Coexistent peripartum cardiomyopathy and preeclampsia: A diagnostic dilemma

Dear Editor,

Prevalence of PPCMP is up to four times higher in preeclamptic patients than in normal parturients. Preeclampsia may further worsen PPCMP by persistently increasing the afterload to the left ventricle resulting in systolic failure, ventricular dysarythmias, thromboembolism, and sudden cardiac death.[1] Certain patients never develop a congestive cardiac failure, thus emphasizing the role of echocardiography for early recognition of PPCMP in preeclamptic parturients. Initial diagnostic criteria of PPCMP was mainly clinical, that is, development in the last month of pregnancy or within five postpartum months; absence of other determinable cause of heart failure and any prior demonstrable heart disease. However, echocardiographic evidence of the left ventricular dysfunction like an ejection fraction <45%, fractional shortening <30% or end-diastolic diameter <2.7 cm/m^2 has recently been added, thus aiding in timely detection of this potentially fatal condition.[2]

Symptoms of the two conditions are often overlapping and misleading. Dyspnea being the commonest presenting feature in PPCMP can be confused as worsening cardiac status or impending eclampsia in preeclamptic patients. Also, hypertension in preeclampsia would make clinical consideration of a low cardiac output state like PPCMP improbable. Thus, coexistence of PPCMP in preeclamptic parturients complaining of dyspnea or fatigue should always be considered, even if signs of congestive heart failure are absent. Rather, heart failure should be kept as a differential in all parturients presenting with dyspnoea and easy fatigability.[3] Further, if hypertension is present, the focus should be given to preeclampsia; if absent, dilated cardiomyopathy should be considered.

Diagnostic confirmation can be achieved by echocardiography. Preeclampsia-induced cardiac dysfunction on echocardiography shows diastolic dysfunction with left ventricular hypertrophy, non-dilated ventricle, and normal ejection fraction; while PPCMP manifests as systolic dysfunction, such as reduced ejection fraction, dilated ventricle, and minimal left ventricular hypertrophy.[3] Thus, echocardiography becomes crucial in differentiating the two etiologies. Choice of anesthetic technique weighed heavily in favor of central neuraxial blockade. General anesthesia can be associated with marked hemodynamic perturbations detrimental to both preeclampsia
and PPCMP. Subarachnoid block otherwise considered preferable in both preeclampsia and cardiac diseases has led to cardiac arrest following sudden preload reduction in patients with PPCMP.[4] Therefore, low-dose combined spinal epidural (CSE) anesthesia along with intrathecal opioid is the preferable anesthetic modality in patients with coexistent preeclampsia and PPCMP. It decreases afterload and preload with titrated hemodynamic control and ensures continuous postoperative analgesia negating the adverse hemodynamic effects of increased afterload after autotransfusion and regression of intrathecal anesthesia.

Current literature also advocates the use of regional anesthesia in such cases, with CSE and intrathecal opioids being successfully used,[5] while subarachnoid block has led to complications like intraoperative cardiac arrest or postoperative cardiac decompensation in certain cases.[4] Following delivery, slow infusion of low-dose oxytocin should be used in order to minimize the chances of hypotension. Lastly, invasive hemodynamic monitoring should be used for early detection of impending or obvious cardiac decompensation.

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Conflicts of interest
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