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Case Report

Severe brain damage after punitive training technique with a choke chain collar in a German shepherd dog

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ABSTRACT

The features of severe ischemic brain damage after strangulation by the owner of a 1-year-old German shepherd dog are described. The dog was disciplined by the owner during training by holding the dog off the ground by his choke chain collar. At first, the dog behaved normally, but he became increasingly ataxic and started circling to the left and showed reduced consciousness. The neurological examination revealed severe disorientation, left lateral pleurothotonus, and circling. The neurological findings were consistent with a multifocal brain lesion. A magnetic resonance imaging scan was performed and showed changes in the T2- and diffusion-weighted images, consistent with severe cerebral edema resulting from ischemia. Because of the severity of the clinical features, the dog was later euthanized. To the author's knowledge, this is the first report of a severe brain ischemia after strangulation in a dog.

Case description

A 1-year-old, male intact, German shepherd dog (weight 40 kg) with incoordination and circling to the left was presented at our small animal clinic. Four hours before presentation, the dog had been disciplined by the owner because of misbehavior during training. The dog was suspended a few feet in the air by its choke chain collar, a common form of punishment among dog handlers (Miller, 2008). By the owners' reckoning, the action was terminated after approximately 60 seconds when the dog panicked and finally lost consciousness for a few seconds. At first, the dog appeared normal to the owner. During the next few hours, the dog became increasingly ataxic on all 4 limbs and was circling to the left. The initial investigation of the referring veterinarian showed no abnormalities on general physical examination. Metamizol, dexamethasone, and vitamin B of unknown quantities were administered by the local veterinarian. For further evaluation of the neurological status, the dog was referred to our institution.

On presentation, the dog was anxious, panting, tachycardic (heart rate, 140 beats per minute), showing a body temperature of 40.0°C, capillary refill time of 2 seconds, as well as dark pink and tacky mucous membranes.

The neurological examination revealed severe disorientation, left-sided pleurothotonus, and circling. The hopping reaction was slightly reduced in all 4 limbs. As a sign of blindness, the menace response and the direct and indirect pupillary light reflexes were negative in both eyes. A bilateral mydriasis was present. The dog showed a variable nystagmus and a left-sided facial motor paralysis. The neurological findings were consistent with a multifocal brain lesion. Differentials include diffuse axonal injury, vascular ischemia, increase in intracranial pressure, and hemorrhage.

The results of the complete blood count and serum biochemistry were within the laboratory reference ranges.

The dog was treated with a shock bolus (20 mL/kg for more than 20 minutes) of crystalloid intravenously, and after 30 minutes showed a physiological body temperature,

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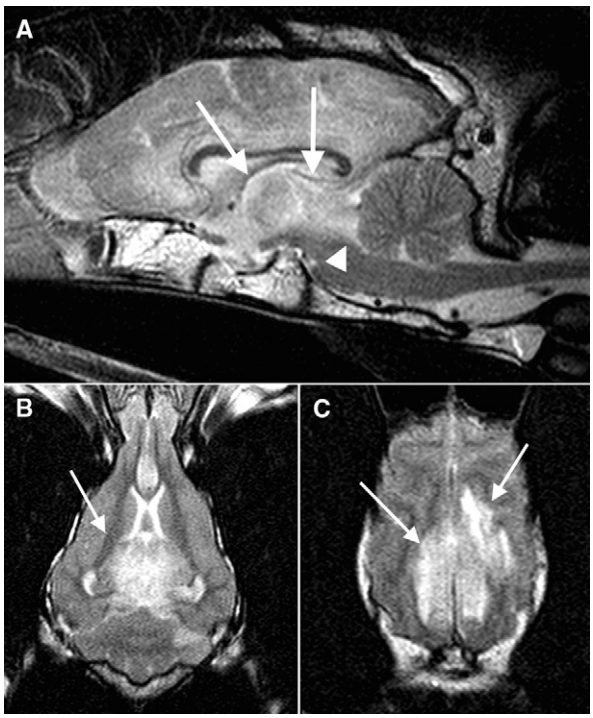

