Case Report

Continuous Sedation-analgesia Delays Diagnosis of Compartment Syndrome in a Patient with Intra-aortic Balloon Pump

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Abstract

Compartment syndrome is a rare, devastating complication of coronary artery bypass grafting (CABG) and intra-aortic balloon pump (IABP). Prompt diagnosis is based on symptoms and signs and is paramount for limb rescue. This report describes a CABG patient with IABP in whom receiving continuous analgesia-sedation obscured the symptoms of compartment syndrome.

Keywords: Analgesia, compartment syndrome, inta-aortic balloon pump, sedation

Cite the article as: Barkhordari K, Yousefshahi F, Khajavi MR, Karimi A. Continuous Sedation-analgesia Delays Compartment Syndrome Diagnosis in a Patient with Intra-aortic Balloon Pump. *Arch Iran Med.* 2012; **15(6):** 387 – 388.

Introduction

Compartment syndrome is an uncommon complication after a coronary artery bypass grafting (CABG) and intra-aortic balloon catheter, in which a delay in treatment can result in significant disability.[1,2] Clinical diagnosis is often possible in conscious patients, but in unconscious patients the diagnosis is very difficult and may be missed.[3]

By reviewing previously published cases, Al-Sarraf et al. have found a relationship between deep sedation, ventilation and compartment syndrome in cardiac surgery patients during the initial postoperative period, which commonly leads to a prolonged delay in detection.[4] This case represents a rare case of delayed diagnosis of compartment syndrome in a non-intubated patient on a continuous moderate sedation-analgesia protocol.

Case Report

A 47-year-old woman with a history of hypertension was admitted to our hospital for a planned CABG. There was no history of other medical conditions. Her pre-operative hemodynamic status was stable.

She underwent on-pump coronary artery bypass surgery by standard median sternotomy. Autologous saphenous vein grafts were used to bypass the left anterior descending and first obtuse marginal arteries. The surgery was uneventful, but at chest closure her blood pressure decreased despite inotropic support. Thus the surgeon placed an intra-aortic balloon catheter via the left common femoral artery.

The patient was admitted to the Surgical Intensive Care Unit with the intra-aortic balloon pump (IABP) support still in place. She was intubated and received propofol 50 $\mu g.kg^{-1}.min^{-1}$, fentanyl 2 $\mu g.kg^{-1}.min^{-1}$, heparin 500u.h^{-1}, NTG 0.3 $\mu g.kg^{-1}.min^{-1}$ and epinephrine 0.03–0.07 $\mu g.kg^{-1}.min^{-1}$. Her Ramsay Sedation Scale (RSS) score was 3–4. Her pulse was repeatedly documented as palpable during this time. On the first postoperative day, mediastinal drainage was continued at about 100cc.h^{-1} for 8 hours, therefore sedation and analgesia continued and the patient was transferred to the operating room where hemostasis was performed.

The patient was returned to the Surgical Intensive Care Unit where she was extubated, however, because she was agitated we provided her with light sedation as an adjunctive to analgesia, using propofol (20–40 $\mu g.kg^{-1}.min^{-1}$) and fentanyl (0.5–2 $\mu g.kg^{-1}.min^{-1}$), adjusted to an RSS score of < 3.

There were no complaints of calf pain until the second postoperative day when we detected a decrease in palpable pulse of the dorsalis pedis artery and erythema on the distal side of the left leg, above the ankle. The intra-aortic balloon catheter was removed and the patient underwent vascular and orthopedic surgery consultations. Sedation and analgesia was stopped. The erythema increased, and the leg was warm but the calf was tense. The patient was unable to flex her left ankle. Pain was elicited on passive dorsiflexion and with calf compression. Although pulse oximetry of the left toes showed an SpO_2 of 97%, the dorsalis pedis pulse was absent on Doppler ultrasound.

We performed an anterior and lateral compartment fasciotomy. There was no hematoma or hemorrhage. The muscles were edematous, but some were necrotic and debrided. An embolectomy of the dorsalis pedis artery was performed, using a Fogarty balloon catheter, but was not successful and the pulse did not return. On the fourth postoperative day, additional muscles in the anterior and lateral compartments became necrotic and they were debrided. On the sixth postoperative day, the posterior compartment also became tense; both superficial and deep fasciotomies were performed. The same scenario that occurred in the anterior compartment also happened in the posterior compartment.

By day 28, the distal parts of the majority of muscles in the anterior and posterior compartments were necrotic and required debridement. Subsequently the patient was transferred to an orthopedic rehabilitation service for supportive care.

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Accepted for publication: 16 November 2011

Discussion

Pain control and sedation after surgery reduces discomfort, stress response and inflammation, provides anxiolysis and improves the tolerance of ventilatory support. Current evidence supports the use of intravenous (IV) delivery methods of opioids following CABG, especially if pain is not controlled by the patient during the first 24 hours following a CABG.[5]

Although compartment syndrome is a relatively rare complication after CABG and intra-aortic balloon catheter insertion, it is a dangerous, limb-threatening condition.[1,2,4] Early diagnosis is based on clinical signs and symptoms that require a detailed examination, a vigilant examiner, and a cooperative patient. Pain and tenderness are initial signs of compartment syndrome.

Compartment pressure measurement, near-infrared spectroscopy (NIRS), pulse oximetry, and many other methods have been used for diagnosis, but each has some shortcomings.[6] Pain and sensory alterations in a leg with an intra-aortic balloon catheter in place should prompt evaluation for acute compartment syndrome even if pulses are continuously present.[2] Pulse oximetry is not an appropriate method for detecting or monitoring impaired perfusion. It cannot measure intracompartmental tissue oxygen saturation and is unable to detect intracompartmental hypoperfusion.[6]

Deep sedation in cardiac surgery patients during the initial postoperative period commonly leads to prolonged delay in the diagnosis of compartment syndrome.[4] Although a systematic review that has collected data on 28 cases concluded that there was no evidence for a relationship between patient-controlled analgesia (PCA) opioids or regional analgesia and delay in diagnosis of compartment syndrome, most cases in that review received epidural analgesia.[7] Information on the effect of the combination of sedation and IV analgesics is less known. The use of analgesic methods that impair the sensation of normal levels of pain may place patients at risk of the devastating sequelae of a missed compartment syndrome.[8]

In our patient, we assume that the combination of pain relief and reduced consciousness due to continuous analgesia and sedation made the assessment of the sensory and motor functions of the lower limb more challenging. When other signs of compartment syndrome had developed and compartment syndrome was suspected, it was too late and treatment measures were not immediately effective.

We recommend that continuous postoperative sedation analgesia be used cautiously in patients at risk of compartment syndrome, especially in patients with multiple risk factors such as a CABG operation and an IABP. In addition, it should be interrupted frequently to look for signs and symptoms of this syndrome. If continuous sedation analgesia is used, careful monitoring of the patient with several of the modalities mentioned above should be performed.

Consent: We have presented this case report based on written consent from the patient.

Competing interests: The authors declare that they have no competing interests.

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