulted in IVC occlusion leading to relative hypovolemia. Cardiac arrest occurred during positioning from supine to prone in our second case. This happened due to the ascending level of the block before the drug could have got fixed or may be due to redistribution of venous filling in prone position again leading to relative hypovolemia.

Literature shows that there is variability in fixing of the neuraxial administered drugs, and risks of cardiorespiratory complications are higher in prone position following SA block. In a review by Limongi and Lins, hypovolemia and position change during surgery have also been shown to be a risk factor for cardiac arrest in addition to the sympathetic blockade.

In tangent with our case series where the USG-guided fluid preloading was done based on the IVC indices under SA, the ongoing (ProCRHYSA randomized trial) is also aimed to study the similar outcome of limiting the hypotension postspinal anesthesia.

Hypotension prevention with the judicious use of USG-guided fluid therapy before SA could be affected by various confounding factors. It includes the patient factors, for example, age, positioning, presence of comorbidities such as diabetes, medications (β blockers and ACE inhibitors), surgical factors, and the intrinsic vagal tone, which all can affect the hemodynamics independently in patients undergoing SA.

To conclude, etiology of cardiac arrest post-SA block is multifactorial. The high index of suspicion, diligent monitoring, careful positioning, correct choice of anesthesia technique (general anesthesia in place of SA), especially in high-risk cases where the surgery involves major fluid shifts with tissue handling leading to traction and parasympathetic response, these all could prevent the adverse cardiac events in the perioperative period. Inclusion of noninvasive methods to assess the volume responsiveness and fluid repletion before SA block could limit but could not prevent completely the adverse hemodynamic events that will require larger studies and trials like ProCRHYSA randomized trial to validate its role in preventing morbidity and mortality.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

References
1. Auroy Y, Narchi P, Messiah A, Litt L, Rouvier B, Samii K, et al. Serious complications related to regional anesthesia: Results of a prospective survey in France. Anesthesiology 1997;87:479-86.
2. Pollard JB. Cardiac arrest during spinal anesthesia: Common mechanisms and strategies for prevention. Anesth Analg 2001;92:252-6.
3. Mackenzie DC, Noble VE. Assessing volume status and fluid responsiveness in the emergency department. Clin Exp Emerg Med 2014;1:67-77.
4. Corl KA, George NR, Romanoff J, Levinson AT, Chheng DB, Merchant RC, et al. Inferior vena cava collapsibility detects fluid responsiveness among spontaneously breathing critically-ill patients. J Crit Care 2017;41:130-7.
5. Donati A, Mercuri G, Iuorio S, Sinkovetz L, Scarcella M, Trabucchi C, et al. Haemodynamic modifications after unilateral subarachnoid anaesthesia evaluated with transthoracic echocardiography. Minerva Anestesiolog 2005;71:75-81.

6. Kundra P, Anunsekar G, Vasudevan A, Vinayagam S, Habeebullah S, Ramesh A, et al. Effect of postural changes on inferior vena cava dimensions and its influence on haemodynamics during caesarean section under spinal anaesthesia. J Obstet Gynaecol 2015;35:667-71.

7. De Vivo S, Ceruti S. Non-invasive methods to predict hypotension after spinal anesthesia. Austin J Emerg Crit Care Med 2015;2:1021.

8. Veering BT, Immink-Speet TT, Burm AG, Stienstra R, van Kleef JW. Spinal anaesthesia with 0.5% hyperbaric bupivacaine in elderly patients: Effects of duration spent in the sitting position. Br J Anaesth 2001;87:738-42.

9. Standl T, Burmeister MA, Hempel V. Is spinal anesthesia for operations in the prone or jackknife position suitable? Anaesthesist 1999;48:242-50.

10. Limongi JA, Lins RS. Cardiopulmonary arrest in spinal anesthesia. Rev Bras Anestesiolog 2011;61:110-20.

11. Ceruti S, Minotti B, De Vivo S, De Chustophores P, Anselmi L, Saporito A. Prrotocolised care to reduce hypotension after spinal anaesthesia (ProCRHYSA randomized trial): Study protocol for a randomized controlled trial. Contemp Clin Trials Commun 2016;4:39-45.