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Abstract

Trauma represents a big challenge for emergency doctors both in human and veterinary medicine. There have been great advancements in trauma medicine in people and these often provide an inspiration for veterinary surgeons. However, the vast differences in the facilities and finances available in human and veterinary medicine make the approach to trauma in both fields quite different. This article, the second in a two-part series, describes how to manage canine trauma patients. The first part focused on the initial investigation of canine abdominal trauma (Humm and Cortellini 2017). Although this article will mention guidelines in people, these should not be automatically applied to dogs, especially when more relevant evidence for this species exists.

Introduction to the condition

ALTHOUGH this article focuses on abdominal trauma, this rarely occurs in isolation (Streeter and others 2009). Part 1 of this two-part series on canine abdominal trauma (Humm and Cortellini 2017) emphasised the importance of identifying dysfunction of all major body systems (cardiovascular, respiratory and neurological systems). Once these have been identified, treatment is essential and is prioritised over other, non-life threatening injuries. Up to 72 per cent of patients with trauma have thoracic injury (Simpson and others 2009); therefore, they need to be assessed for the presence of hypoxaemia and oxygen supplementation should be administered via flow-by, oxygen mask or nasal prongs or cannulas, if tolerated by the patient.

If there is evidence of hypoperfusion, fluid resuscitation is required. In most trauma causes, this will be secondary to hypovolaemia; therefore, fluid therapy is the most appropriate first step for resuscitation. Vascular access should be achieved as soon as possible, with minimal stress of the patient. Subcutaneous fluids should never be used for resuscitation purpose in hypotensive patients. The optimum fluid choice for resuscitation in the traumatic patient is a debated topic in both human and veterinary medicine. As a first-line treatment, isotonic crystalloids, such as lactated Ringer's or 0.9 per cent NaCl, are both thought appropriate. A bolus of 10 to 15 ml/kg over 15 minutes is usually effective in re-establishing normovolaemia, depending on the degree of haemorrhage. Repeated boluses can be given, up to a maximum of 90 ml/kg. After each bolus, clinical parameters indicative of improved cardiovascular function, such as pinker mucous membranes, reduced heart rate, improved pulse quality, and higher blood pressure, should be used to assess the response to fluid therapy and the need for additional boluses (Figs 1, 2).

Synthetic colloids, such as tetrastarch (Volulyte; Fresenius-Kabi) remain within the intravascular compartment for a longer period and so lower doses are required; for example, 5 ml/kg over 10 to 20 minutes up to a maximum of 20 ml/kg/day. Potential adverse effects of these fluids include dilutional coagulopathy, volume overload and possible colloid-induced kidney injury (Hayes and others 2016). Other injuries induced by the trauma, such as pulmonary contusions or pre-existing comorbidities (eg, cardiac disease), may mean a less aggressive fluid approach is indicated, and if traumatic brain injury is present the use of hypertonic saline may be indicated.

Analgesia is also an essential aspect of management of the trauma patient, not only for welfare reasons, but also because of the clinical implications of untreated pain. Pain may result in hypoventilation causing atelectasis and increase catecholamine release, with tachycardia and vasoconstriction leading to increased oxygen consumption and potentially development of cardiac arrhythmias. In patients with head trauma or severe shock, behavioural manifestations of pain, such as vocalisation and reaction to palpation, may not be detectable. In such situations analgesia should be administered if it is felt likely the injuries present would cause pain (which would be likely in most trauma cases). Regardless of the degree of trauma, the first-line analgesic should be an opioid.

Full μ-agonists, such as methadone, morphine or fentanyl given slowly intravenously, are an excellent first choice as they provide a margin for additional dose increments to further control pain. In addition, full μ-agonist can be antagonised with naloxone at any point. Buprenorphine, a partial μ-agonist, is not recommended as a first-line treatment due to its inability to provide further analgesia above a certain dose (ie, ceiling effect). Butorphanol, an agonist-antagonist, has good sedative but not analgesic properties and should not be used to control pain. Other classes of analgesics can be used as an adjunctive treatment for pain.

Ketamine may be used as additional analgesia with an initial bolus (0.1 to 1 mg/kg, administered intravenously) followed by constant rate infusion (0.1 mg/kg/hour). The action is exerted mainly by inhibition of the N-methyl-D-aspartate receptors and reduces central hyperalgesia (ie, wind-up phenomenon), while causing minimal cardiovascular and respiratory depression. Similarly, lidocaine can be used as a systemic analgesic with a constant rate infusion (2 to 3 mg/kg/hour), in addition to its anti-arrhythmic action. Lidocaine may also theoretically reduce the damage induced by ischaemia-reperfusion injury, although these effects have not been proven in dogs with trauma. While the use of corticosteroids is no longer recommended in any trauma patient, non-steroidal medications are excellent drugs for controlling the inflammation and pain through inhibition of COX-2 and can be given both enterally and parenterally, once the patient is considered cardiovascularly stable and well hydrated. However, the adverse effects associated with inhibition of COX-1 may induce gastrointestinal disease, acute kidney injury and thrombocytopathia, especially in patients with cardiovascular instability or pre-existing renal or gastrointestinal disease. Therefore, these drugs should not be given unless the patient has been stable for 24 to 48 hours.

