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Cardiac Decompensation in a Patient with Eisenmenger Syndrome Undergoing T5-T7 Levels Laminectomy in the Sitting Position

Oturur Pozisyonda T5-T7 Laminektomi Yapılan Eisenmenger Sendromlu bir Hastada Kardiak Dekompenzasyon

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ABSTRACT

Although several different anesthetic techniques have been employed for non-cardiac surgery in Eisenmenger syndrome (ES), the best anesthetic technique is still controversial. A 17-year-old male with ES and an epidural abscess at the T5-T7 levels was scheduled to undergo laminectomy in the sitting position. Anesthesia was induced by ketamane and maintained with an opioid-isoflurane technique. A few minutes after extubation, respiratory distress appeared and the patient was reintubated. He was transferred to the intensive care unit (ICU) and maintained on spontaneous intermittent mandatory ventilation and pressure support for 24 hours, and discharged fully recovered on the 4th postoperative day. To our knowledge this is the first case where the sitting position has been adopted for evacuation of an epidural abscess at the T5-T7 levels in a patient with ES. We recommend late extubation and an overnight observation in the ICU for such patients to prevent dangerous sequelae.

KEYWORDS: Eisenmenger syndrome, Epidural abscess, Ketamine, Sitting position, Late extubation

ÖZ

Eisenmenger sendromunda (ES) kardiak olmayan cerrahi için birçok değişik anestezi tekniği kullanılmış olmasına rağmen en iyi anestetik teknik hala tartışmalıdır. T5-T7 seviyelerinde epidural absesi olan ES`li 17 yaşında bir erkek hastaya oturur pozisyonda laminektomi planlandı. Anestezi ketaminle indükte edildi ve opioid-isoflurane tekniğiyle idame edildi. Ekstübasyondan birkaç dakika sonra solunum sıkıntısı görüldü ve tekrar entübe edildi. Hasta yoğun bakım ünitesine alınarak 24 saat spontan aralıklı zorlayıcı ventilasyon ve basınç desteğinde kaldı ve postoperatif 4. günde tamamen iyi olarak taburcu edildi. Bildiğimiz kadarıyla bu vaka, ES`li bir hastada T5-T7 seviyelerinde bir epidural absenin boşaltılmasında oturur pozisyonun ilk kez kullanılmasıdır. Tehlikeli sonuçların engellenmesi için böyle hastalarda geç ekstübasyon ve bir gecelik yoğun bakım gözlemi öneriyoruz.

ANAHTAR SÖZCÜKLER: Eisenmenger sendromu, Epidural abse, Ketamin, Oturur pozisyon, Geç ekstübasyon

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INTRODUCTION

The Eisenmenger syndrome (ES) was coined for the first time by Wood (12) and was defined as increased pulmonary vascular resistance (PVR) with pulmonary hypertension resulting from a left-to-right shunt which can become bidirectional or right to left.

Complications after non-cardiac surgery, such as congestive heart failure (25%) and hemoptysis (15%) are important causes of death in ES (3). Depending on the surgical procedure; perioperative mortality in patients with ES is 4-25% (7). The underlying reason is loss of control over the direction and flow of intracardiac shunt, with increasing right-to-left shunt reducing systemic oxygen delivery. Balance of systemic vascular resistance (SVR) and PVR is crucial during surgery (7). Factors altering the balance of SVR and PVR include hypercarbia, hypothermia, atelectasis, and uncontrolled vasodilation by anaesthetics, hypovolemia, pain-induced stress, altered intrathoracic pressures by artificial ventilation, and hypoxia. The best anaesthetic technique (regional anaesthesia, general anaesthesia or a combination of both) to control SVR and intracardiac shunt is controversial (4, 7).

The sitting position is customarily used for posterior fossa surgery (8) in ES, but to our knowledge there is no report about using this position for spinal surgery. In this paper we describe a patient with ES and epidural abscess (T5-T7) who underwent spinal surgery in the sitting position.

CASE REPORT

A 17 year old male having an epidural abscess due to septic emboli at T5-T7 levels was scheduled to undergo laminectomy because of worsening paraparesis. The surgery was scheduled to be conducted in the sitting position. Important congenital anomalies in this patient included transposition of the great vessels (D- TGV), and complete endocardial cushion defect (atrioventricular canal defect) and suprasystemic pulmonary hypertension. Cardiac catheterization prior to surgery had revealed subtle findings (Table I) which helped us in evolving an anesthetic strategy suited to the patient's condition and to serve as a useful base line predictor of postoperative functional status.