Figure 1. (A) Sagittal T2-weighted image. There is a hyperintense lesion (white arrows) in the dorsal and ventral aspect of the thalamus, both hippocampi, the lamina tecti of the midbrain. A mild rostrotentorial herniation of the lamina tecti was noticed (white arrowhead). (B) and (C) Dorsal T2-weighted images at the level of the interthalamic adhesion. There is a severe hyperintense ill-defined lesion in the thalamus (white arrow) at the level of the dorsal surface of the cortex. Both marginal gyri and left-sided parts of the ectomarginal gyrus present ill-defined hyperintense lesions (white arrows).

normal heart rate, normal pink mucous membranes, and a capillary refill time of below 1.5 seconds. For the purpose of diagnostic imaging, the dog was sedated with 0.1 mg/kg diazepam (Ratiopharm GmbH, Ulm, Germany) and 0.3 mL/kg propofol (Rapinovet, Essex, Tierarznei, Germany) given intravenously. General anesthesia was maintained after intubation by inhalation of 2% isoflurane (IsoFlo, Dr. E. Graeub AG, Germany) in 100% oxygen.

Imaging

Magnetic resonance (MR) tomography was performed with a 1.0 Tesla system (Gyroscan Phillips, Hamburg, Germany) and a knee coil. The T2-weighted scans showed (Figure 1A-C) (sagittal, field of view [FOV]: 222 mm, 15 slices, slice thickness: 4.0 mm, GAP: 0.3 mm, repetition time [TR]: 1756 milliseconds, echo time [TE]: 108 milliseconds, matrix: 360 × 360 and dorsal: FOV: 222 mm, 18 slices, slice thickness: 3.5 mm, GAP: 0.3 mm, TR: 1969 milliseconds, TE: 108 milliseconds, matrix: 320 × 320) a diffuse, nonhomogeneous, poorly demarcated hyperintense lesion within the dorsal and ventral aspect of the thalamus, both hippocampi, the lamina tecti of the midbrain and the splenal, suprasplenal, and marginal gyrus. The lesion was slightly more pronounced on the left side. A mild rostrotentorial herniation of the lamina tecti was noticed.

In the T1-weighted scan (transversal, FOV: 200 mm, 26 slices, slice thickness: 4.0 mm, GAP: 0.4 mm TR: 701 milliseconds, TE: 15 milliseconds, matrix: 403 × 403), the lesion presented with a mild hypointense signal intensity in contrast to the surrounding brain tissue, most severely in the thalamus, and could not be delineated.

In the diffusion-weighted scan (Figure 2A-F) (dorsal, b: 0 and b: 700, FOV: 230 mm, slices: 18, slice thickness: 5.0 mm, GAP: 1.0 mm, TR: 3096 milliseconds, TE: 0.0 milliseconds, matrix: 77 × 77), a hyperintense signal was visible in the same areas as in the T2-weighted sequences. Because diffusion-weighted images are also sensitive to T1 and T2 relaxivity contrast, which can sometimes be confusing, an apparent diffusion coefficient (ADC) calculation was performed. The ADC measures the magnitude of diffusion within the cerebral tissue. In the resultant ADC maps, this area appears hypointense compared with the diffusion-weighted images. No contrast studies were performed.

The presumptive diagnosis of a severe cerebral edema resulting from ischemia was made. As a result of the neurological findings and magnetic resonance imaging (MRI) findings, the owners chose to euthanize the dog. Postmortem examination was declined by the owner.