Antimicrobials should only be administered in dogs with abdominal trauma if there is a clear indication such as bite wounds, septic peritonitis or a penetrating abdominal injury. In these

cases, early initiation of a broad-spectrum antimicrobial, such as clavulanate-potentiated amoxicillin is warranted. Cultures should always be taken and antimicrobials de-escalated once results guide the appropriate treatment.

Treatment/management options

In this section we will review the management of the most common emergency injuries secondary to abdominal trauma.

Haemoabdomen

In a study of 235 dogs involved in a traumatic episode, 50 per cent had an abdominal injury. The most common injury was haemoabdomen (23 per cent), with only a small percentage having abdominal wall rupture (5 per cent) or urinary tract rupture (3 per cent) (Simpson and others 2009). The most common sites of organ injuries causing haemoperitoneum are the liver, spleen and kidney. Unlike spontaneous haemoabdomen, surgical management is rarely required in dogs with trauma-induced haemoabdomen and appropriate medical management is often sufficient to stabilise the patient and allow the resolution of the haemorrhage. In a prospective canine trauma study, exploratory surgery was not performed in any of the 38 dogs with haemoabdomen (Boysen and others 2004). However, in this study two of three dogs that died had haemoperitoneum, leading to the speculation that the haemorrhage may have been one of the contributors to the cardiopulmonary arrest of these dogs. If the patient, despite an appropriate medical management, continues to show signs of recurrent hypovolaemia (such as tachycardia, hypotension and pale mucous membranes) with a progressive drop in peripheral packed cell volume (PCV) and an increase in the abdominal fluid as assessed by ultrasonography, surgery should be considered.

Aside from the tissue injury, coagulopathy in the trauma patient is an important contributing factor to ongoing bleeding. This condition, named acute traumatic coagulopathy (ATC), has been described in people involved in a traumatic episode, and is associated with an increased mortality (Palmer and Martin 2014).

ATC is thought to be triggered by tissue injury and hypoperfusion (Palmer and Martin 2014), but may be exacerbated by hypothermia, haemodilution and acidosis; therefore, these need to be corrected especially during the first 48 hours of hospitalisation by temperature control and judicious use of intravenous fluids (Spahn and others 2013). Several recent studies have tried to identify and typify this condition in dogs but to date no consistent pattern has been found.

The use of tranexamic acid, an antifibrinolytic medication, appears to reduce mortality in human trauma patients with significant ongoing bleeding (Shakur and others 2010); further trials are underway to clarify its effectiveness and safety. The use of antifibrinolytic medications, such as tranexamic acid or epsilon aminocaproic acid, has not been studied in dogs that have undergone trauma and the application of this research in people to canine patients is unclear so their use cannot be strongly recommended at this stage. Hypoperfusion may contribute to ATC, and considering the association between untreated shock, multiple

organ failure and death, resuscitation should be started immediately with crystalloids and synthetic colloids if their use is considered appropriate, as described above. However, the use of both crystalloids and synthetic colloids should be judicious, as experimental studies have shown high volume resuscitation has been associated with a higher degree of haemodilution, coagulation derangements and hypothermia.

In the situation of active, uncontrollable bleeding, hypotensive resuscitation, defined as targeted mean arterial pressure of 50 mmHg versus a standard normal target of 65 mmHg (Spahn and others 2013), has been considered in the attempt to avoid recurrence or exacerbation of haemorrhage, and reduce the amount of fluid administered and therefore the degree of dilutional coagulopathy. This resuscitative strategy is used in human trauma patients as a temporary solution in cases where haemostatic control is rapidly achieved (generally via surgical intervention) and is followed by full fluid resuscitation to re-establish an optimal perfusion (Spahn and others 2013). Because this strategy is associated with suboptimal tissue perfusion and may lead to multi-organ failure if protracted, it should not be used as an alternative form of medical management in the attempt to arrest the haemorrhage and avoid surgery.

In people, in the case of trauma with loss of more than 30 per cent of blood volume, blood products are considered in the resuscitation plan, to treat anaemia and coagulopathy. Fresh whole blood is the ideal fluid to replace blood loss, as it provides platelets, red blood cells, fibrinogen and clotting factors; however, this resource is rarely available in the emergency setting and packed red blood cells (PRBC) and fresh-frozen plasma (FFP) may represent a more realistic option, even though their availability remains limited in veterinary medicine (Fig 3). In veterinary practice, most of the cases with traumatic haemoabdomen will not require blood products at all (Hall and others 2014).