Laboratory assessments demonstrated hemoglobin of 18.6 g/100ml and hematocrit of 66% revealing polycythemia. Blood chemistry and coagulation profile were normal. An arterial blood gas (ABG) analysis prior to surgery showed a normal pH of 7.45 and an oxygen saturation (O_2 Sat) of 87.2% with a partial pressure of oxygen in the arterial blood (Pa O_2) of 50 mm Hg.

Table I: Cardiac catheterization data prior to surgery.

Site	Pressure (mm Hg)	O_2 Saturation
SVC		50%
RA	8/5	60%
RV	110/0-8	75%
PA	100/50	75%
LV	100/0-8	89%
AO	100/50	87.2% (PO_2 =50 mmHg)

On clinical examination, the patient had marked clubbing of the fingers and toes, and marked peripheral and central cyanosis (Figure 1). On auscultation, S_2 was accentuated and a grade III/IV systolic murmur was audible at the lower left sternal border. The lungs were clear, and the chest X-ray showed a typical replica of an ES (Figure 2).

The pre-induction blood pressure and heart rate were 110/70-mmHg and 124-beats/ min respectively.

After placing the standard monitors, anesthesia was started with midazolam 1mg and sufentanil 10μg given intravenously (I.V) while the patient was receiving 100% oxygen via facemask in the supine position. Later, ketamine 90mg was given as an induction agent, and pancuronium bromide 6mg (I.V) administered to achieve muscle paralysis. Ninety seconds prior to laryngoscopy, lidocaine 60mg (I.V) was given and endotracheal intubation accomplished. Following induction, the heart rate decreased to 104 beats/min, but the blood pressure remained unchanged. Additional monitoring including an arterial line by cannulating the right radial artery and a central venous pressure (CVP) line via the right subclavian vein were established. The patient was then gradually changed over to the sitting position to compensate for the changing hemodynamic variables in this position (Figure 3). Anesthesia was maintained with O_2 and N_2O in a 50:50 mixture along with isoflurane 0.8 – 1.2% and incremental doses of sufentanil as needed.

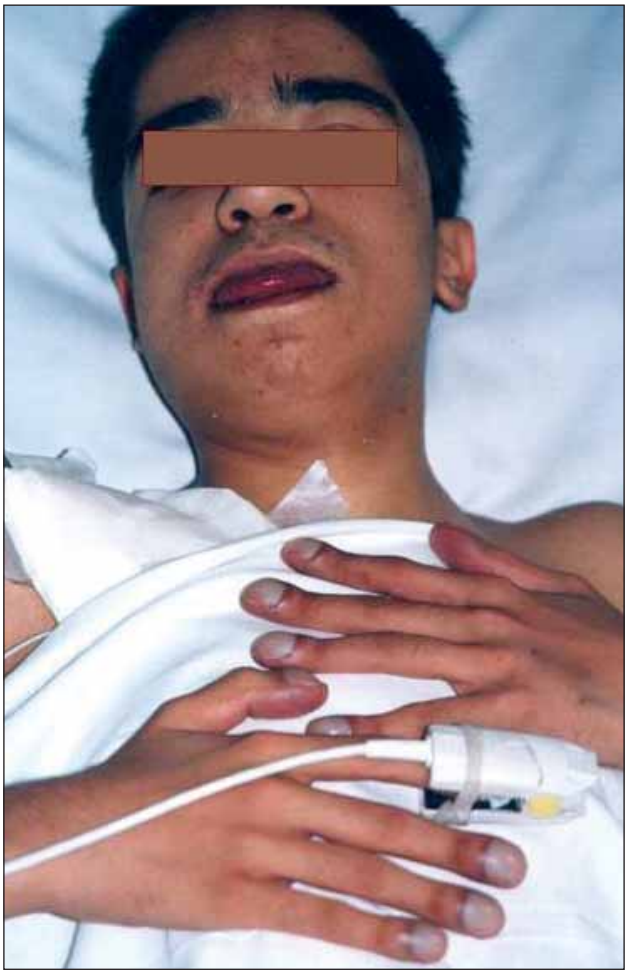

Figure 1. *Marked clubbing of the fingers and toes, and marked peripheral and central cyanosis.*

