Pericardial effusion: Real and false

Sir,

Case 1: A 18-year-old male patient with severe mitral stenosis, severe pulmonary hypertension, tricuspid regurgitation and right heart failure was scheduled for mitral valve replacement surgery. Intraoperative transesophageal echocardiographic (TEE) transgastric view revealed large collection of fluid around the posterior aspect of heart giving picture of tamponade compressing heart [Figure 1 and Video Clip 1].

Case 2: A 48-year-old male patient operated case of mitral valve replacement 1 month back presented with severe breathlessness. Patient was intubated in view of respiratory distress and TEE was done. TEE revealed normal prosthetic valve function, but a large pericardial collection on the anterolateral aspect of heart giving tamponade effect [Figure 2].

In images of above presented two cases, collection
compressing heart is evident in the same transgastric short axis view. However, other TEE views in case 1 did not show pericardial effusion [Figure 3]. In transgastric view, falciform ligament is seen which indicates that the collection is of ascitic fluid [Figure 1]. Hence, this case depicts false appearance of pericardial effusion in presence of ascites. In case 2, other views also demonstrated pericardial effusion giving compression effect [Figure 4]. This was a case of true pericardial effusion with tamponade. Effusion was drained by creating pleuropericardial window.

The echo-free spaces near the heart which can be confused with pericardial effusion are mediastinal cysts, lymphomas, thymomas, diaphragmatic hernia, left atrial aneurysm, left ventricular pseudoaneurysm and coronary artery aneurysm.[1]

Echocardiographically, such ascitic echo-free spaces can be mistaken for pericardial effusions and pericardial cysts, which can also present as echolucent spaces anterior to the right ventricle.[1] Understanding the anatomical relationship of the space to the diaphragm, the liver, and the midline linear echo of the falciform ligament can help to distinguish ascites from pericardial fluid accumulation as in this case. The pericardium, diaphragm and parietal peritoneum separates the heart from ascitic fluid.[1]

On transthoracic echocardiography, falciform ligament can be seen in the subdiaphragmatic view. Identification of falciform ligament helps in the differential diagnosis of echolucent space around the right heart border and the liver. Ascites is always recognized by visualizing falciform ligament in this echolucent space.[2]

Transesophageal echocardiographic is definitely diagnostic in pericardial effusion, but comprehensive TEE examination will give proper diagnosis after ruling out other things.

Figure 1: Transesophageal echocardiographic transgastric view showing collection around heart (marked by red arrow) yellow arrow showing falciform ligament of liver margin. RV: Right ventricle, LV: Left ventricle

Figure 2: Transesophageal echocardiographic transgastric view showing collection around heart (marked by red arrow)

Figure 3: Transesophageal echocardiographic midesophageal view showing no collection around heart. Colour Doppler suggesting severe mitral regurgitation. LA: Left atrium, RA: Right atrium

Figure 4: Transesophageal echocardiographic midesophageal four chamber view showing pericardial collection around left side of heart. LA is huge with spontaneous echo contrast. Pr MV: Prosthetic mitral valve
Letters to Editor

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Mishap due to look alike ampule: Matter of serious concern of infusion. Immediately patient was ventilated with 100% oxygen using Bains circuit. Injection propofol (1%)

100 mg was given, and the patient was intubated with size 7.5 endotracheal tube and connected to Bains’ circuit. She was manually ventilated with 100% oxygen. Propofol infusion was started at 50 µg/kg/min. Intravenous infusion in which drug was injected intraoperatively was immediately stopped. Blood sugar and electrolytes were checked and were within normal limits. After about 10 min, patient’s vitals were stabilized and she was shifted to the intensive care unit.

On enquiring, it was found that injection atracurium was inadvertently loaded in place of injection diclofenac as the new batch of atracurium ampules was very similar to ampules of diclofenac. These two ampules had similar label and color coding [Figure 1].

Patient had spontaneous respiratory efforts after around 30 min. Propofol infusion was stopped, and injection neostigmine with glycopyrolate was given. Around 40 min later, patient developed adequate muscle power and was responding to verbal commands. After proper suctioning, extubation was done, and the patient was shifted to postoperative recovery room for further monitoring. She was hemodynamically stable and had 100% SpO₂ on room air. This case highlights the human error, which resulted in inadvertent loading of the wrong drug, resulting in neuromuscular paralysis.

Sir,
The drug errors in anesthesia can be life-threatening. Drug errors not only include administration of wrong drug, incorrect dose or through wrong route, it also includes repetition and omission of drug. Appropriate step must be taken to reduce its incidence.

We are reporting a case of a 45-year-old female scheduled for an appendectomy. She had no other co-morbidities, and all other routine investigations were in normal limit. In the operation theater, monitoring with electrocardiogram, noninvasive blood pressure (BP) and oxygen saturation (SpO₂) was started, and a 20-gauge intravenous cannula was secured. Patient was given subarachnoid block at L3-L4 level in sitting a position using 15 mg of bupivacaine heavy (0.5%) and immediately after that patient was laid supine. Surgery started when adequate sensory and motor levels were achieved.

After around 45 min when surgery was about to end, injection diclofenac was injected in intravenous infusion to avoid positional pain and to provide postoperative analgesia to the patient. Within few minutes of starting the infusion, suddenly patient became restless and dyspneic. She stopped responding to verbal commands and had apnea. BP increased from 110/70 to 150/90 mm Hg, SpO₂ fell from 100% on room air to 65% within 5-10 min.