The use of multimodal analgesia in the management of suspected extremity compartment syndrome in the pelvic limb of a horse

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Keywords: extremity compartment syndrome (ECS); horse; ketamine; paracetamol; epidural; opioids; multimodal analgesia

Summary

A 12 year old Thoroughbred cross Dartmoor mare was referred to the clinic with marked lameness and swelling involving the left stifle region. There was poor initial response to medical management and therefore arthroscopic examination of the stifle joint was performed under general anaesthesia. Following surgery, the lameness and swelling worsened and extremity compartment syndrome was suspected. A multimodal analgesia protocol was instigated to provide adequate analgesia and improved mobility, aiding the use of physical therapy in resolving the swelling. This report demonstrates the successful combination of non-steroidal anti-inflammatories, paracetamol, ketamine infusion and epidural opioid administration to manage the clinical signs. The mare was discharged from hospital after 15 days and at short-term (three months) follow-up, there was no reported residual swelling or lameness.

Introduction

Compartment syndrome occurs when increased pressure within a tissue in an enclosed space compromises visceral and neuromuscular function within that area (Rorabeck and McGee, 1990). This increased tissue pressure restricts local perfusion, leading to physiologic dysfunction of cells, including cytokine release and oxygen free radical formation, ultimately resulting in cell death (Vegar-Brozovic and Stoic-Brezak, 2006). Compartment syndrome may occur directly as a result of the disease process itself (haemorrhage, oedema, thrombosis) or iatrogenically as a result of external compression from improper surgical positioning, constrictive bandages or excessive fluid resuscitation (Nielsen and Whelan, 2012). Definitive diagnosis requires measurement of intra-compartmental pressure using a needle and manometer, wick catheter, slit catheter or non-invasive, near-infrared spectroscopy (Rorabeck and McGee, 1990, Garr et al., 1999, Nielsen and Whelan, 2012). In a

clinical setting however, these tools may not be available and therefore diagnosis is usually based on history and clinical signs consistent with compartment syndrome (Rorabeck and McGee, 1990, Nielsen and Whelan, 2012).

Compartment syndromes are well-recognised in human medicine and have been documented in the thorax, abdomen and limb extremities (Nielsen and Whelan, 2012). Skeletal muscle or extremity compartment syndrome (ECS) is the most widely recognised type in veterinary medicine and involves increases in pressure within fascial compartments surrounding limb muscles (Nielsen and Whelan, 2012). Literature in the veterinary field is mainly confined to case reports (Sullins et al., 1987, Dodman et al., 1988, Norman et al., 1989, Nelson et al., 2015) but intra-compartmental muscle pressures have been measured experimentally in horses (Lindsay et al., 1985, McDonell et al., 1985, Nielsen and Whelan, 2012). Clinical signs of ECS include severe pain disproportionate to that expected for the injuries sustained, paresis, tenseness of the limb and weak or absent pulses in the affected area (Rorabeck and McGee, 1990, Nielsen and Whelan, 2012). Effective management of associated pain is essential and treatment with surgical decompression via fasciotomy is often required (Bae et al., 2001). Development of lumbosacral radiculoplexopathy and complex regional pain syndrome secondary to gluteal compartment syndrome was recently reported in the human literature, highlighting the potential for progression to chronic pain syndromes in these cases (Lederman et al., 2016).

Femoral compartment syndrome secondary to intramuscular haemangiosarcoma has been described in two dogs (Bar-Am et al., 2006, Radke et al., 2006). There has also been a report of acute ECS development in the stifle region of a Holstein cow following biopsy of an intramuscular haemangiosarcoma (Vogel et al., 2012). In equine medicine, ECS is most commonly reported associated with post-anaesthetic myopathy, particularly in the gluteal and triceps muscles (Sullins et al., 1987, Dodman et al., 1988, Norman et al, 1989, Kobluk 1995),

but has been described in the forelimb antebrachial region of two horses secondary to trauma at pasture (Nelson et al., 2015).

To the authors' knowledge, this is the first case report of suspected ECS secondary to trauma in the pelvic limb of a horse and describes the pivotal role of multimodal pain management in the successful outcome of this case.

Case History

A 12 year old Thoroughbred cross Dartmoor mare was referred to the clinic following a three day history of swelling around the left stifle with progressively worsening lameness, secondary to a suspected traumatic episode. Treatment prior to referral had included 1.1mg/kg bwt flunixin (Finadyne)[1] intravenously (IV), 12mg/kg bwt procaine benzylpenicillin (Depocillin)[2] intramuscularly (IM) and 6.6mg/kg bwt gentamicin (Genta-Equine)[3] IV. No bony abnormalities were identified on radiography by the referring veterinary surgeon but synoviocentesis of the left femoropatellar joint yielded sanguinous fluid on two subsequent days.

