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Case Report

Epidural anaesthesia for abdominal surgery in a 72-year-old with dilated cardiomyopathy: a case report

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ABSTRACT

A 72-year-old female, retired trader, receiving multidisciplinary care for dilated cardiomyopathy and congestive cardiac failure, with massive cardiomegaly, endomyocardial fibrosis, pericardial effusion, atrial fibrillation, ascites and umbilical hernia was scheduled for elective herniorrhaphy at the University of Port Harcourt Teaching Hospital (UPTH). She successfully had surgery under lumbar epidural anaesthesia achieved with morphine, ketamine and low dose bupivacaine combination, and was eventually discharged home postoperatively after satisfactory recovery. The anaesthetic challenges posed by the presence of multiple grave cardiovascular pathologies in association with dilated cardiomyopathy and the critical significance of judicious epidural anaesthetic technique in the successful management of this patient are highlighted.

Keywords: Epidural anaesthesia, Abdominal surgery, Dilated cardiomyopathy

INTRODUCTION

Being the foremost tertiary hospital and training institution, the UPTH, in Port Harcourt the capital of Rivers state in Southern Nigeria has, over the years, recorded a rising trend in surgical patients with critical comorbidities. Dilated cardiomyopathy (DCM) with congestive cardiac failure (CCF) is a globally established leading cause of sudden death. Thus, patients with DCM presenting with clinical conditions requiring elective or urgent surgical intervention constitute significant high risk for anaesthesia. This report features the case of a patient with DCM and CCF who was successfully managed with epidurally administered morphine, ketamine and low dose bupivacaine for umbilical herniorrhaphy. Grimm et al has documented that, despite advanced therapeutic interventions, sudden cardiac deaths, most often due to ventricular tachycardia (VT) or ventricular fibrillation (VF) and less often due to bradyarrhythmias or asystole, constitute 50% of the annual mortality rate of 5-10% in DCM.[1]

CASE REPORT

A 72-year-old, retired female trader, was scheduled to undergo an elective umbilical herniorrhaphy with surgical mesh, at the UPTH. She was being managed by the cardiology and general surgery teams on account of a diagnosis of DCM, CCF and umbilical hernia. The diagnosis was made four months earlier at the UPTH, based on pathognomonic features of the clinical conditions detected from illness history and physical examination, and rendered evident by ancillary echocardiography, electrocardiography (ECG), chest radiography and abdominal ultrasonography (USG). With relative improvement in her clinical condition, she was electively scheduled for umbilical herniorrhaphy.

During the preanaesthetic assessment done 7 days prior to surgery, she was conscious, alert and afebrile (Temp. 36.5^{o}C), with a current weight and height of 61 kg and 1.63 m respectively (BMI 22.96 kgm^{-2}). Her cardiovascular system examination revealed: irregularly

irregular pulse rate (PR) 80 beats/minute (b/min.), blood pressure (BP) 130/70 mmHg, jugular venous distension, a laterally displaced apex beat, muffled 1st and 2nd heart sounds and premature ventricular contractions up to 8 per minute. Bilaterally, there were fine basal crackles and mild pitting ankle oedema. Her echocardiography done two weeks prior to her admission conclusively reported grossly dilated atrial and ventricular chambers, regurgitant tricuspid and mitral valves, a reduced left ventricular end diastolic volume (LVEDV), diastolic dysfunction with preserved ejection fraction (EF) of 62%, endomyocardial fibrosis (EMF) and pericardial effusion; the 12-lead ECG showed atrial fibrillation, an anteroinferior myocardial ischaemia, nonspecific S-T segment changes and ventricular dysrhythmia; massive cardiomegaly was seen on chest radiograph while her abdominal USG reported hepatic congestion, wide umbilical defect and minimal ascites. Her other laboratory investigations were: packed cell volume 41%, prothrombin time 16.1 seconds, activated partial thromboplastin time 61.5 seconds, international normalized ratio (INR) 1.0 and fasting blood glucose 6.3 mmol/L. She was VDRL nonreactive, seronegative to all viral screening and had normal serum electrolytes, urea and creatinine. She was instructed to withhold solid food for 6 hours but take clear fluid up to 2 hours prior to the time for surgery, assigned to American Society of Anesthesiologists (ASA) class III and premedicated with tablet ranitidine 50 mg orally 10 p.m. the previous night and at 6 am in the morning of surgery. A written high-risk consent obtained for epidural anaesthesia (EA), her clopidogrel was stopped for 1 week and replaced with daily subcutaneous enoxaparin 40 mg which was continued including oral digoxin, spironolactone, frusemide and telmisartan till the night before surgery.

