Perioperative management of a paraplegic dog with blunt cardiac injury

Laura Perez-Klein, Lorenzo Novello, Simon Platt
Animal Health Trust, Centre for Small Animal Studies, Landwades Park, Kentford, Newmarket, Suffolk CB8 7UU, UK
E-mail: lauratapk@hotmail.com

Summary
A 6- and-a-half-year-old dog was referred for investigation of acute onset non-ambulatory paraplegia after a road traffic accident. Electrocardiography revealed a sustained ventricular tachycardia and arterial blood gases showed a mixed acid-base disorder. A lidocaine constant rate infusion was used to control the arrhythmia. An MRI scan revealed a traumatic disc herniation, and the dog underwent a right-sided hemilaminectomy. Twenty-four hours after the infusion was started, the dog showed clinical signs of lidocaine toxicity; lidocaine was discontinued and mexiletine was used as an antiarrhythmic. The arrhythmia resolved in 4 days and mexiletine was stopped. Twenty days after surgery the dog went home needing just sling support to walk.

Traumatic myocarditis has often been under diagnosed due to its delayed onset, and it may or may not be associated with cardiac arrhythmias. Cardiac arrhythmias may decrease cardiac output, and cause hypotension and impaired tissue perfusion. Prompt identification and treatment of cardiac dysrhythmias are essential in management of the trauma patient.

Case History
A six-year-old, intact male, chocolate Labrador weighting 30 kg was referred to the neurology service with a history of acute onset non-ambulatory paraplegia after being hit by a car. On physical examination the dog was depressed, tachypnoeic (45 rpm), tachycardic (220 bpm), the femoral pulse was weak, and mild thoracic and abdominal discomfort was present. On neurological examination the mental status was depressed and the animal was paraplegic on both pelvic limbs. Cranial nerve examination and spinal reflexes were normal and there was no evidence of deep pain sensation on either hind limb. Mild thoraco-lumbar pain was observed on para-spinal palpation. According to findings, the neuroanatomical localization was T3-L3. The main differential diagnoses were: traumatic (vertebral fracture, vertebral luxation or disc herniation) or vascular (ischemic myelopathy).

Following clinical examination the plan was to perform in-house pre-anaesthesia blood panel (including PCV, TP, BUN creatinine, blood gases and electrolytes), ECG, thoracic and abdominal radiographs, thoracolumbar MRI, and surgery if required. A venous blood sample was taken from the right jugular vein, and fluid therapy with Ringer's lactate was immediately started, and titrated according to patient response. The PCV, TP, BUN and creatinine levels were within normal limits as were calcium and chloride. The potassium was slightly low. The six lead ECG showed a sustained ventricular tachycardia (VT) with a heart rate of 220 bpm. At that stage the oxygen saturation, measured with a pulse oxymeter probe placed on an upper lip, was 100% in room air.