Discussion

Swinging or lifting a dog off the ground by the collar (helicoptering or hanging) is a highly controversial form of punitive training technique occasionally applied by dog handlers or trainers (Miller, 2008). Dog handlers in favor of this technique still believe that its correct application "takes the drive and fight out of the dog" and does not cause any pain (Leerburg, 2010). It has been widely recognized by most veterinary behavior specialists that this form of punishment should be avoided as it causes fear and brings about an escalation of defensive aggression and increasing intraocular pressure (Hetts, 2000; Pauli et al., 2006; AVSAB, 2012). Furthermore, choke chains and collars can cause mechanical or ischemical damage to the larynx, esophagus, thyroid, or trachea (Brammeier et al., 2006). Cerebral ischemia may also occur after general anesthesia (Jurk et al., 2001; Stiles et al., 2012) during birth (Levene et al., 1986; Dickey et al., 2011), vascular thrombosis, asphyxia, and cardiac arrest (Püttgen and Geocadin, 2007; Choi et al., 2010). It is reported in humans and animals such as cats or dogs (Panarello et al., 2004; Timm et al., 2008; Choi et al., 2010). To the author's knowledge, there has been no description of ischemic brain damage as a result of strangulation in animals so far.

The neurological deficits in this case were circling, blindness, and ataxia. Blindness is a well-known risk of hypoxic brain damage and has been described in cats (Palmer and Walker, 1970; Jurk et al., 2001; Panarello et al., 2004; Stiles et al., 2012), humans (Siesjö, 1992; Grover and Jangra, 2012), and dogs (Palmer and Walker, 1970; Timm et al., 2008) especially after anesthesia. Because in this case no severe lesions were displayed in the occipital cortex, the blindness may have been a result of damage to the visual pathway before the cortex. Comparable

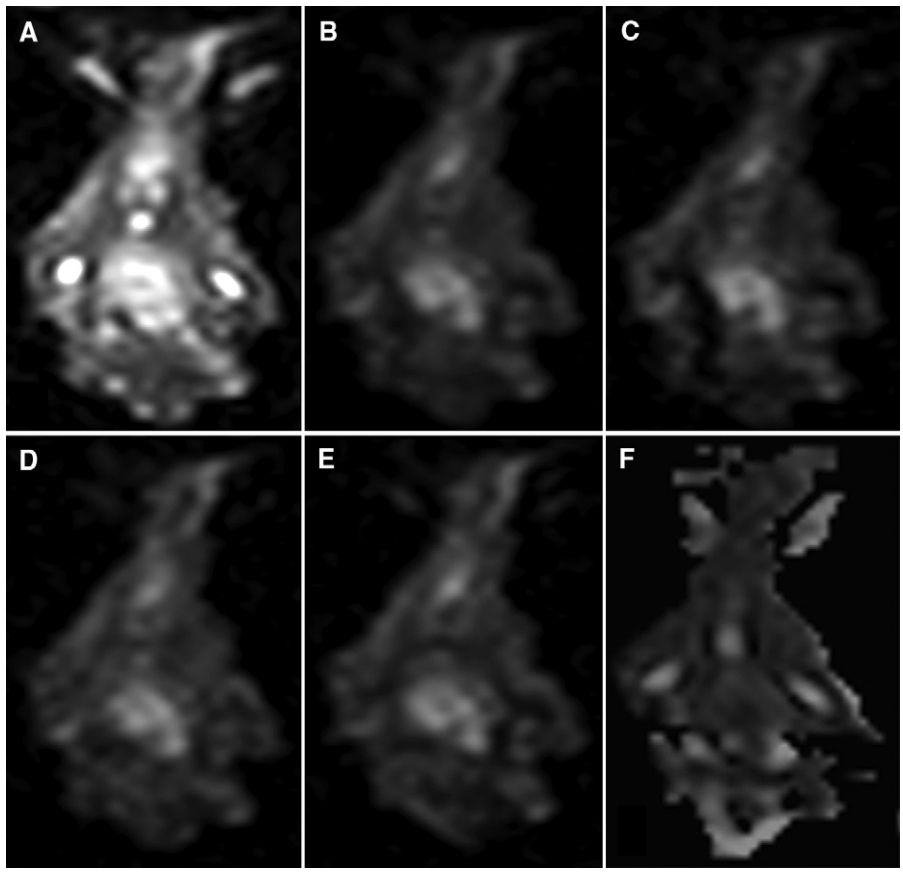

Figure 2. Diffusion-weighted sequences and the corresponding ADC at the level of the thalamus (A) shows the b = 0 and (B) the trace image with a diffusion weighting b = 700, (C-E) the 3 different diffusion directions. The hyperintense signal in the thalamus and midbrain is visible not only in T2 but also in all DWI directions because of the restricted water diffusion associated with edema after acute ischemia. (F) The hyperintense area in DWI appears hypointense on ADC.