Clinical Findings

On presentation, the mare was moderately lame (6/10, Stashak, 2002) on the left pelvic limb at walk with marked swelling in the craniolateral stifle region. Flexion, extension and abduction of the limb were tolerated. No wounds were present on examination and all other findings were within normal limits.

Ultrasonographic examination (Logiq S7 Expert)[4] revealed moderate fluid distension of the left femoropatellar joint and peri-articular subcutaneous swelling over the craniolateral stifle. Intra- and peri-articular fluid were of mixed echogenicity, consistent with haemarthrosis and peri-articular haemorrhage (Figure 1). No abnormalities were detected in

the patellar ligaments or collateral ligaments of the stifle. No abnormalities were found on evaluation of the medial and lateral femorotibial joints. Synoviocentesis of the left femoropatellar joint yielded a sanguinous sample consistent with haemarthrosis (Table 1).

Treatment

On admission to the clinic, medical management was continued (20mg/kg bwt procaine benzylpenicillin IM q. 12 h, 6.6mg/kg bwt gentamicin IV q. 24 h and 4.4mg/kg phenylbutazone (Equipalazone)[5] IV q. 12 h.) combined with walking in hand for 1-2 minutes three times a day. As no clinical improvement was evident after 24 hours, arthroscopic evaluation of the left femoropatellar joint was performed under general anaesthesia. At surgery, marked synovial inflammation was identified. After arthroscopic lavage with 20 litres of sterile, polyionic, isotonic crystalloid fluid (Aqupharm-11)[6], resection of reactive synovium was performed followed by medication of the femoropatellar joint with 0.44mg/kg bwt amikacin (Amikacin)[7] and 0.11mg/kg bwt bupivacaine (Marcain 0.5%)[8]. An extra-articular haematoma on the craniolateral stifle was also drained. Fluid aspirated from the haematoma and biopsies taken from the synovium were submitted for microbiological culture. Phenylbutazone 4.4mg/kg bwt IV q. 12 h was administered and antimicrobials were continued at previous doses whilst awaiting microbiological culture results.

Over the following 24 hours, the level of lameness significantly worsened (9/10) with marked subcutaneous oedema around the stifle extending dorsally into the inguinal area. The distal sutures of the portal incisions were removed to assist drainage (Figure 2) and cold packing and cold hosing of the affected area were commenced. In addition to phenylbutazone, 0.2mg/kg morphine (Morphine sulphate)[9] was administered IV q. 4 h and 20mg/kg paracetamol (Paracetamol)[10] was commenced orally (PO) q. 12 h.

The following day, there was a mild reduction in swelling around the stifle and associated area and 0.1mg/kg bwt dexamethasone (Dexadreson)[11] IV was administered. Haematological and clotting profiles were performed, revealing a reduction in prothrombin time (PT) (10 seconds (reference range 15-20s)) and activated partial thromboplastin time (APTT) (38 seconds (reference range 45-66s)). Haematology was otherwise unremarkable (Table 2).

Three days after arthroscopy, the mare's demeanour deteriorated and the horse was extremely reluctant to walk out of the stable. Due to the lack of improvement in swelling or degree of pain, development of extremity compartment syndrome (ECS) within the fascial planes around the femoropatellar joint was suspected. As dexamethasone resulted in no clinically appreciable improvement, this treatment was not repeated and as the mare was too painful for physical therapy to be effective, the analgesic protocol was modified. A lumbosacral epidural catheter (Perifix®ONE Pediatric Epidural Anesthesia Catheter)[12] was placed (Figure 3) and 60mg (0.13mg/kg bwt) preservative-free morphine (Morphine Sulphate)[13] combined with 50mg (0.11mg/kg bwt) preservative-free methadone (Physeptone)[14], with a total volume of 11mL, were administered via this route. Systemic opioid analgesia was discontinued and the epidural catheter left in situ. The mare was cross-tied in the stable to prevent premature catheter dislodgement and 60mg preservative-free morphine was administered epidurally q. 12 h. A ketamine (Anaestamine)[15] constant rate infusion (CRI) was also commenced at 0.8mg/kg bwt/hour.

No bacterial growth was observed in synovial fluid or synovium following extended culture (>48 hours). Antimicrobial medication was changed to 5mg/kg bwt trimethoprim and 25mg/kg bwt sulphadiazine (Trimediazine)[16] PO q. 12 h.

