Continuous Sciatic Nerve Block with 0.1% Bupivacaine and the Early Detection of Compartment Syndrome in a Pediatric Patient: a Case Report

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Abstract

Concerns for regional anesthesia masking pain as a cardinal symptom, and therefore, leading to a delay in recognition of acute compartment syndrome (ACS) have been raised. A 14-year old male with medial malleolus tibial and distal fibula fractures suddenly developed severe pain on postoperative day 1 with a well-functioning sciatic nerve catheter. This pain was not able to be relieved by increasing catheter infusion rate of 0.1% bupivacaine and additional opioids administration. Acute compartment syndrome (ACS) was diagnosed and the patient subsequently underwent de-compressive fasciotomy.

The main lesson to learn from this case is that with highly index of clinical suspicion and ongoing assessment, sudden loss of efficacy of continuous nerve block with diluted bupivacaine after extremity surgery is a potential alarm of compartment syndrome in a pediatric patient.

Key Words: Continuous Sciatic Nerve Block; Acute Compartment Syndrome; Bupivacaine

Introduction

Regional anesthesia plays an important role in pediatric intra-operative and post-operative pain relief by reducing opioid consumption and decreasing associated side effects of opioids [1]. Continuous sciatic nerve block with diluted bupivacaine is useful in providing lower extremity analgesia for children undergoing orthopedic surgery. Acute compartment syndrome (ACS) is a surgical emergency for which the timing of diagnosis is essential because increased pressures within a closed compartment can compromise circulation and function of tissues within the space. Diagnosis of ACS is based on clinical symptoms, intra-compartmental pressure, or both [1]. Delaying diagnosis and treatment may lead to irreversible neuromuscular ischemic damage, which can occur within 4-6h after the onset of symptoms [2]. Hence concerns about masking pain as cardinal symptom and therefore leading to a delay in diagnosis and therapy have been raised in connection with regional anesthesia.

Patients Presenting Concerns and Clinical Findings

A 14-year-old otherwise healthy male presented with gross deformity of the right ankle to our emergency department (ED). He was found to have right medial malleolus tibia fracture and distal fibula fracture with lateral subluxation, but no nerve injury.

Initial Treatment and Timeline

The patient underwent closed reduction under hematoma block in the ED and received open reduction internal fixation in operating room (OR). The patient received propofol and fentanyl during anesthesia induction. Intra-operatively, a popliteal fossa sciatic nerve block with ropivacaine 0.2% was administrated and a catheter was placed to introduce an infusion of 0.1% Bupivacaine at 8 ml/hr. Post-operatively, the sciatic nerve catheter infusion was continued and no additional long acting opioids were given. The evolution of this case is outlined in Figure 1.

Case Reports

Postoperative Presenting Concerns and Clinic Findings

The patient was assessed in the post-anesthesia care unit (PACU) and was found to be comfortable. He had a normal PACU course and was discharged to the pediatric inpatient floor. The patient’s sensory and motor functions were checked every 2 hours by the

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nurse. Both patient and nurse used visual analogue scale (VAS) to evaluate the pain. The patient reported minimal pain rated 0/10 overnight on his initial post-operative day (POD). However, at 6 am on POD1, the patient waked up with 4/10 pain.

**Therapeutic Interventions and Diagnoses with Acute Compartment Syndrome**

The patient received 2 mg intravenous administration of morphine with minimal relief. Two hours later, the pain worsened to a scale of 10/10 and the acute pain service was urgently called. His sensory function was found to be intact at this point. The acute pain anesthesiologist assessed the patient and the nerve catheter and felt that the nerve catheter is in the correct position and functioning properly. The nerve catheter infusion rate was increased to 10 ml/hr and another 4 mg IV morphine was given to the patient after which the patient received some relief. However, 30 minutes later, the pain elevated again and the patient developed severe upper thigh muscle spasm. This spasm was minimally relieved with 4 mg Diazepam. The anesthesiologist was called again to evaluate the patient at which point there was a concern for ACS. The orthopedic team was called at 9:30 am. Meanwhile, severe pain persisted despite effective sensory block and additional IV opioids.

At 12:00 pm, the patient was evaluated by the orthopedic surgery team and diagnosed with ACS based on the clinic findings and increased intra-compartment pressure measurement. The patient subsequently underwent de-compressive fasciotomy. He eventually made complete recovery with no residual neuromuscular deficits.

**Discussion**

Acute compartment syndrome (ACS) is an increase in intra-compartmental pressure leading to a decrease of perfusion pressure and hypoxemia of the tissues. The incidence of ACS is 7.3 per 100,000 men and 0.7 per 100,000 women [3]. Muscle mass, fracture pattern and fracture location are important factors for ACS [4]. Mid-diaphyseal tibia fractures, tibial plateau fractures, medial knee fracture-dislocations and bicondylar tibial plateau fracture are the surgeries that are prone to ACS. The intrinsic causes of ACS are tissue injury caused by a direct traumatic event or ischemia/reperfusion [1]. The elevation of pre-capillary vasodilation in the arteriole system, the collapsing venules and the increased permeability of the capillary bed together result in raised interstitial fluid pressure and consequently decreased tissue...
perfusion. The tissues at risk are subject to hypoxia, elevation of oxidant stress and development of hypoglycaemia, which further worsen the cellular metabolic balance and result in a positive feedback loop worsening ischemia.

