Abstract: Opinions diverge as to whether or not regional anaesthesia delays the diagnosis of evolving acute compartment syndrome. Withholding regional anaesthesia from patients with painful orthopaedic injuries may be ethically unacceptable, however. In this report, we describe a case of acute compartment syndrome in a 4-year old child who underwent resection of a forearm osteochondroma. Analgesia was satisfactory during the first post-operative night, but the child later complained of pain despite an effective infra-clavicular block. Motor function and sensibility were disturbed and the fingers were swollen. The forearm cast was removed as it was suspected to be causing external compression. Pain disappeared while motor function and sensation recovered. The child was discharged without any complications.

Key words: Peripheral nerve block, paediatric, breakthrough pain, compartment syndrome.

Introduction

After limb surgery or orthopaedic trauma, the inflammatory response, oedema, ischemia-reperfusion syndrome or haemorrhage can cause elevated pressure within a confined facial compartment, resulting in diminished or compromised microcirculation and decreased physiological function of all tissues involved. The inelastic fibrous facial layers that separate the muscle groups prevent expansion of the soft tissues giving rise to increased intra-compartmental pressure. In turn, this increased pressure further compromises tissue perfusion, resulting in a vicious cycle that leads to further ischaemia and potential necrosis. These events are clinically observed as an acute compartment syndrome (ACS) and are due directly to increased pressure within the anatomical compartment. External compression from circumferential casts, tight bandages or dressings may also be responsible for an ACS (1, 2). These types of ACS can be masked by Regional Anaesthesia (RA) techniques.

Increased pressure in a confined muscle compartment increases the venous pressure and reduces perfusion, causing ischaemia, pain, oedema, appearance of neurological symptoms, dysfunction of all tissues involved and finally necrosis with irreversible damage (3). The diagnosis is made when an identifiable risk factor is present and there are clinical symptoms such as disproportionate pain, paraesthesia or hypo-aesthesia in combination with examination findings such as pain during passive stretching and a tense, swollen muscle compartment. It is well accepted that RA has a role in pain relief after a trauma or after extensive orthopaedic surgery. However, does RA delay or mask the diagnosis of an evolving ACS? In recent years, increasing numbers of case reports have shown the superiority of peripheral nerve block (PNB) in pain relief following orthopaedic surgery or limb trauma, without masking the diagnosis of ACS (1, 2, 4).

We present a case of ACS during PNB, where close follow-up and adequate clinical examination enabled rapid therapeutic intervention. Diagnosis in this young child was challenging due to difficulties in obtaining a typical subjective history.

Case report

Written informed consent for publication was obtained from the child’s mother.

A 4-year-old boy weighing 17kg was scheduled for the resection of an extensive osteochondroma
was given at induction to ensure that the child would no longer be curarized when the surgeon performed neuro-stimulation during dissection of the wrist.

After an uneventful operation an open cast was placed and an ultrasound guided (Flex Focus BK Medical, linear probe 18MHz) infra-clavicular block was performed using a 19G × 50 mm needle (SonoLong Sono, Pajunk). A peripheral nerve stimulator was used with low current stimulation (0.28 mA) to minimize the risk of an intra-neural injection.

The probe was placed in a sagittal infra-clavicular plane. The needle was introduced in plane and the tip positioned inferior to the medial and posterior cords. As no muscular response was observed with low current stimulation, implying the needle tip was probably not in the cord, 5 mL of saline was injected to create space for the introduction of a PlexoLong catheter (Pajunk). Real-time catheter placement using ultrasound, followed by an injection of saline, confirmed the correct localization of the catheter below the 3 cords. Following emergence and recovery from general anaesthesia, neurological examination of the child’s hand showed normal movement of his fingers.

A bolus of levobupivacaine (0.125%, 7 mL) was injected through the catheter and a PCA pump (parent controlled analgesia) installed. The infusion parameters were regulated as follows: continuous infusion of 3 mL/h with a demand dose of 3mL and lock-out interval of 120 minutes. Analgesia was supplemented with a bolus of 300 mg paracetamol intravenously every 6 hours.

