Successful management of head trauma in a Leopard

Severe head trauma is associated with high mortality in human and animal. The appropriate therapy for head trauma patients remains controversial in veterinary medicine due to lack of objective information on the treatment of dogs and cats with head injuries. Treatment for affected animals must be immediate if the animal is to recover to a level that is both functional and acceptable (Platt 2008).

In big cats common causes of head trauma are automobile accidents and fighting; automobile accidents are most common. Concussion with depressed mentation, facial fractures and ocular injuries were common; other complications include soft tissue damage and proptosis (Opperman 2014).

There are very few clinical data available regarding head trauma that are specific to leopards and cats; however, the guidelines produced for human head trauma patients can be almost directly transposed to veterinary patients for practical use (Sato et al. 2003).
Case Report

A one year old male leopard was presented to Animal Rescue & Rehabilitation Centre, Katraj, Pune with a history of automobile accident. Clinical examination revealed semi-comatose condition, dilated pupil, PLR absent, lateral recumbence, injury to left eye, tilting of head towards left side, swelling of frontal bone. To assess the condition of bones radiological examination was carried out which reported hair line fracture at the proximal end of humerus. Neurological examination was carried out as reported by Platt (2015) and documented in Table 1.

Treatment was initiated by administration of fluid therapy Inj. Cefotaxim @ 20 mg/kg b.wt, Inj. Mannitol (20% solution) 0.5 gm/kg b.wt. IV over 15-20 minutes, Inj. Ringer lactate 4-5 ml/kg b.wt. IV, Inj. Voluven (Hetastarch) @ 10 ml/kg bwt IV, (Beal 2008), Inj. Solumedrol (Methylprednisolone acetate) 2 mg/kg b.wt. IM, Inj. Neurokind 2ml IV, Inj. Sodium Bicarbonate 7.5% solution twice a day. To maintain blood oxygen level oxygen therapy was administered @ 3 lit/hr. For treatment of hair line fracture bandaging was carried out with Robert Johns bandage. Ketler eye drop was use thrice a day.

After two days of aggressive treatment animal responded to it and showed improvement. Further, six day of treatment leopard showed complete recovery with normal head position, normal PLR, and appetite. Its left eye showed permanent sub-conjunctival hemorrhage due to which he had partial vision.

The aim of treating traumatic brain injury was to minimize or reverse the secondary changes that occur after the initial impact damage. Blood flow and oxygenation to the brain need to be optimal to prevent ischemia and cell death, while at the same time avoiding sudden increases in intracranial pressure (ICP). Secondary damage occurs in the hours and days following the incident.

In the present study animal was in recumbent position and fluid therapy was initiated with ringers lactate solution to avoid shock and maintain blood volume as per
documented by Platt (2015). Hypertonic saline resuscitation has the advantages of smaller volume resuscitation, rapid restoration of intravascular volume thus improving cerebral perfusion pressure, improved contractility, and its osmotic effect at the level of the brain thus lowering intra cranial pressure (Beal 2008).

The goal of oxygen therapy in the present case was to maintain arterial partial pressures of oxygen \( (\text{PaO}_2) \). As hypercapnea produces cerebral vasodilatation that increases ICP (Adamantos & Garosi 2011).

The serum protein concentration was 5.71gm/dl on day first (Table 2). To avoid hypoproteinemia and withdrawn of fluids from interstitial and intracellular space hypertonic colloids (Hetastarch) was administered in the present study. According to Beal (2008) hypertonic and colloids fluid therapy can rapidly restoration of blood pressure. It has an ability to improve cardiac output, restore normovolemia, and reduce inflammation after trauma. Hypertonic saline maybe preferred in hypovolemin, hypotensive patient with increase intra cranial pressure (ICP) because it improves cerebral perfusion pressure and blood flow by rapidly restoring intravascular blood volume.

Table 1. Day wise neurological and other examination values

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
<th>Day 7</th>
<th>Day 8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurological examination</td>
<td>Modified Glasgow Coma Scale &gt;15</td>
<td>9</td>
<td>9</td>
<td>11</td>
<td>12</td>
<td>12</td>
<td>15</td>
<td>18</td>
<td>22</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Map 80–120 mmHg</td>
<td>75</td>
<td>95</td>
<td>110</td>
<td>125</td>
<td>126</td>
<td>128</td>
<td>120</td>
<td>124</td>
</tr>
<tr>
<td>Pulseoximeter</td>
<td>SPO2&gt;95</td>
<td>80</td>
<td>90</td>
<td>95</td>
<td>99</td>
<td>98</td>
<td>100</td>
<td>96</td>
<td>100</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>10–25 /min</td>
<td>11</td>
<td>12</td>
<td>19</td>
<td>22</td>
<td>25</td>
<td>24</td>
<td>26</td>
<td>26</td>
</tr>
<tr>
<td>Body temperature</td>
<td>98.6–101.3 °F</td>
<td>96.4</td>
<td>98.0</td>
<td>100.1</td>
<td>100.6</td>
<td>101.4</td>
<td>101.6</td>
<td>101.2</td>
<td>101.5</td>
</tr>
</tbody>
</table>
Hopper (2005) reported that high sodium concentration effectively makes $\text{NaHCO}_3$ a form of hypertonic saline and as such it is a potent volume expander. If given undiluted, it will cause movement of water out of cells and into the interstitial and intravascular space. The result is cell shrinkage and vascular volume expansion. Bourdeaux & Brown (2010) study demonstrates that an infusion of 8.4% sodium bicarbonate is effective for the reduction of raised intra cranial pressure.

In the present case Inj. Mannitol was administered to reduce the intra cranial...
pressure after traumatic brain injury. Beal (2008) documented that mannitol is a sugar with a strong osmotic effect that has been shown to decrease both ICP and cerebral edema after traumatic brain injury. Mannitol exerts its advantageous effects through intravascular volume expansion, reflex cerebral vasoconstriction (decreased CBV) secondary to decreased blood viscosity, osmotic effects, and possibly through its free-radical scavenging effects.

In the present case methylcobalamin was used to treat the nerve injury as it is an active form of vitamin B12, which was used to treat anemia as well as nerve damage by promoting regeneration to injured nerves (Gupta & Qureshi 2015).

To control the secondary bacterial infection cefotaxime was administered as it is a semisynthetic, third generation Cephalosporin’s which enter the CSF in therapeutic levels (at high dosages) when the patients meninges are inflamed documented by Plumb & Pharm (1999). Further Methylprednisolone was use as anti-inflammatory and manage shock, dose should be tapered while discontinuing (Ramsey 2007).

References

A.G. Dubey1*, G.D. Pardeshi2, N.K. Nighot3 & A.A. Sanghai4
1 Animal Rescue and Rehabilitation Centre, Katraj, Pune, Maharashtra 411046, India, 2 Rajiv Gandhi Zoological Park and Wild Life Research Center, Katraj, Pune, Maharashtra 411046, India 3 *Corresponding author: drankush.dubey@gmail.com

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