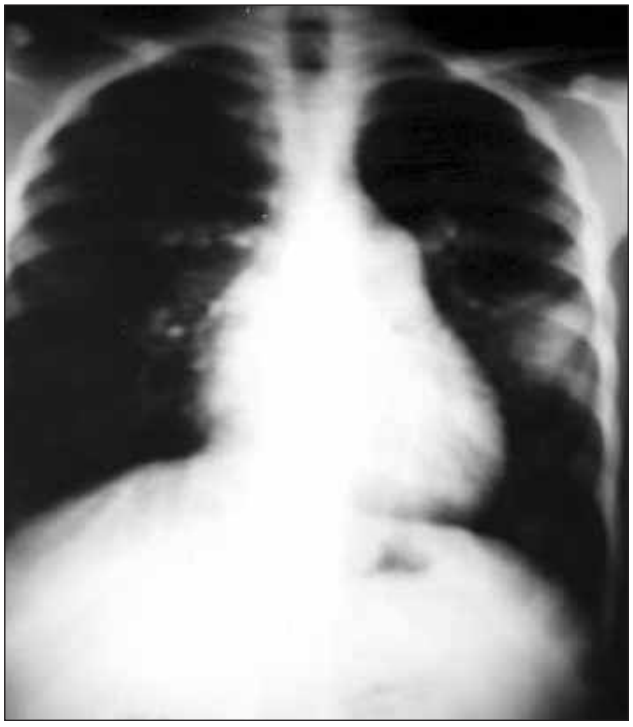

Figure 2. *Chest X-ray showing a typical replica of an Eisenmenger syndrome.*

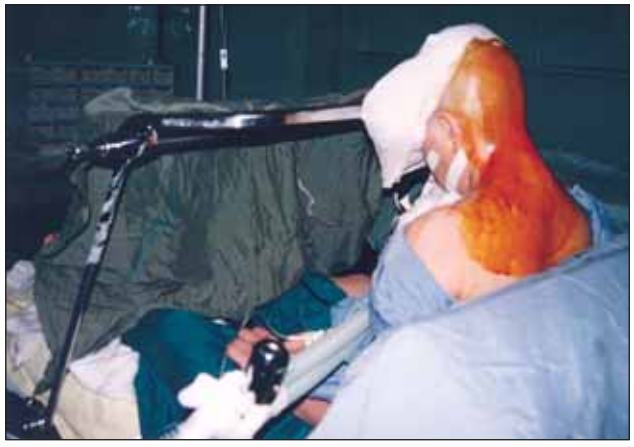

Figure 3. *Patient positioned in the sitting position prior to surgery.*

Ventilation was adjusted to maintain the partial pressure of carbon dioxide in the arterial blood (PaCO2) between 35-40 mmHg. During the surgery the patient received 1500 ml of Ringer lactate and the CVP was maintained between 10-15 mmHg. The mean arterial blood pressure (MABP) remained stable around 80 mmHg except for a brief period an hour after surgery when it touched 50 mmHg (a short period necessitating discontinuation of isoflurane and judicious administration of fluids). The MABP showed a gradual rise reaching 80 mmHg again without resorting to vasoactive drugs. The 2-hr surgery went uneventfully. After completion of surgery and shifting the patient to the supine position, the patient was extubated after return of airway reflexes and reversal of the muscle relaxation. The patient was alert when he was extubated. He was then observed in the recovery room receiving 6L/min O2 via facemask. After a lapse of a few minutes, paradoxical movements of the chest and abdomen were observed indicating respiratory embarrassment.

A jaw thrust was applied but was not helpful. Auscultation of the heart and lungs revealed generalized crackles and a high-pitched accentuated murmur. The patient was re-intubated under lidocaine spray, and furosemide 30 mg and digoxin 0.5mg were administered intravenously to achieve

rapid digitalization. After 45 min., with control of ventilation made feasible by morphine 5 mg and pancuronium 2mg, the lungs became clear and the murmur lost its harsh high pitch.

The patient was transferred to the Intensive Care Unit (ICU) and maintained on spontaneous intermittent mandatory ventilation and pressure support for 24 hours, and discharged fully well on the 4th postoperative day. One month later, the patient underwent a heart and lung transplant but died 7 days later due to septicemia.