In our hospital, pain in children between the ages of 4 and 8 years is assessed using the Wong Baker faces pain scale. The scale uses a series of ‘smileys’ to indicate the severity of pain and gives a score ranging from 0 to 5. When the child was fully awake the faces pain scale was 1 at rest and he could move the fingers. Evaluation of sensory function seemed to be normal, as far as the child’s answers were objective. The child was sent to the ward.

In the evening, the child was very comfortable, playing and eating, and had no pain with only the continuous infusion of local anaesthetic (3 mL/h) and the paracetamol 300 mg every 6 hours.

In the early morning of postoperative day 1, the child complained of pain in the distal part of his forearm. The faces pain scale score was 3, following which the ward nurse administered a supplemental intravenous injection of paracetamol 300 mg. As the pain did not diminish, the on call anaesthetist was summoned. Despite a well-functioning PCA pump, the anaesthetist gave the first supplemental injection.

Fig. 1. — Extensive osteochondroma of the distal Radius. Surgical resection was expected to be very painful in the postoperative period.

localized to the distal part of the left radius (Fig. 1). Following the application of routine monitoring and establishment of venous access, general anaesthesia was induced with fentanyl (40 µg) and propofol (60 mg). Tracheal intubation was facilitated by the administration of atracurium (8 mg) and the lungs ventilated with pressure/volume control. Anaesthesia was maintained with sevoflurane 1.8-2% in oxygen-enriched air. Ketorolac 8 mg and paracetamol 300 mg were administered just before tourniquet inflation and incision. The child received extra boluses of fentanyl during the operation to a total dose of 80 µg. Surgery was performed without a peripheral nerve block, because the orthopaedic surgeon wished to evaluate the neurological function of the hand immediately after awakening. The operation lasted for 2 hours. Due to the likelihood of severe post-operative pain, an infra-clavicular block was performed at the end of the surgical procedure. The block was placed when the child was still under general anaesthesia and the effect of the neuromuscular-blocking agent had dissipated. A low dose of neuromuscular blocking agent (atracurium 8 mg)
of 3 mL levobupivacaine 0.125% and reported his observation to the senior staff member who had performed the block and had evaluated the child immediately post-operatively.

Concomitant clinical findings were reduced motor function of the fingers, pain in the fingers and a red, swollen hand with disturbed capillary refill. The staff member who had been alerted by the sequence of events stopped the pump and asked the orthopaedic surgeon to give his clinical advice.

The breakthrough pain and the findings on clinical examination were sufficient to necessitate opening and removal of the cast. Subsequent examination of the arm showed no signs of internal compartment syndrome, indicating that the cast had been responsible for the clinical features. The cast was replaced but left open with a loose circumferential bandage. Pain in the fingers disappeared progressively over 30 minutes. The child was intensively observed for 3 hours with his arm elevated until motor and sensory function of the fingers were comparable to the day before, when the block was initiated. Pain, consistent with postoperative discomfort reappeared, without signs of compartment syndrome and the pump was restarted. On post-operative day 3, the pump was stopped and the catheter removed 12 hours later. Further analgesia was provided via the IV route. The child was able to leave hospital on post-operative day 4 without further complaints.

Clinical evaluation was satisfactory at follow-up, three weeks later.

DISCUSSION

Although injuries of the tibia or forearm are the most common risk factors in the young, any injury regardless of its location within the limbs can cause ACS (2, 4). For Walker et al. an ACS is the result of elevated pressure within a confined facial plane or compartment, finding his origin in a great variety of intrinsic and extrinsic causes. This elevation in pressure may originate in an anatomical compartment (intrinsic) after an injury, haemorrhage, ischemia-reperfusion or from external compression (casts, circumferential dressings, patient-positioning devices, burns). Whatever the reason, intrinsic or extrinsic, the rise in pressure will eventually exceed that in small venules, resulting in venous capillary hypertension, which decreases microcirculatory perfusion. Further rises in compartment pressure lead to occlusion of small arterioles, ischemia and ultimately necrosis (1).

Literature from the last decade suggests that practically every type of limb injury can be implicated in the development of ACS, even elective surgery for total knee replacement (2, 5). The question therefore arises as to whether regional anaesthesia should be used for limb surgery, otherwise leaving patients in pain for many days with the risks of prolonged rehabilitation, increased risks of developing chronic pain syndromes and increasing the costs for health care and society. In modern anaesthesia practice the treatment of postoperative pain is a basic human right and an essential part of perioperative care. It is important to point out that an intravenous analgesic regime can also mask an ACS. This suggests that the choice of analgesic technique per se is not a determining factor in masking or missing the diagnosis of ACS (6, 7).