Over the next 24 hours, the mare's comfort level and demeanour significantly improved. The mare was able to ambulate effectively and the swelling around the stifle and inguinal regions had reduced. The ketamine CRI was discontinued after 24 hours and epidural morphine administration was reduced to 30mg q. 12 h four days after catheter placement, before further reduction in dose to 15mg q. 12 h after another two days. Phenylbutazone was reduced to 2.2mg/kg bwt q.12 h IV after five days of hospitalisation. Physical therapy was implemented, consisting of local tissue massage and in-hand walking three times daily for five to 10 minutes initially, increased to 15 minutes over the following week. Oral paracetamol was discontinued five days after epidural catheter placement. The epidural catheter was removed after eight days. At this point, the mare was almost sound on the left pelvic limb with marked reduction in swelling evident (Figure 4). Day to day pain assessment and analgesic management is summarised in Table 3.

Outcome

The mare was discharged 15 days after admission to the referral clinic receiving 2.2mg/kg bwt phenylbutazone (Butagran Equi)[17] PO q. 12 h and 5mg/kg bwt trimethoprim with 25mg/kg bwt sulphadiazine PO q. 12 h for four days. Further assessment of lameness and repeated ultrasonographic examination of the left femoropatellar joint were also recommended once the swelling had completely resolved.

At follow-up three months after hospital discharge, the referring veterinary surgeon reported that the horse was sound in the left pelvic limb at trot in a straight line with no evidence of lameness after flexion of the left pelvic limb. The owner declined further ultrasonographic examination.

Discussion

This report describes the successful management of a suspected case of ECS in the pelvic limb of a horse. Femoropatellar haemarthrosis and subcutaneous haematoma worsened after arthroscopic intervention, most likely due to additional subcutaneous oedema formation in the region. ECS has been described in humans and animals with few case reports detailed in the horse, although the fascial compartments of the equine proximal pelvic limb have been described in anatomical literature (Sisson, 1975). Most equine ECS case reports are related to post-anaesthetic myopathy with the gluteal and triceps muscles being most susceptible (Norman et al., 1989; Nielsen and Whelan 2009). Surgical decompression via fasciotomy (Bae et al., 2001) is often required in these cases but appropriate pain management is paramount for a successful outcome. Through the use of multi-modal pain management, physical therapy was able to be instigated, assisting in the resolution of the clinical signs in this case without fasciotomy.

It is possible that femoropatellar joint sepsis contributed to the pain associated with this case and antimicrobial medication varied over the course of treatment. Penicillin and gentamicin are considered first line treatments for synovial sepsis (British Equine Veterinary Association, 2015), had been administered prior to referral and were continued as synovial sepsis had not been ruled out on admission. Intra-articular amikacin was administered at surgery as an alternative aminoglycoside to gentamicin due to reported increases in gentamicin resistance in equine isolates (Johns and Adams, 2015) and synovial infection remained a possibility at this stage. Systemic antimicrobial treatment was continued at previous doses whilst awaiting microbiological culture results. As no bacterial growth was observed in synovial fluid or synovium following extended culture, it was considered unlikely that a septic process was a contributing factor and antimicrobial medication was changed to trimethoprim and sulphadiazine as a prophylactic measure against ascending

185 infection via the open arthroscopic portals. This combination is recommended as a first line
186 treatment for contaminated limb wounds (British Equine Veterinary Association, 2015).

187 Due to the extreme nature of the swelling post-operatively, the chronic nature of the
188 injury and the indication of initial haemarthrosis, the possibility of a coagulation disorder was
189 considered. Haematological and biochemical profiles were all within normal limits with the
190 exception of PT and APTT. Whilst reductions in PT and APTT due to premature activation of
191 clotting factors may result from technical errors with sample acquisition and storage (Song et
192 al., 2016), evidence in humans and dogs suggests that shortened PT and APTT may be
193 associated with hypercoagulability and increased risk of thrombosis (Lippi et al., 2010, Song
194 et al., 2016). Therefore, PT and APTT could have been measured on subsequent samples to
195 monitor these changes.

196 Compartment syndrome results in cellular hypoxia and necrosis through two proposed
197 mechanisms. The 'arteriovenous pressure gradient theory' describes how an increase in
198 venous pressure in a compartment reduces the arteriovenous pressure gradient and hence
199 reduces oxygen delivery to those tissues (Mars and Hadley, 1998). Ischaemia-reperfusion
200 injury also occurs where interstitial fluid pressure within a compartment initially rises above
201 capillary pressure, preventing perfusion of tissues in that compartment. Subsequent
202 reperfusion leads to production of reactive oxygen species in addition to reduced oxygen
203 delivery, resulting in a cycle of hypoxia, anaerobic metabolism and further vasoconstriction
204 which continues to damage cells (Matsen and Krugmire, 1978).

205 Compartment syndrome is associated with severe pain due to inflammation, increased
206 intra-compartmental pressure, ischaemic damage and tissue necrosis. Movement of the
207 affected limb can help to encourage venous blood flow and hence movement of fluid out of a
208 particular compartment. In this case, the severity of the pain and swelling precluded physical

209 therapy of the affected limb, hence augmenting the underlying condition. The key objective
210 was to modulate the associated pain thus allowing mobilisation of the limb, reduction in
211 swelling and hence return of function.