DISCUSSION

General, epidural and spinal anesthesia have been used successfully in patients with ES undergoing non-cardiac surgery, but they have not been compared in a randomized study (4). Of prime importance hemodynamically is control of shunt direction, maintenance of normovolemia, a stable heart rate, and avoidance of myocardial depression (4). Control of ventilation with manipulation of Pco_2, pH and lung volumes are at present the only available means of altering PVR independently of systemic vascular resistance "SVR". We used ketamine as an induction agent on the assumption that it would provide hemodynamic stability in terms of arterial blood pressure, cardiac output and SVR. Peripheral muscarinic inhibition could explain the preponderance of sympathetic tone noted after ketamine administration (9).

Maintenance of the preoperative SVR is considered the cornerstone of the anesthetic management of patients with ES (6). Whereas thiopentone and propofol decrease SVR (5) and could be deleterious, ketamine has been recommended as the drug of choice for induction because of little effect on pulmonary and systemic vascular resistance. Although the exact mechanism of ketamine inducing vasodilator effect in the pulmonary bed is not fully understood, ketamine may involve an effect on calcium influx or release from the sarcoplasmic reticulum (1,2). However ketamine causes minimal increases in PVR as long as ventilation and FiO_2 are kept constant. We adhered to this principle in maintaining controlled ventilation and providing an acceptably high FiO2. Although halothane, enflurane, and isoflurane have all been incriminated to cause systemic hypotension because of a varying combination of myocardial depression and vasodilatation, the opiod-N2O-isoflurane anesthetic technique was employed in this patient. The concentration of isoflurane was not large enough to incur any deleterious effects on the hemodynamic variables.

One thing unique about our case is the sitting position during surgery, which entails its own inherent risks and imparts situations that can imperil the patient's life. It has been shown that changing the position of ES patients from sitting to supine elicits significant reductions in PaO_2 and SaO_2 (10). The foremost and life threatening complication is venous air embolism or paradoxical embolism (11). To prepare for such outcomes, the right subclavian vein had been cannulated to monitor CVP, and to serve as a conduit to retrieve air if it inadvertently gushed in via an open vein. To prevent the occurrence of paradoxical air embolism, all intravenous lines were de-aired. However, the sitting position is associated with an increased SVR (8) and an increment of this hemodynamic variable suits the scenario in favor of ES. A reduction in cardiac output- and MABP, which are seen with the sitting position (8), could change the shunt direction and desaturate the patient, were taken care of by judicious volume loading and could also have been prevented because of the enhanced sympathetic tone attributed to ketamine's action in this case.

After extubation, the perilous heart showed signs of decompensation, which effectively responded to re-intubation and administration of furosemide and digoxin. It is hypothesized that the extubation in this patient was ill-conceived and ushered in a plethora of events such as an increase in afterload and an increase in preload exacerbated and potentiated by changing over the patient to the supine position and discontinuation of controlled ventilation. Thus, it is conceivable that the patient who was meticulously attended to lost the entire umbrella of vigilant care, and failed to accommodate to the plethora of sudden hemodynamic changes culminating in overt signs of heart decompensation. The CVP maintained at a high level in the sitting position was perhaps not tolerated in the supine position and could also have contributed to the scenario of acute decompensation of the heart.

This report has some limitations. First, we used pancuronium as a neuromuscular blocking agent while neuromuscular junction monitoring did not apply. It can be postulated that the residual paralysis produced acute onset of respiratory signs which

triggered cardiac decompensation in our patient. Second, we did not perform intraoperative or immediate post-operative echocardiography which could have helped us in clinching the incriminating causes (respiratory vs. cardiac) of this event. Third, although the role of N_2O on pulmonary vascular resistance remains controversial, nevertheless logic dictates that it should have been omitted from the anesthetic armamentarium owing to its potential and theoretical role of increasing PVR and thus exacerbating the right-to-left shunting and ushering in air embolism which are the possible and expected sequelae in the sitting position in such a patient.

In conclusion, this case provides some important lessons, the foremost of which is that early extubation should be avoided in such patients, especially those who undergo non-cardiac operations in the sitting position. They should preferably be observed overnight in the ICU under close surveillance to overt the occurrence of complications which can overshadow an otherwise proper perioperative care. Secondly, the return of airway reflexes and other criteria of extubation should not motivate us to extubate such patients because there seemingly are other important criteria – the hemodynamic variables that only remain optimized under ventilatory control.

Although it is difficult to generalize on one single patient, nevertheless this single report with its rare occurrence coupled with the sitting position which has been rarely reported in literature for such cases, and the development of acute cardiac decompensation are some of the points that motivated us to report this case.

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