If transfusion is indicated, a post-transfusion PCV of 25 per cent could be aimed for. A definitive transfusion trigger (a PCV at which blood product transfusion is recommended) is a controversial topic and no set points have been established. Transfusion should be administered based on a global patient assessment, using clinical parameters and perfusion biochemical indexes (eg, serum base excess or lactate). In some cases, when PRBC may not be available, an auto-transfusion may be a safe and life-saving procedure if performed in a sterile manner and with the use of anticoagulants and haemofilters.

Canine FFP is a natural colloid containing clotting factors and so is indicated for the treatment of coagulopathies. Deciding when it is required is, again, a controversial issue, and generally taking the whole clinical picture into account (including owner finances) is advised. In patients with active haemorrhage, an increase of 1.5 times above the reference range of prothrombin time (PT) and activated partial thromboplastin time (aPTT) may be used as an indication to initiate plasma therapy. Newer technologies, such as viscoelastic monitoring (eg, thromboelastography) are more sensitive and may become useful in decision making once more widely available. The dose of FFP is somewhat empirical; however, a starting dose of 10 to 20 ml/kg is usually recommended (Culp and Silverstein 2014).

An alternative or adjunctive method to control active bleeding is external abdominal counterpressure (Fig 4). This technique consists of applying gradual compression to the abdomen with the use of a generous layer of soft padding bandage first, followed by a compressing elastic bandage (Vinayak and Krahwinkel 2004). The bandage should extend from the xiphoid to the pubis and some clinicians recommend including the hindlimbs. The aim is to achieve an intra-abdominal pressure of 20 to 25 cm H_2O, reducing the degree of haemorrhage and redirecting blood flow to major organs, such as the heart, brain and lungs. However, this method induces abdominal compartment syndrome with consequent renal hypoperfusion and oliguria, intestinal wall oedema and decreased hepatic blood flow, as well as indirect effects on the cardiovascular and respiratory systems; therefore, this bandage should be kept only for a short period (ie, under six hours) until the haemorrhage is controlled. The bandage should then be removed by cutting it in a craniocaudal direction (2 to 5 cm every 15 minutes) in a staged manner, as the sudden removal of the bandage may induce hypotension (Vinayak and Krahwinkel 2004).

One of the biggest challenges in traumatic cases is decision-making regarding surgical intervention. The inability to achieve cardiovascular stability in a dog with haemoabdomen, despite appropriate medical management, means immediate surgical intervention is indicated (Culp and Silverstein 2014). Other indications for surgical treatment are haemoabdomen as a consequence of a penetrating injury, an increasing effusion detected by sequential ultrasonographic assessment and an increase in sequential abdominal PCV with a reduction in the peripheral PCV (Vinayak and Krahwinkel 2004) (Box 2). Therefore, while medical management should still be considered the main initial treatment, surgical requirement should not be delayed if required.

The importance of early intervention to avoid further bleeding is highlighted by the principle of damage control surgery, which is an abbreviated surgery to achieve immediate haemostatic control, stabilisation of the patient by intensive care to correct acidosis, coagulopathy and hypothermia so that they are ready for an operation for definitive repair 24 to 48 hours later (Peterson and others 2015). As interesting as this strategy can sound it has never been reported in dogs and cats and may be considered only in selected cases in which medical management proves ineffective and the patient requires haemostatic control and stabilisation before definitive repair.

Penetrating injury and septic peritonitis

Penetrating injuries are usually a result of bite wounds in dogs (74 per cent of the total penetrating trauma in a retrospective study [Hall and others 2014]), with other causes including gunshots and stab wounds. As a general rule, when a trauma results in penetration of the abdomen an exploratory surgery should be planned to assess if there is any organ damage (Culp and Silverstein 2014) and to treat any contamination (either from the penetrating wound or due to organ damage). Vascular injuries can result in haemorrhage; however, other organs may be damaged causing septic peritonitis and/or uroabdomen. If septic peritonitis is present,

as soon as the patient is stabilised, surgery is required to decontaminate and resolve the underlying cause.

In a recent study of shotgun injuries in dogs and cats, four of five patients with abdominal injury had rupture of the intestine resulting in septic peritonitis, with the remaining patient not having further investigations and being discharged against medical advice (Olsen and others 2014). In bite wounds, the current opinion is that surgical exploration should always be performed to assess the severity of the injury (as abdominal penetration is not always obvious) and to debride necrotic tissue (Culp and Silverstein 2014). In people, the requirement for surgery for abdominal penetrating injury is a topic of debate. A careful evaluation is performed by trauma surgeons following advanced imaging (CT scan) and repeated FAST scans and include the trajectory of the penetrating object, presence of cardiovascular stability and lack of diffuse abdominal pain and signs of peritonitis. A selective non-operative management may be the preferred option to avoid non-therapeutic or negative exploratory laparotomies and the associated increase in morbidity (Butt and others 2009). However, the current veterinary literature suggests that a surgical exploration should always be performed in dogs with traumatic penetration of the abdomen.