In order to rule out a pain-related tachycardia, to provide analgesia, and to keep the animal relaxed and still during radiographic examination, fentanyl was slowly injected intravenously to effect (0.003 mg kg⁻¹ total dose). Oxygen was started at 4 L/min via a flow by. An arterial catheter was placed in the dorsal pedal artery, and an arterial blood sample was taken. The arterial blood gas analysis showed normal pH (7.390), low CO₂ (4.24 kPa), high O₂ (23.7 Kpa) and low bicarbonate (20.7 mmol/L). As the dog was tachypnoeic and on oxygen, both high O₂ and low CO₂ were expected. As the pH was normal, the presence of a mixed acid-base disorder was suspected: respiratory alkalosis due to increased ventilation and metabolic acidosis that might be due to the shock. Ventricular tachycardia responded to a single lidocaine bolus (2 mg kg⁻¹) given intravenously. The thoracic radiographs were normal. As the abdominal radiographs revealed poor abdominal serosa detail, suggesting the presence of peritoneal effusion, and a narrowing of the T7-T8 disc space, an MRI scan was performed. General anaesthesia was induced with propofol IV slowly to effect (2.3 mg kg⁻¹ total dose) and the trachea was intubated with a 12 mm ID Red Rubber ET-tube. Then anaesthesia was maintained with isoflurane in oxygen and air, and the patient was mechanically ventilated using a low tidal volume (8 ml kg⁻¹, P₉₀peak 10 cmH₂O) and high respiratory rate (16 rpm) approach in order to maintain 4.6 KPa ETCO₂. During anaesthesia the patient was monitored via an MRI-compatible apparatus (ECG, Spo₂, IBP, inspired and expired oxygen, isoflurane and CO₂), and a sinus rhythm was present throughout the procedure. The MRI revealed a right-sided ventral extradural compression of the spinal cord at T7-T8 level, and was consistent with the diagnosis of intervertebral disc extrusion. The extrusion of the disc might be associated with prior vertebral subluxation, though at that time misalignment or evidence of instability were not present. A right-sided hemilaminectomy at T7-T8 was performed and the extruded disc material was removed from the
vertebral canal. During surgery anaesthesia was maintained with isoflurane in oxygen and intermittent positive pressure ventilation was provided. A constant rate infusion of fentanyl (0.07 mcg kg\(^{-1}\) min\(^{-1}\)) and low-dose ketamine (10 mcg kg\(^{-1}\) min\(^{-1}\)) were used to control pain. Lidocaine boluses were administered and a constant rate infusion was started (50 mcg kg\(^{-1}\) min\(^{-1}\)) when ventricular premature complexes (VPCs) and few episodes of VT recurred. During surgery monitoring included heart rate, a constant ECG, respiratory rate, pulse oxymetry, inspired and ET oxygen, CO\(_2\) and isoflurane, direct arterial blood pressure, and oesophageal temperature; they were stable over all the procedure. The surgery was straightforward, and the patient recovered from anaesthesia in the intensive care unit.

**Intensive Care Unit (ICU)**

Fluid therapy was maintained with lactate Ringer’s solution supplemented with potassium (20 mmol L\(^{-1}\)). Ranitidine and sucralfate were given as gut protectors, and an indwelling urinary catheter was placed. Ventilation, capillary refill time, mucous membrane colour, pulse quality and mentation were monitored every 30 minutes. ECG and invasive blood pressure (IBP) were monitored continuously, temperature and clinical monitoring every two hours, whereas urinary output and urinary specific gravity, electrolytes and arterial blood gases were monitored every four hours. The post surgical pain was monitored by reaction to paraspinal palpation and by adrenergic responses to pain sensation. The constant rate infusion of lidocaine (0.05 mg kg\(^{-1}\) min\(^{-1}\)) was continued and several boluses (no more than three boluses/hour up to 3 mg kg\(^{-1}\) hr\(^{-1}\) total dose) were administered to control VPCs and few episodes of VT. During the first twelve hours the dog was tachycardic, the pulse was weak, the blood pressure low (SBP 90, Mean 85, DBP 65) and the urinary output was slightly low suggesting a decreased cardiac output. However the few episodes of VT were effectively controlled by lidocaine boluses. The fluid therapy was increased to achieve a higher urinary output and higher blood pressure and the consequent increase on the cardiac output. The respiratory status and oxygenation was good, but the dog was still depressed and no deep pain sensation was observed on either hind limbs.

After twelve hours the VT was not responding to lidocaine anymore, and the dog started to show twitching of facial muscles suggestive of lidocaine toxicity. Lidocaine infusion was discontinued, and because no other intravenous antiarrhythmic drugs were available, oral mexiletine was started. Twelve hours later, the dog was alert, eating and drinking normally, the heart rate was back within the normal range (80 bpm) and some sinus rhythm was seen on the ECG. The blood pressure was back within normal range, and deep pain sensation was present on the right hind limb. However the dog started to be tachypnoeic. A blood sample was taken, and CBC (Complete Blood Count) and biochemistry were performed showing a mild anaemia, neutrophilic leucocytosis, thrombocytopenia, increased liver enzymes and very high levels of creatine kinase and cardiac troponin I. All these changes might be secondary to the trauma and the high level of cardiac troponin was consistent with the diagnosis of traumatic myocarditis. Eight hours later the dog started coughing and increased chest sounds were found on auscultation. An arterial blood gas showed PaO\(_2\) below normal range, PaCO\(_2\) below normal range, pH above normal range and bicarbonates within normal range suggesting poor oxygenation and respiratory alkalosis. Oxygen therapy was started. At that stage the ECG was normal and the deep pain sensation was present on both pelvic limbs. Thoracic radiography was performed and revealed a defined multifocal area of alveolar pattern that was suggestive of an inflammatory process or pneumonia, possibly due to recumbency. The oxygen therapy was maintained by nasal catheter over 3 days until the pH, oxygen, CO\(_2\) and oxygen saturation were stable in room air. The ECG was completely normal, no VT or VPCs were seen and the antiarrhythmic therapy was stopped. Fifteen days after surgery, the dog was still very paraparetic and incontinent.

