Small Animal Emergency Medicine Case Challenges for Advanced Practitioners Mini Series

Session 3: Approach to the trauma patient

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The Trauma patient

Introduction:

Cats can suffer a traumatic event from a multitude of sources. Common causes include vehicular accidents ('hit by car'), being stepped on or having furniture dropped on them, secondary to being attacked by a dog or other larger animal and high-rise syndrome.

Some points regarding the general approach to the traumatised cat:

- General triage and primary survey rules apply to all traumatised cats

Triage means "sorting out"/prioritising treatment necessities based on urgency. It is a very quick procedure and should be done immediately on patient arrival at the clinic, which requires good co-ordination between the receptionist and the medical team. Severely affected/unstable patients should be taken to the treatment area without delay

Thoracic trauma:

- The feline thorax is very resilient, therefore clinical compromise due to thoracic injuries can be an indicator of severe trauma
- Severe thoracic pain can lead to hypoventilation, hypercapnia and in some cases to hypoxaemia, pain relief is essential.
  Pain should be controlled early, but consider the effect of each analgesic on neurological system/mentation. Multimodal strategies are very effective but once you have started an analgetic regimen, check these strategies frequently.
- If the patient is dyspnoeic, the pattern and sounds are you most valuable tools to distinguish between the underlying pathology/location of the disturbance: If you can hear loud sounds without the aid of an stethoscope, this indicates an upper airway problem. If no hear no loud sounds, auscultate the thorax. If you find dull lung sounds, this could point towards a pleural space injury, if you hear increased sounds, it usually means parenchymal lung pathology. Wheezing or increased effort on expiration points towards a problem within the lower airways (narrowing/secretions, etc.).
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- Pneumothorax
- Haemothorax
- Flail chest: Pendulous airflow
- Decreased vital capacity
- Decreased residual capacity
- Decreased compliance
- Increased airway resistance
- Might position the flail chest part down on table
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- Spalling effect: shearing at gas/liquid interfaces
- Inertial effect: different rates of acceleration of different density tissue
- Implosion effect: overexpansion of gas bubbles after a pressure wave passes
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- Resulting hypoxaemia due to ventilation/perfusion mismatch, diffusion impairment and hypoventilation.
- Tension pneumothorax
- Consider thoracocentesis
- The three bottle draining system
- Chest drains (trocar, seldinger)

Temperature

FAST: Focussed Assessment with Sonography for Trauma) Main function is to detect fluid within the abdomen following traumatic injury

Should take less than 5 minutes, should be done fairly quickly after triage

No in-depth examination of intra-abdominal structures

Lateral recumbency, four different regions to assess:

Ventral midline just under end of sternum

Flank, left

Flank, right

Ventral midline just before pelvis

Two views each site! Subsequent one after fluid therapy (if the first one was negative!)

Head trauma:

- Clinical signs can vary from mild and insignificant to being presented seizuring or in a coma.
- Other major body systems may also be affected including the respiratory and cardiovascular system (e.g. blood loss) making these cases some of the most challenging encountered.
- Trauma to the head results in soft tissue injury (e.g. face, mouth), orthopaedic and dental injury (e.g. mandibular fractures), ocular injury, and most importantly traumatic brain injury (TBI).

<u>Primary and secondary Injury</u>

- Primary injury is the injury that is sustained as a direct result of the trauma. As is the case for injury anywhere in the body, this can be considered as orthopaedic injury or soft tissue injury. Skull fractures may occur, but direct soft tissue injury such as axonal injury/laceration may also occur, as well as localized or diffuse haemorrhage/bruising (haematoma and contusion formation respectively). Extra-axial haemorrhage can cause a space occupying lesion that will directly or indirectly (through raised intracranial pressure) cause cerebral compression. With the exception of depressed skull fractures and haematomas primary injury is not amenable to treatment. In these specific cases, skull fragments and haematomas can be removed surgically.
- Secondary injury is the injury that occurs indirectly, as a result of the primary injury. Following any primary injury in the body, inflammatory cells such as neutrophils and monocytes are activated and migrate into the site of the injury. Inflammatory mediators are released via the arachidonic acid pathways, and reactive oxygen species are produced through ischaemia reperfusion injury. Platelets are activated and lead to microvascular thrombosis and compromise of local perfusion. The inflammation leads to oedema and swelling, as it does in any other organ. However, because of the axons that are damaged, there is release of excitatory amino acids such as glutamate and calcium leading to excitotoxicity and intracellular accumulation of sodium and calcium that leads to worsening cellular oedema. The result of all of these processes (that occur secondary to the original primary injury) is progressive neuronal cell death and the development of cerebral oedema. The mainstay of treatment of the cat with traumatic brain injury is to reduce the development of secondary injury and to treat the consequences of it.