212 Administration of anti-inflammatories is beneficial in ischaemia-reperfusion injury
213 (McMicheal, 2004). Studies assessing the effect of non-steroidal anti-inflammatories
214 (NSAIDs) in experimentally induced ECS in rats and dogs demonstrated increased perfusion
215 to the affected compartment, decreased muscle necrosis and lower intra-compartmental
216 pressures (Dabby et al., 1998, Manjoo et al., 2010). Both phenylbutazone and flunixin have
217 been shown to reduce prostaglandin production in experimentally induced inflammation in
218 horses (Higgins et al., 1984, Lee and Higgins, 1984) and the efficacy of both drugs has been
219 demonstrated for musculoskeletal pain (Johnson et al., 1993, Hamm et al., 1997, Kallings et
220 al., 1999). Phenylbutazone was chosen in this case based on several factors including reduced
221 cost compared to flunixin and therefore anticipated improved owner compliance with
222 potential long term treatment. The degree of pain, potential requirement for systemic opioids,
223 ongoing NSAID treatment, dietary changes and stabling during hospitalisation are
224 additionally risk factors for developing colic (Senior et al., 2004, Williams et al. 2011,
225 Scherrer et al., 2016). Flunixin is more likely to mask the cardiovascular changes associated
226 with endotoxaemia, should this develop as a consequence, potentially delaying identification
227 and appropriate intervention (King and Gerring, 1989, Mair and Edwards, 1998). Newer
228 NSAIDs such as firocoxib could have been considered due to demonstrable efficacy in
229 reducing musculoskeletal pain and potentially improved safety profile with higher cyclo-
230 oxygenase-2 (COX-2) selectivity (Koene et al., 2010, Orsini et al., 2012). There is however
231 insufficient evidence for superior efficacy or safety of firocoxib compared to phenylbutazone
232 when used at recommended doses (Doucet et al., 2008).

Concurrent glucocorticoid and NSAID administration is contraindicated in drug datasheets due to potential increased risk of gastric ulceration (National Office of Animal Health, 2017). Experimental studies in dogs concluded that concurrent administration of NSAIDs and glucocorticoids increased the risk of developing gastric mucosal erosions observed via endoscopy and faecal occult blood measurement (Dow et al., 1990, Boston et al., 2003, Narita et al., 2007). Evidence supporting NSAID administration as a cause of equine glandular gastric ulceration syndrome (EGGUS) at a population level is weak (Sykes and Jokisalo, 2015). Although there is potentially an increased risk of EGGUS with concurrent NSAID and corticosteroid administration, the authors felt that this risk was outweighed by the benefit of a potential reduction in peri-articular swelling, and as colic was a concern due to the aforementioned factors, the horse was monitored closely for signs of abdominal pain.

Although not licensed in horses, oral paracetamol was added to the treatment protocol when clinical signs did not improve with phenylbutazone. There is a paucity of literature on the analgesic efficacy of paracetamol in horses but there is a report of its successful use as an adjunctive analgesic to phenylbutazone in a pony with acute laminitis (West et al., 2011). Mechanism of action is different from NSAIDs as analgesia is thought to be centrally mediated, involving both cannabinoid and serotonergic pathways (Oscier and Milner, 2009).

Ketamine is a widely used anaesthetic agent in horses but its use as an analgesic in horses is not commonly reported. Ketamine, an N-methyl-D-aspartate (NMDA) receptor antagonist, is believed to mediate analgesia by preventing transmission of pain stimuli and modulating pain perception. In particular, ketamine may prevent hyperalgesia and central pain sensitisation, preventing development of chronic pain (Koizuka et al., 2005, Latremoliere and Woolf, 2009). Reports demonstrating this benefit in horses are however limited to pain management in chronic laminitis (Jones et al., 2007, Muir, 2010). Additional

potential benefits of ketamine that have been demonstrated in rodent and human experimental studies include potentiation of opioid analgesia as well as reduction of opioid tolerance and side effects (Inturrisi, 1994, Lauline et al., 2002, Shulte et al., 2004, Zhang et al., 2009), but these effects have not been demonstrated in horses. It has been suggested that ketamine infusion rates ranging from 0.8 to 1.5 mg/kg bwt/hour in horses are likely to result in analgesia whilst reducing central sensitisation and hyperalgesia (Fielding et al., 2006, Muir, 2010). As chronic pain is a reported consequence of compartment syndrome in humans (Lederman et al., 2016), in the authors' opinion it was logical to include ketamine in the analgesic protocol. Lameness dramatically reduced following 12 hours of ketamine infusion at 0.8mg/kg bwt/hour in this case, although concurrent epidural opioid administration precluded the ability to determine the analgesic effect of each drug individually.