**Outcome**

The dog went home, needing sling support to walk 20 days after surgery, and at that time normal voluntary urination was present. Two months later the dog was slightly paraparetic but walking and running without support. Urination and defecation were voluntary and no cardiovascular clinical signs were noted.

**Discussion**

Traumatic myocarditis is a term used to describe cardiac arrhythmias following a non-penetrating thoracic trauma. Myocardial contusion results from direct damage to the myocardium without traumatic involvement of the coronary arteries. Mechanisms of injury include thoracic compression, abdominal compression causing increased intrathoracic pressure, sudden acceleration or deceleration of the thorax causing the heart to slap against the chest wall, and acute neurological trauma. The pathogenesis of arrhythmias is unknown but may be multifactorial. Potential causes of this syndrome include blunt trauma of the heart with secondary cell damage and myocardial irritation, myocardial ischaemia due to hypoxia and acidosis associated with shock, and sympathetic over stimulation as a consequence of acute CNS injury causing myocardial necrosis. Myocardial damage may result in cardiac arrhythmias due to slow conduction time in damaged areas, re-entry of impulses in damaged areas, or automatic focus of activity due to injured Purkinje fibres. Pathologically, there is evidence of myocyte injury with cell necrosis, oedema and interstitial haemorrhage. Myocarditis is not usually present. The ECG is the most reliable indicator of myocardial contusion at the moment. It is important to remember that arrhythmias may not occur until 24-48 hours following injury. Reported arrhythmias in the dog include ventricular tachycardia, accelerated idioventricular rhythms, ventricular premature complexes, atrial fibrillation, sinus rhythm with bundle branch block, and atrioventricular block.

The question of when to treat the arrhythmias associated with trauma remains unclear. The decision depends on careful assessment of the electrocardiogram in the context of the patient’s haemodynamic status. Ventricular
arrhythmias responsible for clinical signs (depression, weakness, syncope, shock) should be treated with appropriate antiarrhythmic therapy. Antiarrhythmic drugs are not without complications, as they may be arrhythmogenic themselves and can also cause myocardial depression. Metabolic disturbances are common in the trauma patient and will worsen the arrhythmias seen. Because of that, underlying problems such as hypovolaemia, electrolyte disturbances, acid-base disorders, or pain should be addressed prior to considering antiarrhythmic therapy.

Ventricular arrhythmias can be treated by direct current counter shock (cardio version), programmed electrical stimulation, or, more commonly, antiarrhythmic drugs.

Lidocaine is often the first drug used to control ventricular arrhythmias associated with trauma in dogs. The most common toxic effect of lidocaine is central nervous system excitation. Agitation, disorientation, muscle twitches and nystagmus are the first signs seen which, depending on dose, may progress to generalized seizures and even unconsciousness and respiratory arrest. In the event of toxicity, lidocaine should be discontinued until the toxicity signs disappear; many of these signs are self-limiting due to the rapid redistribution of the drug from the brain to other tissues. If lidocaine is not successful, procainamide, mexiletine or propanolol are often administered. The current approach is empirical, although tempered with clinical rationale.