On the morning of surgery in the theatre, a multiparameter monitor (Dash 4000®, GE Medicals) was attached and her noninvasive baseline vital signs recorded were: PR 84 b/min., BP 138/74 mmHg, SpO_2 98-99% and axillary temperature 36.7ºC; a 5 lead ECG was also connected. Forearm venous access secured with a 16-gauge Mediflon cannula, prewarmed lactated Ringer's was commenced at 100 ml/hr without prior preloading, ceftriaxone 1g and dexamethasone 8 mg were administered intravenously. Aseptically, her epidural space at the L3/L4 intervertebral level was located at first attempt with a 16 gauge Tuohy needle using loss of resistance to saline technique, following infiltration with 1% plain lidocaine in her sitting position; an 18 gauge epidural catheter was then advanced 4 cm beyond the needle tip, its correct placement confirmed by negative aspiration test to blood and cerebrospinal fluid, as well as by nondetection of HR increase ≥20% of baseline on injecting 3 ml of plain lidocaine 1.5% containing epinephrine 5 µg/ml. The puncture site was covered with sterile gauze and the patient returned to the supine position with slight headup. EA was instituted with 14 ml of 0.20% plain bupivacaine containing morphine 3 mg and preservative free ketamine (Ketamine hydrochloride, Rotex Medica) 30 mg titrated over 8 minutes via the catheter. Post epidural BP was checked every 2 minutes for 10 minutes, then every 2-3 minutes subsequently. Intrao-peratively, her respiratory and haemodynamic parameters remained within stable ranges: BP 102/50-140/70 mmHg, PR 68-77 b/min., SpO_2 97-99% and axillary temperature 36.1-36.4ºC.

Surgery commenced 16 minutes from time of epidural injection, the patient having achieved adequate sensory block height (T7) and spontaneously breathing supplemental oxygen delivered at 3 L/min. via nasal prongs. Her estimated blood loss was 80 ml while she received a total of 150 ml of lactated Ringer's intraoperatively. At the end of surgery which lasted 83 minutes, she was shifted to the ward following satisfactory observation for 1 hour in the recovery room and discharged home after 7 days to continue receiving outpatient care.

DISCUSSION

In 1995 the World Health Organization/International Society and Federation of Cardiology Task Force defined cardiomyopathies as diseases of the myocardium associated with cardiac dysfunction, classifying them as DCM, hypertrophic cardiomyopathy, restrictive cardiomyopathy, and arrhythmogenic right ventricular cardiomyopathy.[2] This spectrum of cardiac muscle diseases can be primarily idiopathic or secondarily arise from such clinical conditions as coronary ischaemic damage, hypertension, myocarditis, HIV, thyrotoxicosis/hypothyroidism, peripartum state, cardiac amyloidosis, chemo-/radiotherapy, substance abuse and Duchenne's muscular dystrophy.[2]

DCM is the ranking variety of cardiomyopathy and the most common indication for cardiac transplantation.[3] The disease, being the third most common cause of cardiac failure, is typically characterized by left ventricular or biventricular dilatation, impaired myocardial contractility, systolic dysfunction [reduced stroke volume (SV)], and decreased cardiac output with ejection fraction (EF) <40%.[3,4] Pathophysiologically, a cardiomyopathic process trigerred idiopathically, or by a secondary cause, results in progressive ventricular chamber dilatation, tricuspid and mitral valvular regurgitation, consequent further decrease in EF with increase in ventricular wall stress and end systolic volumes causing fixed cardiac output state. Activation of neurohumoral mechanisms increases peripheral vascular tone elevating ventricular afterload which stimulates geometric ventricular remodeling that worsens myocardial injury. The associated compensatory tachycardia, through reducing coronary perfusion, causes myocardial ischaemia with resultant conduction abnormalities precipitating ventricular dysrhythmia and predisposing to thromboembolism.[5] In most cases, without transplant, progression to death occurs.[4]