Whilst the analgesic efficacy of opioids, particularly mu receptor agonists, in humans and other veterinary species is well reported, there are few studies that convincingly demonstrate the efficacy of opioid analgesics in horses. Inconsistent results and discrepancies exist between experimental and clinical studies (Lowe, 1978, Kamerling et al., 1985, Brunson and Majors, 1987, Kamerling et al., 1988, Bennett and Steffey, 2002). Intra-articular morphine could have been considered during arthroscopy in this case as this has been shown to reduce lameness scores in horses with experimentally induced synovitis compared to systemic morphine (Lindegaard et al., 2010). However, the authors considered the administration of systemic and epidural opioids pre-operatively in addition to intra-articular bupivacaine intra-operatively as provision of sufficient analgesia. Studies investigating epidural opioid administration have provided some of the most convincing evidence supporting use of opioids in providing analgesia in horses (Valverde et al., 1990, Robinson, 1994, Bennett and Steffey, 2002, van Loon et al., 2012). Epidural opioid administration can produce segmental analgesia, resulting in a higher local concentration with longer analgesic

duration as well as fewer CNS and cardiorespiratory side effects compared to systemic opioid administration (Natalini and Robinson, 2000, Torske and Dyson, 2000). Lipid solubility of opioids can affect onset and duration of analgesia when administered into the epidural space. Relatively hydrophilic compounds such as morphine have a slow onset of action but longer duration of action than more lipophilic agents such as methadone and fentanyl (Cousins and Mather, 1984, Natalini and Driessen, 2007). It is for this reason that methadone and morphine were administered concurrently following epidural catheter placement in this case. Methadone provides rapid onset of analgesia, whilst duration is enhanced with the inclusion of morphine (Olbrich and Mosing, 2003, Martin-Flores et al., 2014). Epidural administration of opioids was considered likely to be of benefit in this case to target the source of pain in the left pelvic limb, to allow a more convenient dosing interval of 12 hours instead of four hours with systemic morphine and to reduce reported systemic side effects associated with opioid usage in horses (Martin-Flores et al., 2014). Given the increasing severity of lameness and poor response to systemic opioids post-operatively, epidural opioid administration could have been considered earlier in this case. Placement of an epidural catheter facilitates regular opioid administration without the need to perform repeated needle punctures. Complications associated with the use of epidural catheters in horses include premature dislodgement, obstruction, leakage, inflammation around the catheter site, generalised muscle tremors, ataxia, pruritus and epidural steatitis with cauda equina neuritis symptoms (Martin et al., 2003, Robinson and Natalini, 2002, Steblaj et al., 2013). The horse in this case was cross-tied after epidural catheter placement to reduce risk of dislodgement. Lameness dramatically reduced and mobility improved within 12 hours of epidural opioid administration but simultaneous commencement of epidural opioids and ketamine CRI made it difficult to assess which was most beneficial or whether there was a synergistic effect.

Assessment of pain in equids can be challenging and there is no universally accepted or validated pain scale in horses. Numerous pain scales have been designed to try to quantitatively assess pain due to various pathologies (Pritchett et al., 2003, Bussières et al., 2008, Lindegaard et al., 2010, Wagner, 2010, van Loon and van Dierendonck, 2015). Lameness scoring (Stashak, 2002) was performed in this case as an indicator of improved mobility and hence response to analgesia but recent research has focused on assessment of facial expression to interpret pain (Dalla Costa et al., 2014, Gleerup et al., 2015, Dalla Costa et al., 2016). 'Low' positioning of ears and a tense stare were identified in this case, which were considered indicative of pain (Gleerup et al. 2015). Pain was assessed largely subjectively in this case and was managed accordingly although it would have been more advantageous to adopt a more structured and consistent method of assessing pain to monitor progress and response to analgesic medication more quantitatively. Palpation of the swollen area could have been performed to determine how the mare reacted. This can be assessed more objectively with the use of pressure algometers or von Frey filaments but these are not readily available in a clinical setting (Gleerup and Lindegaard, 2016). An Equine Pain Scale incorporating pertinent findings from previous studies which is quick and relatively easy to perform in a clinical setting has recently been proposed (Gleerup and Lindegaard, 2016). This could therefore be considered in similar cases in the future.

In conclusion, provision of multimodal analgesia played a pivotal role in the management of a suspected case of ECS. Reduction in pain allowed the mare to be walked out of the stable regularly, assisting in the resolution of swelling and leading to marked clinical improvement over a short period. Short-term outcome was favourable in this case but long-term follow up of this and similar cases in horses is recommended to identify any sequelae such as development of chronic pain, muscle contracture and sensory neuropathy as

reported in humans and dogs (Taylor and Tangner, 2007, Frink et al., 2010, Lederman et al., 2016).