Timely diagnosis of ACS is essential regarding the disease progression. Although the time for initiating events to ACS can vary from minutes to hours, the muscular and peripheral nerve function are affected by ACS in a time-dependent manner. Ischemia of 1 hour can lead to reversible neurapraxia [5] and irreversible damage could occur as early as 2 hours depending on the degree of pressure elevation.

Diagnosis of ACS can be based on clinical symptoms, intra-compartmental pressure, or both. For the pressure measurement, the absolute compartment pressure greater than 30mm Hg is thought to be an indication of impaired tissue perfusion in adults and children [4]. However, because the perfusion pressure depends on the blood pressure, the differential pressure (∆P=diastolic blood pressure – intra-compartmental pressure) with a threshold of 30 mmHg was proposed. The classic clinical symptoms of ACS are the 6 P’s: pain, paresthesia, pallor, paralysis, pulselessness and poikilothermia. These signs can have low sensitivity but high specificity [6]. Among these P’s, pain and paresthesia are most essential while others may be misleading or present as late signs [1]. When it comes to the pediatric patients, the lack of cognitive or verbal ability to provide meaningful clinical information can bring more challenge to care providers. In a survey conducted at Boston Children's Hospital between 1992-1997, 75% pediatric cases reported pain while only 39% patients reported pain and 1 or 2 other signs [7]. The most sensitive indicator of ACS is the increase in requirement for pain medications, which preceded other clinical symptoms by an average of 7.3 hours [2]. Agitation, anxiety and analgesic requirement escalation are more likely to occur in children than the 6 P’s.

**Figure 2:** Timeline of postoperative pain and acute compartment syndrome (ACS) diagnoses on postoperative day (POD) 1. VAS: Visual Assessment Scale. IV: intravenous.
Regional anesthesia is an effective method to control postoperative pain after pediatric orthopedics surgery. Whether analgesia provided by regional anesthesia techniques would mask the symptom of ACS and consequently delay the diagnosis of ACS remains a controversial issue. Currently, there is no convincing evidence that patient-controlled opioid analgesia or regional analgesia would delay the diagnosis while patients are adequately monitored.

In this case, we provided a sciatic nerve block with 0.2% ropivacaine and 0.1% bupivacaine infusion to our patient for postoperative pain control. Both the block and the catheter placement were under the guidance of ultrasound. After being transferred to pediatric floor, the patient complained minimal pain overnight but slept well after intravenous administration with 2mg morphine. However, despite efficacy of regional anesthesia, the patient was still able to develop symptoms heralding ACS on the morning of the first postoperative day. Of note, neither increasing the rate of 0.1% bupivacaine infusion nor intravenous administration of opioids medication could completely relieve the pain caused by ACS.

Before ACS diagnoses, reasons accounting for the severe and aggressive pain include the followings: Catheter-related complications, postoperative pain or acute compartment syndrome. The primary catheter-related complications include catheter failure owing to pump dysfunction or catheter obstruction, inadequacy of analgesia due to misplacement, continuous motor block, insertion point inflammation or infection [8]. A prospective study including 1010 ultrasound-guided consecutive blocks pointed out that new, all-cause neurological symptoms were present in 8.2%, 3.7% and 0.6% blocks at day 10, 1 month and 6 months [9]. To our best of knowledge, reports about neurological symptoms happened immediately after surgery is lacking. In our case, the pain control is sufficient and effective as evidenced by the pain score immediately post-operatively. The sensory testing and frequent patient assessment also ensured that our catheter was in correct position. The insertion point is clear and there is no sign of infection or severe inflammation. Therefore, we don’t consider that catheter-induced pain contributed to the severe pain. Nevertheless, since we only block the sciatic nerve for this patient, the pain stimulation from saphenous nerve may also contributed to the uselessness of anesthetics.

This acute pain of ACS remained between moderate to severe degree and presumably is ischemic pain rather than surgical pain. This phenomenon is in accordance with other case reports in literature [10-12]. This may due to the different mechanism for surgical pain and ischemic pain. Although the mechanism of ischemic pain remains to be further discerned, there is a distinction in character and severity of pain. First, the nociceptors activation of ischemic pain is probably similar to sympathetic nerve system [12,13]. Second, there is some evidence that surgical pain is and ischemic pain may be mediated through different nerve fibers. Surgical pain may be primarily through C-fibers and Aδ-fibers while A-β fibers may play a bigger role in ischemic pain. Third, as shown in Frolich et al. [14], functional magnetic resonance imaging (fMRI) study, cold and heat pain changed the regional cerebral blood flow in brain cortical areas whereas ischemic pain caused a decrease in gray matter blood flow in brain. Therefore, regional anesthesia is likely not sufficient to eliminate ischemic pain while it is sufficient to alleviate surgical pain [15]. Dense local anesthetic blocks may influence the diagnosis of compartment syndrome without invasive pressure monitoring [16]. However, diluted concentrations of local anesthetic with or without systemic opioid administration can avoid dense motor and sensory blocks and does not influence the timely diagnosis of ACS [17]. Although whether epidural analgesia is associated with delay of ACS diagnosis is controversial, most consider that continuous peripheral nerve block delivered and titrated in a diluted concentration could avoid masking the intense pain of a developing compartment syndrome. High concentrations like 0.5% Bupivacaine infused by catheter in patients at risk of ACS should be avoided [2]. Notably, no matter which type of analgesia is used, a high index of clinical suspicion, ongoing assessment of patients and compartment pressure measurements are essential for early diagnosis.