References


Gestione perioperatoria di un paziente paraplegico con trauma cardiaco concussivo

Laura Perez-Klein, Lorenzo Novello, Simon Platt

Animal Health Trust, Centre for Small Animal Studies, Landwades Park, Kentford, Newmarket, Suffolk CB8 7UU, UK
E-mail: lauretapk@hotmail.com

Riassunto

Un cane di 6 anni di età venne riferito per un approfondimento diagnostico per una paraplegia verificatasi in seguito ad un investimento stradale. L’elettrocardiogramma mostrò la presenza di tachicardia ventricolare, mentre l’emogasanalisi suggerì la presenza di un disordine misto dell’equilibrio acido-base. L’aritmia venne controllata con un’infusione continua di lidocaina. Una risonanza magnetica (RMN) mostrò la presenza di un’ernia discale di origine traumatica, e il cane venne interrotta e sostituita dalla somministrazione orale di mexiletina. Dopo 4 giorni di terapia l’aritmia si risolse e la paziente poté essere sostenuta durante la deambulazione.

La miocardite traumatica spesso non viene diagnosticata a causa della sua insorgenza tardiva, e sembra non essere sempre associata ad aritmie cardiache. Le aritmie cardiache possono ridurre la gittata cardiaca, oltre a poter causare ipotensione e ipoperfusione tissutale.

Caso clinico

Un cane Labrador, maschio, di 6 anni e mezzo e 30 kg di peso venne riferito al dipartimento di neurologia con un’anamnesi di insorgenza acuta di paraplegia in seguito ad incidente stradale. All’esame clinico il cane era depresso, tachipneico (45 rpm), tachicardico (220 bpm), con polso femorale debole e dolorabilità diffusa a torace ed addome.

All’esame neurologo si riscontrarono depressione del sensorio e plegia ad entrambi gli arti posteriori. L’esame dei nervi cranici e dei riflessi spinali non evidenziò nulla di anormale, mentre il dolore profondo risultò essere assente in entrambi gli arti posteriori. Venne anche evidenziata la presenza di dolore alla palpazione paraspinale in corrispondenza della zona toracolombare. In base ai riscontri clinici la lesione neurologica venne localizzata tra T3 e L3.

Nell’occasione le possibili diagnosi differenziali furono: lesione traumatica (frattura vertebrale, lussazione vertebrale o ernia discale) o lesione vascolare (mielopatia ischemica).

In seguito a tali riscontri alla visita clinica vennero programmati un esame del sangue preoperatorio (comprendente ematocritto, protein totali, BUN, creatininina, emogasanalisi ed elettroliti), un ECG, radiogrammi toracici e addominali, una risonanza magnetica (RMN) alla colonna toracolombare, ed eventualmente una chirurgia d’urgenza. Un campione di sangue venne prelevato dalla vena giugulare destra e subito dopo venne istituita una fluidoterapia con soluzione di Ringer lattato adattandone la velocità d’infusione alla risposta clinica del paziente.

I valori di ematocrito, proteine totali, BUN, creatinina, calcio e cloro ematici risultarono nell’intervallo di riferimento, mentre il potassio risultò essere leggermente diminuito. L’ECG a 6 derivazioni rivelò la presenza di tachicardia ventricolare (TV)
con frequenza di 220 bpm. La saturazione dell’emoglobina, misurata con un polsoossimetro con campionamento sulla lingua, risultò essere 100% con il paziente che respirava aria ambiente. A questo punto 0.003 mg kg⁻¹ di fentanil vennero somministrati lentamente per via endovenosa per controllare il dolore, per tranquillizzare il paziente durante l’esecuzione dei radiogrammi e per escludere il dolore come possibile causa della tachicardia. Si iniziò anche la somministrazione di ossigeno tramite flow-by con flusso di 4 L/min. Successivamente l’arteria metatarsale dorsale venne cateterizzata e iniziò anche la somministrazione di ossigeno tramite flow-by con flusso di 4 L/min. Successivamente l’arteria metatarsale dorsale venne cateterizzata e iniziò anche la somministrazione di ossigeno tramite flow-by per controllare il dolore, per eseguire anche la risonanza magnetica (RMN). L’anestesia veniva mantenuta con un’infusione continua di lidocaina (50 mcg kg⁻¹ min⁻¹) nel tentativo di controllare i numerosi complessi ventricolari prematuri ed alcuni episodi di tachicardia ventricolare. Durante la chirurgia vennero somministrati nel tentativo di controllare i numerosi complessi ventricolari prematuri ed alcuni episodi di tachicardia ventricolare. Durante la chirurgia vennero somministrati nel tentativo di controllare i numerosi complessi ventricolari prematuri ed alcuni episodi di tachicardia ventricolare. Durante la chirurgia vennero somministrati nel tentativo di controllare i numerosi complessi ventricolari prematuri ed alcuni episodi di tachicardia ventricolare.
Il cane venne dimesso a distanza di 20 giorni dall’intervento chirurgico, quando pur necessitando di essere sostenuto durante la deambulazione era tuttavia in grado di urinare spontaneamente. Due mesi più tardi il cane era lievemente paraparetico ma in grado di camminare e di correre senza alcun sostegno. A quell’epoca urinazione e defecazione risultarono essere normali, così come l’apparato cardiovascolare.