Manufacturers' addresses

[1] MSD Animal Health, Walton, Milton Keynes, Buckinghamshire, UK

[2] MSD Animal Health, Walton, Milton Keynes, Buckinghamshire, UK

[3] Dechra Veterinary Products, Hadnall, Shrewsbury, Shropshire, UK

[4] GE Healthcare, Chalfont St. Giles, Buckinghamshire, UK

[5] Dechra Veterinary Products, Hadnall, Shrewsbury, Shropshire, UK

[6] Animalcare Ltd, York, Yorkshire, UK

[7] Hospira UK, Hurley, Maidenhead, Berkshire, UK

[8] AstraZeneca UK Ltd, Luton, Bedfordshire, UK

[9]Wockhardt UK, Wrexham, North Wales, UK

[10] Zentiva UK, Guildford, Surrey, UK

[11] MSD Animal Health, Walton, Milton Keynes, Buckinghamshire, UK

[12] B Braun Medical Ltd, Sheffield, Yorkshire, UK

[13] Martindale Pharmaceuticals, Romford, Essex, UK

[14] Martindale Pharmaceuticals, Romford, Essex, UK

[15] Animalcare Ltd, York, Yorkshire, UK

[16] Vetoquinol UK Ltd, Buckingham, Buckinghamshire, UK

[17] Bimeda, Llangefni, Anglesey, UK

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606 **Table 1**

607 **Synovial fluid analysis on day of presentation at referral clinic**

Parameter	Result	Reference Interval	Units
Total nucleated cell count	12.9	<1	$x10^9$/L
Lymphocytes	74.8	<20	%
Monocytes and macrophages	2.3	>80	%
Granulocytes	22.9	<10	%
Red blood cells	2.42	<1	$x10^{12}$/L
Total protein	78	<25	g/L
Microbiological culture	No bacterial growth after 48 hours	N/A	N/A

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610 **Table 2**

611 **Haematology results two days after arthroscopy**

Parameter	Result	Reference Interval	Units
White blood cells	6.22	4.3-14.8	$x10^9/L$
Red blood cells	7.03	7.2-12	$x10^{12}/L$
Haemoglobin	12.7	11.6-18.9	g/dL
Haematocrit	35	31-50	%
MCV	49.8	35.7-53.9	fL
MCH	18.1	11.9-20.3	pg
MCHC	36.3	35-38.2	g/dL
Platelets	116	69.9-250.8	$x10^9/L$
Neutrophils	4.45	2.2-8.1	$x10^9/L$
Lymphocytes	1.39	1.7-5.8	$x10^9/L$
Monocytes	0.22	0-1	$x10^9/L$
Eosinophils	0.14	0-0.8	$x10^9/L$
Basophils	0.01	0-0.3	$x10^9/L$
PT	10	16-20	seconds
APTT	38	45-66	seconds
Fibrinogen	242	100-400	mg/dL

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613

614 **Table 3**

615 **Day to day pain evaluation and analgesic management during hospitalisation**

Day(s) since admission to hospital	Pain evaluation	Analgesic management
0	6/10 lame left pelvic limb (LPL)	• 4.4mg/kg bwt phenylbutazone IV q.12h
1 (arthroscopy)	6/10 lame LPL	• Intercoccygeal epidural with 60mg morphine and 50mg methadone • 50mg intra-articular bupivacaine • 4.4mg/kg bwt phenylbutazone IV q.12h
2	9/10 lame LPL Marked peri-articular swelling around left stifle	• 4.4mg/kg bwt phenylbutazone IV q.12h • 0.2mg/kg bwt morphine IV q.4h • 20mg/kg bwt paracetamol PO q.12h • Distal portal sutures removed and cold packing/hosing commenced
3	8/10 lame LPL Only mild reduction in peri-articular swelling	• 4.4mg/kg bwt phenylbutazone IV q.12h • 20mg/kg bwt paracetamol PO q.12h • 0.2mg/kg bwt morphine IV q.4h • 0.1mg/kg bwt dexamethasone IV • Cold packing and hosing continued
4	9/10 lame LPL Low head carriage and 'low' ear position with tense face.	• Lumbosacral epidural catheter placed - 60mg morphine and 50mg methadone administered followed by 60mg