Uroabdomen

Another consequence of abdominal trauma can be uroabdomen (seen in approximately 3 per cent of dogs in several canine trauma studies). The affected organs can range from the kidneys to the urethra; however, the bladder seems to be the most common site of rupture.

Urinary tract rupture is most commonly recognised in the trauma patient due to the presence of free abdominal fluid on presentation, or by increasing azotaemia and hyperkalaemia. Potassium serum concentration can increase markedly and rapidly causing life-threatening bradyarrythmias. Initial treatment of the uroabdomen patient should begin with resuscitation with isotonic crystalloid fluid therapy. No benefit has been shown to using either 0.9 per cent NaCl versus potassium-containing isotonic crystalloids in hyperkalaemic animals, although a study of cats with urethral obstruction found that a more balanced and less acidic solution than 0.9 per cent NaCl, such as lactated Ringer's or Hartmann's solution, may correct the acid-base disorders more quickly (Drobatz and Cole 2008).

In the presence of hyperkalaemia-induced bradycardia, a 10 per cent solution of calcium gluconate bolus (0.5 to 1.5 ml/kg intravenously over five to 10 minutes) should be administered to ameliorate the effect of potassium on the myocardium within five minutes from administration, lasting for approximately 30 minutes. For longer-term control, medications that induce potassium translocation into the cells and reduce serum concentration should be given. Regular insulin (0.5 iu/kg intravenously) can be given, but must be followed by 2 g/iu of dextrose and initiation of an isotonic crystalloid infusion with added dextrose (2.5 to 5 per cent) infusion, as blood glucose will usually decrease after the effects of the dextrose bolus. Sodium bicarbonate also induces intracellular potassium translocation and can be given at 1 to 2 mEq/kg slowly over 15 minutes to decrease its serum concentration (Stafford and Bartages 2013). Generally, uroabdomen patients have a concurrent metabolic acidosis and so acid-base

derangement does not result from sodium bicarbonate administration. In cases with marked cardiovascular instability or when surgical intervention is not possible in the short term, drainage of the abdominal fluid can be performed. Therapeutic abdominocentesis requires an aseptic technique and can be performed as described for removal of haemoabdomen fluid above (Fig 5). However, if repeated drainage is thought likely to be required, a temporary abdominal drain can be placed, such as a pigtail catheter, a Foley catheter, or a narrow bore thoracostomy tube placed via the modified Seldinger's technique (not using a standard trochar thoracostomy tube) under a brief general anaesthetic and/or regional anaesthesia (Stafford and Bartages 2013). Because of the risk of occlusion that these catheters may carry, in cases in which surgical repair may have to be delayed and/or require specialist intervention, a more invasive approach may involve insertion of a peritoneal drain, such as a Jackson-Pratt drain or peritoneal dialysis catheters, which allows reduction of the risks associated with occlusion. A partial omentectomy may also be performed in the same approach.

Urethral catheterisation by means of an indwelling soft catheter (eg, silicon or Teflon) is also useful as urine often drains despite bladder rupture. However, particular care should be taken not to damage the urethra in case where there may be some degree of injury at this level. In cases of urethral rupture, medical management may be attempted by catheterising the urethra and allowing for bladder drainage via a urethral catheter or cystostomy tube (Culp and Silverstein 2014). Medical management may be required from five to 21 days (Stafford and Bartages 2013); however, in cases where a catheter cannot be placed, complete urethral rupture or unsuccessful medical management, surgical repair is required.

In patients with uroabdomen, surgery is strongly recommended and often necessary, as conservative management may lead to delayed repair and should only be considered in an unstable patient. In certain scenarios, diversion methods, such as nephrostomy or cystostomy tubes may be used as a temporary solution to control urine leakage in the peritoneum and normalise the electrolyte-acid base abnormalities while awaiting for a definitive surgical repair.

Conclusion

Abdominal trauma can cause several degrees and type of injuries; however, the initial approach should always include appropriate fluid resuscitation, analgesia and careful assessment of the patients. Most canine abdominal trauma patients can be medically managed, even if haemoabdomen is present; however, certain conditions require surgical repair. In these cases, efforts should focus on initial aggressive medical management to achieve optimal perfusion and stabilisation of the patient and surgery performed once the patient is considered a good anaesthetic candidate.

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