Using a RA technique in the face of risk factors for ACS remains a controversial subject frequently debated among and between anaesthetists and orthopaedic surgeons. Recently, cases and reviews of ACS have been published including a paper on evidence-based case management (1, 2, 8). All of these have stated that a functioning PNB should not necessarily risk a delay in the diagnosis of ACS. This does not signify, however, that there are no published cases in which regional anaesthesia has masked such a diagnosis, although it remains debatable as to whether the analgesic technique as such was the reason for delay. Excessive dosing of analgesic medication, a lack of close follow-up, incomplete clinical examination and inadequate or delayed relief of the compartment pressure are likely explanations for a prolonged interval between development and diagnosis of an ACS with subsequent limb dysfunction (4).

The clinical signs and symptoms which are classically described as being suggestive of an ACS are excessive pain, pain remote from the site of injury, paresthesias, hypoaesthesia with reduced light touch and 2-point discrimination, pain on passive stretching and a tense, swollen muscle compartment. Invasive compartment pressure measurements can help in diagnosing an internal ACS.

In this 4-year-old child a PNB was used for analgesia after resection of an extensive osteochondroma. This procedure was expected to be very painful in the post-operative period. Assessing sensory function and pain in such a small child is quite challenging, especially in the presence of a well-functioning PNB. Neurological evaluation showed a difference in motor and sensory function compared between the days before and after initiation of the analgesic block. The fact that the child
was neither playing nor eating indicated that the pain was moderate to severe. In this case, the presence of a cast might have been sufficient to explain the clinical presentation, although the cast might also have masked an evolving internal ACS, making the features of tense, swollen soft tissues and pain on passive stretching difficult to recognise. As a consequence, removal of the cast was mandatory to allow correct evaluation of the muscle compartments. The presence of breakthrough pain with increased analgesic requirements, the changes in neurological findings and the red, swollen hand with disturbed capillary refill provided sufficient evidence to indicate removal of the cast. Escalating analgesic requirements may precede the classic signs of an ACS by several hours (9).

The main question remains as to whether a PNB may mask ischaemic pain and limit the clinician’s ability to make the diagnosis of ACS. Undoubtedly it may do so when high concentrations of local anaesthetics are used causing a dense block. However, patients do not require such a profound extremity block for post-surgical or post-traumatic pain relief. In most published cases, including the one presented here, the diagnosis of an evolving ACS in the presence of an effective PNB has been possible due to the use of low concentrations of local anaesthetics. This has facilitated the assessment of motor function and enabled the detection of breakthrough pain (1, 4, 10). A delay in diagnosis might arise due to the use of supplementary analgesic regimens such as administration of other pain relieving agents, or increasing the concentration of local anaesthetics, without examination of the involved limb. In this case, the breakthrough pain was certainly not enough to diagnose ACS. A high degree of suspicion, identification of patients at risk and a thorough physical examination are mandatory before increasing the dosing of local anaesthetics.

This implies that if a PNB is to be considered for post-operative analgesia for limb surgery or trauma, the anaesthetist should be aware of the possibility of ACS. At risk patients need to be identified, regular acute pain follow-up needs to be organized and only low dose local anaesthetics and adjuvants should be used. When in doubt, the anaesthetist, rather than a nurse from the Acute Pain Service, should examine the patient and ask for surgical advice before administering further analgesics. If regional anaesthetic techniques are to be used by anaesthetists, it is of paramount importance that the nursing staff, as well as the physician on the ward, are aware of the clinical presentation and management of ACS.

In conclusion, ACS can occur and despite a continuous PNB with adequate surgical pain relief, an ACS can be diagnosed through identification of the patient at risk, the presence of breakthrough pain, maintaining a high level of suspicion and performing a thorough clinical assessment. The key factors allowing detection of ACS are the use of low dose infusions of LA and in the presence of breakthrough pain, the avoidance of supplementary analgesia before clinical examination.

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References