**Conclusion**

In our case, continuous sciatic nerve block with 0.1% bupivacaine in patients at risk for acute compartment syndrome did not mask the severe pain caused by ACS and cause delay in diagnosis of ACS. On the contrary, with highly index of clinical suspicion and ongoing assessment, sudden loss of efficacy of continuous nerve block with diluted bupivacaine after extremity surgery is a potential alarm of compartment syndrome in a pediatric patient.

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This case report was prepared according to the CARE guidelines [18].

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**References**

1. von Keudell AG, Weaver MJ, Appleton PT, Bae DS, Dyer GSM, et al. (2015) Diagnosis and treatment of acute extremity compartment syndrome. The Lancet 386(10000): 1299-1310.

2. Aguirre JA, Gresch D, Popovici A, Bernhard J, Borgeat A (2013) Case scenario: compartment syndrome of the forearm in patient with an infraclavicular catheter: breakthrough pain as indicator. Anesthesiology 118(5): 1198-1205.
3. Dewangan M, Khare MK, Mishra S, Marhual JC (2016) Acute Exercise-Induced Compartment Syndrome of the Leg: Don't Miss It. J Clin Diagn Res 10(2): PD03–PD04.

4. Schmidt AH (2016) Acute Compartment Syndrome. Orthop Clin North Am 47: 517-525.

5. Hargens AR, Romine JS, Sipe JC, Evans KL, Mubarak SJ et al. (1979) Peripheral nerve-conduction block by high muscle-compartment pressure. J Bone Joint Surg Am 61(2): 192-200.

6. Ulmer T (2002) The clinical diagnosis of compartment syndrome of the lower leg: are clinical findings predictive of the disorder? J Orthop Trauma 16(8): 572-577.

7. Bae DS, Kadiyala RK, Waters PM (2001) Acute compartment syndrome in children: contemporary diagnosis, treatment, and outcome. J Pediatr Orthop 21(5): 680-688.

8. Saporito A, Petri GJ, Sturini E, Borgeat A, Aguirre JA (2014) Safety and effectiveness of bilateral continuous sciatic nerve block for bilateral orthopaedic foot surgery: a cohort study. Eur J Anaesthesiol 31(11): 620-625.

9. Fredrickson MJ, Kilfoyle DH (2009) Neurological complication analysis of 1000 ultrasound guided peripheral nerve blocks for elective orthopaedic surgery: a prospective study. Anaesthesia 64(8): 836-844.

10. Munk-Andersen H, Laustrup TK (2013) Compartment syndrome diagnosed in due time by breakthrough pain despite continuous peripheral nerve block. Acta Anaesthesiol Scand 57(10): 1328-1330.

11. Ganeshan RM, Mamoowala N, Ward M, Sochart D (2015) Acute compartment syndrome risk in fracture fixation with regional blocks. BMJ Case Rep 2015.

12. Cometa MA, Esch AT, Boezaart AP (2011) Did continuous femoral and sciatic nerve block obscure the diagnosis or delay the treatment of acute lower leg compartment syndrome? A case report. Pain Med 12(5): 823-828.

13. Sanni A, Hamid A, Dunning J (2005) Is sympathectomy of benefit in critical leg ischaemia not amenable to revascularisation? Interact Cardiovasc Thorac Surg 4(5): 478-483.

14. Frolich MA, Deshpande H, Ness T, Deutsch G (2012) Quantitative changes in regional cerebral blood flow induced by cold, heat and ischemic pain: a continuous arterial spin labeling study. Anesthesiology 117(4): 857-867.

15. Kucera TJ, Boezaart AP (2014) Regional anesthesia does not consistently block ischemic pain: two further cases and a review of the literature. Pain Med 15(2): 316-319.

16. Mar GJ, Barrington MJ, McGuirk BR (2009) Acute compartment syndrome of the lower limb and the effect of postoperative analgesia on diagnosis. Br J Anaesth 102(1): 3-11.

17. Garner MR, Taylor SA, Gausden E, Lyden JP (2014) Compartment syndrome: diagnosis, management, and unique concerns in the twenty-first century. HSS J 10(2): 143-152.

18. Gagnier JJ, Kienle G, Altman DG, Moher D, Sox H, et al. (2013) The CARE guidelines: consensus-based clinical case reporting guideline development. Headache 53(10): 1541-1547.