The clinical presentation and course of DCM may vary widely, ranging from progressive cardiomegaly and CCF to left ventricular reverse remodeling (LV-RR).[6] In LV-RR, there is decrease in LV volumes combined with a significantly improved systolic function. This patient, instead of manifesting the characteristic decrease in DCM, had a preserved EF (prEF) of 62%. The prEF may be apparent, indicating a superimposed restrictive cardiac pathology such as EMF, or real, showing favourable therapeutic response. In EMF, LV stiffening occurs resulting in reduced LVEDV. Since, mathematically, EF=(SV)/**(**LVEDV), a decrease in LVEDV due to restricted ventricular relaxation will falsely raise the EF even though there is SV deficit. Based on her echocardiographic evidence of EMF and pericardial effusion, therefore, the prEF of 62% is not attributable to treatment outcome alone. EMF has been documented as aetiological to restricted ventricular diastole which is worsened by pericardial effusion.[7]

Due to its association with high sudden cardiac death rate, DCM complicated by CCF poses a significant challenge to the Anaesthesiologist. An insight into the pathophysiology and into the likely resultant from the interplay between it and anaesthetic agents/procedures, therefore, is indispensable to the successful delivery of anaesthesia. The goals of functional airway security, adequate oxygenation/ventilation, adequate analgesia, abdominal muscle relaxation, maintenance of normovolaemia and haemodynamic stability are critical to achieving desirable perioperative outcome. General anaesthesia (GA) with tracheal intubation and controlled ventilation can be used for this patient though, the attendant sympathoadrenal axis response to laryngoscopy, tracheal intubation and extubation, and consequent hypertension, tachycardia and arrhythmias can aggravate an already critical cardiac physiology.[8] The use of clinical doses of preintubation propofol and beta-blockers for desirably obtunding this stress response carries risk of deleterious myocardial depression for a failing heart. Besides, there is the likelihood of an unanticipated difficult airway predisposing to hypoxia and aggravating preexisting myocardial ischaemia.

Importantly, Marana et al observed that surgical procedures are associated with a complexity of stress response characterized by neurohumoral, immunologic, and metabolic alterations including increased oxygen consumption, catabolism, and impaired immune function leading to poor postoperative course and clinical outcome.[9] That this stress response is, however, modifiable by the choice of anaesthetic modality through the inhibition or modulation of the pathophysiologic pathways has been reported.[10] In this regard, EA has documented superiority in efficacy over endotracheal GA, as neuraxial blockade from EA completely prevents impulse transmission, thereby significantly attenuating the stress response and its sequelae.[11] Local anaesthetic based EA, in contrast with subarachnoid block that may cause precipitous hypotension and bradycardia, is usually accompanied by a mild, more gradual onset neuraxial blockade and sympathectomy with slight decrease in peripheral resistance and LV afterload, and without significant haemodynamic derangements, especially when titrated low doses are used.[12,13] Therefore, EA was chosen for this patient keeping in view the anaesthetic and haemodynamic goals, and this supports the reports by previous authors.[12,13]

A total of 14 ml plain bupivacaine 0.20% including morphine 3 mg and preservative free ketamine 30 mg was titrated epidurally in this patient, to deliver an adequate volume of an optimally low concentration of bupivacaine, so as to achieve desired analgesic quality with longer duration than can be achieved using bupivacaine without adjuvant. Empirically, Verghese et al demonstrated that a caudal epidural block with larger volume (1 ml/kg) of dilute (0.20%) bupivacaine was more effective than a smaller volume (0.80 ml/kg) of a more concentrated (0.25%) solution in blocking spermatic cord traction response in children.[14] The slower onset and much longer analgesic duration of epidural morphine underpins its being the preferred choice for this patient while the addition of ketamine was to implement targeted multimodal central neuraxial analgesia. Research has documented the synergistic analgesic efficacy of epidural bupivacaine, morphine and ketamine combination.[15]

Patients with DCM necessarily receive anticoagulant therapy for thromboprophylaxis and are thus predisposed to developing postepidural haematoma with consequent cauda equina syndrome.[16] Therefore, ensuring an optimal INR and timely conversion of long acting to short acting anticoagulant in such patients is judicious. This patient was discontinued from clopidogrel 7 days, and converted to daily subcutaneous enoxaparin which was temporarily stopped 12 hours, prior to surgery. Lactated Ringer's 150 ml was administered without preloading as meticulous fluid administration is required in fixed cardiac output state.[17] No perioperative complications occurred.

CONCLUSION

Given the high sudden cardiac death rate in association with DCM, an in-depth understanding of its pathophysiology, good preoperative evaluation and preparation, as well as judicious choice of anaesthetic technique are prerequisites to circumventing perioperative mortality in patients suffering the condition. Lumbar epidural anaesthesia involving titrated administration of morphine, ketamine, and low dose (0.20%) bupivacaine combination proves to be safe for umbilical herniorrhaphy in a patient with DCM.

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