Discussione
Il termine “miocardite traumatica” viene utilizzato per descrivere le aritmie cardiache che si verificano in seguito ad un trauma toracico non penetrante o concussivo. La contusione miocardica è una lesione del solo miocardio, senza coinvolgimento delle arterie coronarie. I meccanismi responsabili della lesione sono riconducibili a compressione toracica, compressione addominale con conseguente aumento della pressione intratoracica, accelerazioni e decelerazioni violente che fanno sbattere il cuore sulla parete toracica, e traumi neurologici acuti.

La l’eziopatogenesi delle aritmie è sconosciuta ma potrebbe essere multifattoriale. Tra le possibili cause vanno incluse trauma cardiaco concussivo con danno miocardico secondario, ischemia miocardica da ipossia ed acidosi da shock e stimolazione simpatica eccessiva dovuta ad una lesione traumatica acuta al tessuto nervoso con conseguente necrosi miocardica. La lesione miocardica può dare origine ad una diminuzione nella velocità di conduzione delle aree interessate, fenomeni di rientro, o depolarizzazione spontanea legata a lesioni delle fibre del Purkinje. All’esame patologico si riscontrano lesioni delle cellule miocardiche con necrosi cellulare, edema ed emorragia interstiziale. Non si riscontra di solito miocardite. L’elettrocardiogramma è al momento l’indicatore più affidabile di contusione miocardica. Bisogna però ricordare che le aritmie possono avere insorgenza ritardata o debuttarono a distanza di 24-48 ore. Nel cane sono state riportate tachicardia ventricolare, ritmi idioventricolari ad alta frequenza, complessi ventricolari prematuri, fibrillazione atriale, bloch di branca e blocchi atrio-ventricolari.

Quando trattare le aritmie di origine traumatica è ancora poco chiaro. La decisione dipende dai segni elettroencefalografici e da come questi influenzano l’emodinamica. Le aritmie ventricolari che causano segni clinici (depressione del sensorio, debolezza, sincope, shock) dovrebbero essere trattate con agenti antiaritmici appropriati. Gli antiaritmici non sono infatti scelti da possibili effetti collaterali, dal momento che essi stessi possono essere essi stessi aritmogenici e possono causare depressione miocardica. Alterazioni metaboliche sono rarissime come comunque nei traumatizzati e possono precipitare le aritmie presenti in quel momento. Pertanto tutti i problemi componibili, come ipovolemia, alterazioni elettrolitiche, alterazioni dello stato acido-base e dolore, andrebbero trattati prima di istituire la terapia antiaritmica.

Le aritmie ventricolari possono essere trattate con terapia elettrica (cardioversione), pacing o, più semplicemente, con antiaritmici: in questo caso la lidocaina è di solito l’antiaritmico di prima scelta. L’effetto tossico più frequente della lidocaina è una stimolazione del sistema nervoso centrale: agitazione, disorientamento, tremori muscolari e nistagmo rappresentano i primi segni clinici e, a seconda della dose utilizzata, possono progredire fino a convulsioni, perdita della coscienza e arresto respiratorio. In caso di segni di tossicità sistemica la lidocaina andrebbe interrotta fino a che i segni clinici scompaiono, dal momento che la maggior parte di tali segni sono autolimitanti dal momento che il farmaco viene rapidamente ridistribuito dal cervello agli altri tessuti.

Se la lidocaina non è efficace nel controllare le aritmie di solito si utilizzano di procainamide, mexiletina o propranololo. L’approccio qui riportato può essere considerato empirico ma è supportato dall’evidenza clinica.

Bibliografia