	Extremely reluctant to walk out of stable	morphine q.12h • Ketamine CRI at 0.8mg/kg bwt/hour • 4.4mg/kg bwt phenylbutazone IV q.12h • 20mg/kg bwt paracetamol PO q.12h
5	3/10 lame LPL Marked improvement in demeanour - normal head carriage and ear position	• 2.2mg/kg bwt phenylbutazone IV q.12h • 20mg/kg bwt paracetamol PO q.12h • 60mg epidural morphine q.12h • Ketamine CRI discontinued overnight (after 24 hours)
6	5/10 lame LPL Demeanour and facial expression normal Peri-articular swelling reduced	• 2.2mg/kg bwt phenylbutazone PO q.12h • 20mg/kg bwt paracetamol PO q.12h • 60mg epidural morphine q.12h • Physical therapy commenced - local massage and 5 minutes in-hand walking q.8h
7	4/10 lame LPL Tolerating physical therapy well	• 2.2mg/kg bwt phenylbutazone PO q.12h • 20mg/kg bwt paracetamol PO q.12h • 60mg epidural morphine q.12h • In-hand walking increased to 10 minutes q.8h
8	3/10 lame LPL	• 2.2mg/kg bwt phenylbutazone PO q.12h • 20mg/kg bwt paracetamol PO q.12h • 30mg epidural morphine q.12h • Physical therapy continued

9	2/10 lame LPL	• 2.2mg/kg bwt phenylbutazone PO q.12h • 30mg epidural morphine q.12h • In-hand walking increased to 15 minutes q.8h
10	2/10 lame LPL	• 2.2mg/kg bwt phenylbutazone PO q.12h • 15mg epidural morphine q.12h
11	1/10 lame LPL	• 2.2mg/kg bwt phenylbutazone PO q.12h • Epidural catheter removed
12-15	1/10 lame LPL	• 2.2mg/kg bwt phenylbutazone PO q.12h

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Figure 1. Longitudinal ultrasound image of the craniolateral aspect of the left stifle showing intra-(arrows) and peri-articular (arrowheads) material of mixed echogenicity consistent with haemarthrosis within the femoropatellar joint and peri-articular haemorrhage, respectively *(proximal is to the left)*.

Key: * = lateral patellar ligament; † - lateral trochlear ridge; ‡ = joint capsule of the femoropatellar joint