Intracranial Pressure and Cerebral Perfusion Pressure

- Blood flow to the brain (perfusion) is determined by the cerebral perfusion pressure (CPP). In essence this is the pressure difference between the force driving blood to the brain, and the resistance to blood flow entering the brain. This is determined by the mean arterial pressure (MAP; the driving pressure) and the intracranial pressure (ICP; which will impede blood flow). *CPP = MAP – ICP*

- CPP represents the amount of blood going to the brain. In normal patients this is kept relatively constant in spite of changes to mean arterial pressure. However, once MAP has dropped to a critical point (50-60 mmHg) perfusion is linearly correlated to MAP. If we assume that ICP remains constant in the absence of neurological disease, this explains why people pass out (from lack of oxygen) if they become severely hypotensive and why animals are obtunded when they are presented with severe shock. There just isn't sufficient oxygen delivery for the brain to function.

- Now, if we consider a cat that has sustained head trauma but no other organ system such that its MAP remains constant. As a result of the primary and secondary injury the brain swells, and since it lies within a solid non-distensible cranium, the ICP increases. As the ICP increases so the CPP decreases. Oxygen delivery to the brain will be reduced, and the brain is ischaemic. This in turn will worsen the secondary injury and neurological signs will get progressively worse.

Physical Examination

Initial assessment of the cat should focus on patient mentation. Assessment of mentation needs to be made in light of the findings of the cardiovascular system. Due to decreased perfusion to the brain from shock, a cat may be obtunded following trauma without having any primary neurological injury. In addition, prior administration of drugs (e.g. atropine and opioids) should be considered when performing a neurological examination.

Clinical Management

- The primary aim of treatment of cats with TBI is to ensure that they have adequate delivery of oxygen and essential nutrients to their brain. Since CPP is determined by MAP and ICP it follows that the priority is to optimize the blood pressure and to minimize intracranial pressure.

- Normovolaemia should be restored using the fluid challenge technique. Multiple boluses of isotonic crystalloids or colloids should be given followed by frequent reassessment of endpoints of resuscitation as outlined in the previous lecture on shock. In addition, hypertonic saline (HTS; 7% NaCl) should be considered. Not only does hypertonic saline raise MAP by drawing in fluid from the interstitium and intracellular space into the intravascular space, but it also decreases ICP. 2-3 mL/kg should be given over 5-10 minutes. HTS also has positive inotropic effects and is thought to have beneficial vasoregulatory and immunomodulatory effects.

- Appropriate monitoring of the cardiovascular system including frequent physical examinations, assessment of arterial blood pressure, and monitoring of urine output is essential in these cases. In addition, electrolytes, blood gasses and PCV, total solids and blood glucose should all be closely monitored.

- The cat should be well oxygenated to ensure delivery of oxygen to the brain. As such oxygen supplementation should be provided during initial stabilization, and SPO2 maintained above 95%,

- If there is any concern of traumatic brain injury, the patient should be elevated 15-30º with a flat board, to decrease intracranial pressure. The jugular veins should not be occluded for blood collection or by the method of head elevation, and any stimulus which may induce sneezing should be avoided.

- Intracranial pressure can be decreased using either mannitol or hypertonic saline as described above. Mannitol is an osmotic diuretic that is able to reduce ICP by reducing cerebral oedema. It has also been proposed that mannitol decreases ICP by reflex vasoconstriction of cerebral vasculature via decreasing blood viscosity, reduction of CSF production and scavenging reactive oxygen species. It should be given at a dose of 0.5-1.5

g/kg IV over 15-20 minutes. Care should be exercised so that the cat does not become dehydrated and hypovolaemic. Fluids should therefore be given concurrently, and the cardiovascular system frequently reassessed.

- Hypoventilation (leading to increased CO2) causes cerebral dilatation, pooling of blood within the calvarium and potentially an increase in ICP. Altered mentation and possible upper airway obstruction can lead to hypoventilation. Blood gasses can be performed to ensure that the cat is ventilating appropriately, and if it is not then the cat should be intubated and ventilated. Particular attention should be paid to cats that are anaesthetized or sedated as they may hypoventilate. If the cat is anaesthetized it should be intubated and have a capnograph attached.

- Blood glucose should be closely monitored in cats with neurological trauma since hyperglycaemia has been associated with the severity of head trauma in dogs and cats and is likely to be inversely related to outcome. Glucocorticoids such as high dose methoprednisolone sodium succinate (MPSS) and prednisolone were advocated for a long time following head trauma. The benefit was thought to lie as a neuroprotective agent. However, large human clinical trials have demonstrated a worse outcome in with patients with TBI that have been treated with MPSS. In addition, corticosteroids are known to raise blood glucose, the one parameter known to be linked to a negative outcome in people, and thought to be associated with a negative outcome in veterinary species. As such, corticosteroids should not be used in cats with head trauma.

- Cats should be frequently assessed and have the results of the neurological examination written down. These patients are very dynamic, and their condition can deteriorate extremely quickly. Frequent examinations will permit subtle changes to be detected and appropriate therapy to be initiated. If the cat's clinical signs are worsening despite appropriate aggressive medical care, further diagnostics including CT and MRI should be considered, and if indicated decompressive craniectomy.