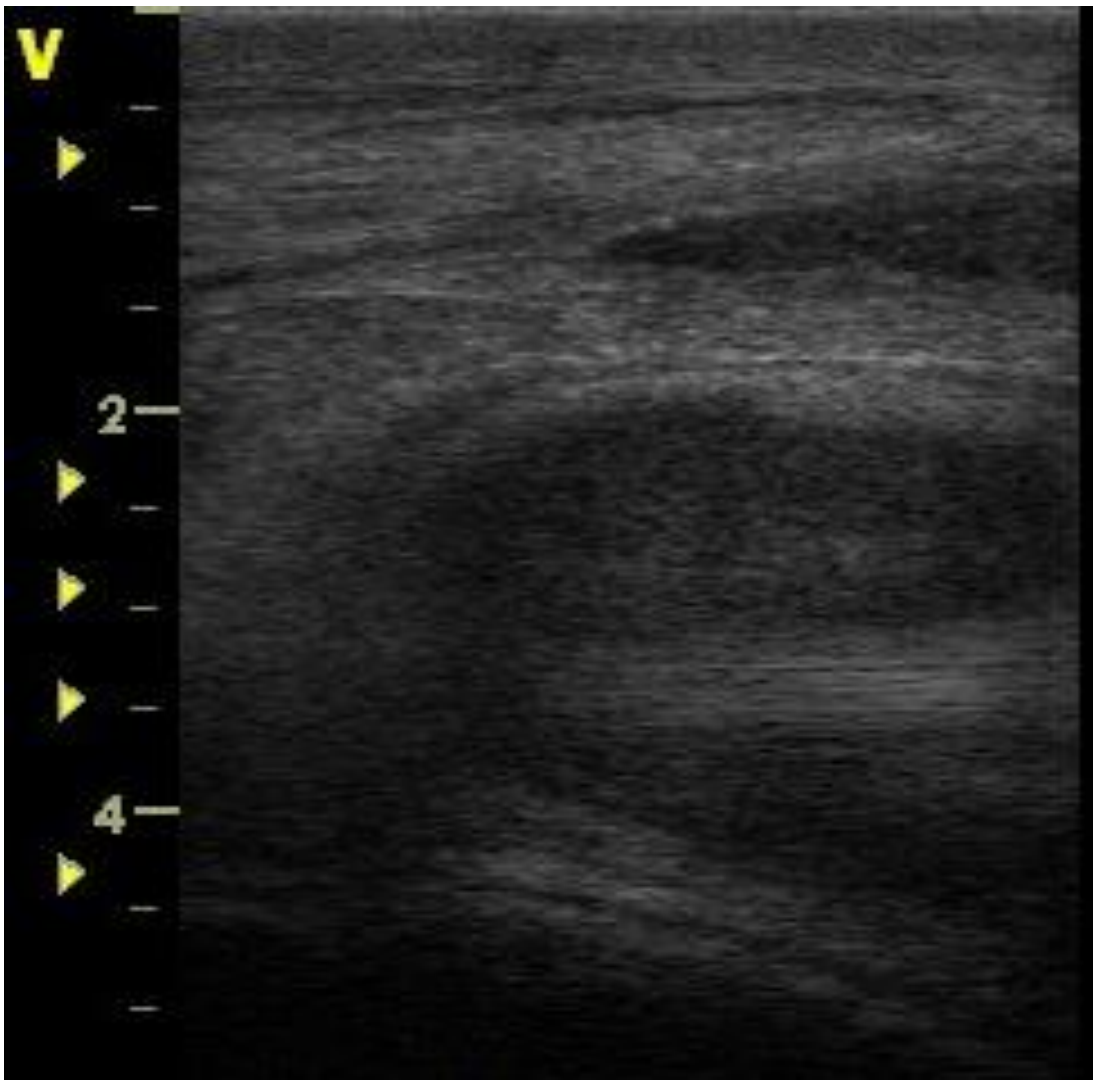

Figure 2. Photographs of the craniolateral (a) and caudal (b) aspects of the left stifle/thigh region 24 hours post-arthroscopy demonstrating marked swelling. Serosanguinous discharge is visible at arthroscopy portals after suture removal.

Figure 3. Indwelling lumbosacral epidural catheter

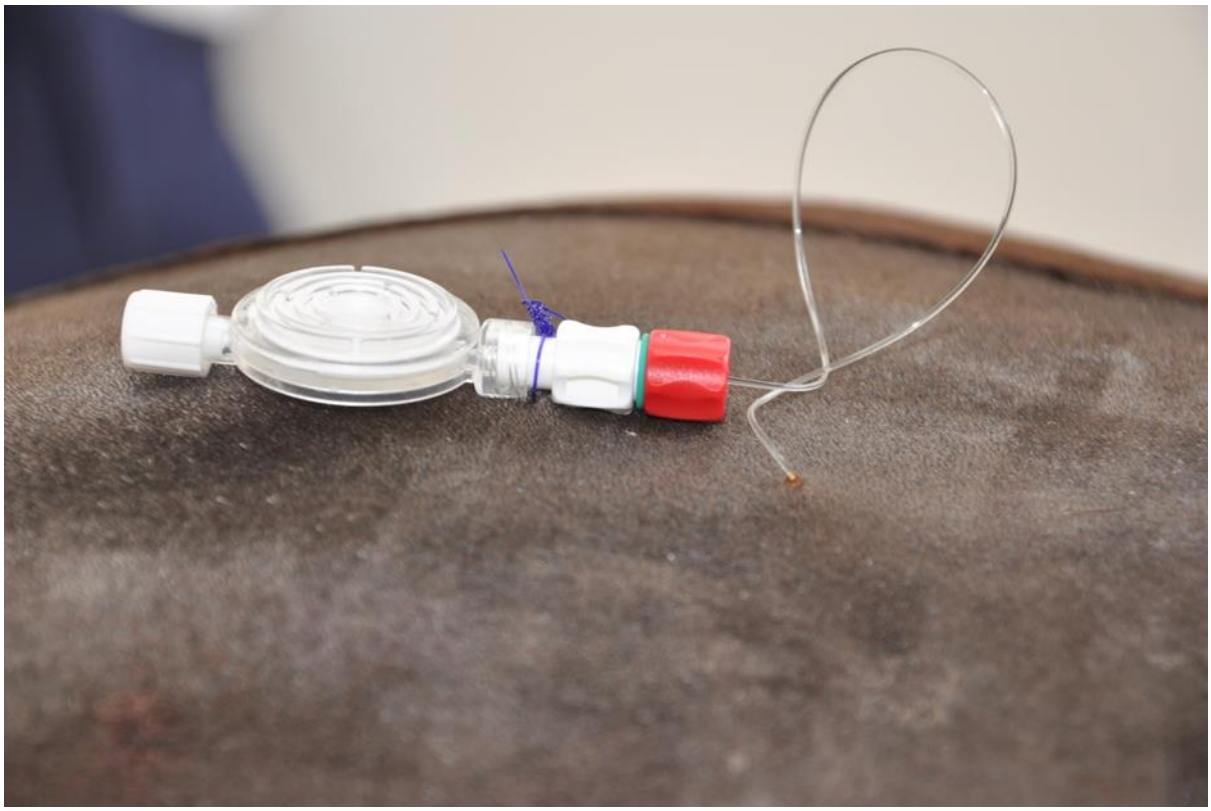

Figure 4. Photograph of caudal view of the pelvic limbs demonstrating marked reduction in swelling around left stifle/thigh after eight days of hospitalisation (immediately prior to epidural catheter removal).