neurological deficits have been described in cats and horses (Jurk et al., 2001; McKay et al., 2002; Stiles et al., 2012), and deterioration of clinical signs as in our case is a common risk in hypoxic brain damage (Jurk et al., 2001).

The pathophysiology of strangulation has been described in human medicine. Pressure on the neck of a victim causes pain followed by anxiety from the subjective sensation of breathing trouble. The rapid loss of consciousness within 10-15 seconds is because of apnea as well as arterial obstruction, venous obstruction, and response of autonomic nervous system reflexes (Kiani and Simes, 2000). Forces applied to the carotid artery impair the flow through the artery by mechanical obstruction or the induction of spasm, leading to a loss of consciousness as observed in our patient. Damage to the vessel walls and intima may induce thrombosis and produce clinical signs occurring over 12-24 hours (Kaki et al., 1997; Clarot et al., 2005; Chokyu et al., 2006). Furthermore, the thrombosis caused by the traumatized internal carotid artery can embolize to the cerebrovascular circulation (Cothren and Moore, 2005). Venous outflow obstruction causing stagnant ischemia is a significant factor that produces loss of consciousness (Khokhlov, 2001). In humans, loss of consciousness because of manual strangulation is considered one of the most significant signs of danger to life (Christe et al., 2010).

Brain tissue has a high oxygen and energy demand, yet little reserve. Under normothermic conditions, the oxygen reserves are exhausted within 20 seconds, and glucose required for the production of adenosine triphosphate is consumed within 5 minutes without perfusion (Safar and Kochanek, 2000). The lack of oxygen results in a switch to anaerobic metabolism, followed by a depletion of high-energy phosphate reserves, lactate accumulation, and instability to maintain cellular homeostasis. The consequential disease pattern is described as hypoxic brain damage or rather as postanoxic encephalopathy (Safar and Kochanek, 2000). The return of normal perfusion does not resolve the cell damage and instead may lead to a reperfusion injury, characterized by the release of free radicals, nitric oxide, and further glutamate release (Hallenbeck and Dutka, 1990; Ikeda and Long, 1990). This finding might explain the progressive neurological worsening observed in the present patient.

Hypoxic cell damage is possible throughout the entire brain, yet not all regions of the brain are equally sensitive. Especially vulnerable are the basal ganglia, cerebellum (especially the Purkinje cells), hippocampus, and the layers

3, 5, and 6 of the cortex (Püttgen and Geocadin, 2007; Choi et al., 2010). The regions of occipital and parietal cortexes showed changes after reduced perfusion (Choi et al., 2010). Human patients after a cardiovascular standstill showed an unfavorable outcome when defects in the basal ganglia and cortex occurred (Choi et al., 2010). Panarello et al. (2004) reported a dog and a cat that recovered to almost normal except for a persistent blindness. Therefore, the outcome in pets appears to be favorable compared with humans. The MRI changes presented in this patient relate to those found in humans and dogs (Safar and Kochanek, 2000; Timm et al., 2008) with hypoxia. As in humans and dogs, the areas affected in this German shepherd were principally the hippocampus, mesencephalon, as well as occipital and parietal cortexes. In contrast to veterinary cases where the occipital cortical area is also considered a vulnerable region, it does not apply to the German shepherd dog presented in this case (Safar and Kochanek, 2000; Jurk et al., 2001).

For the diagnosis of hypoxic brain damage, conventional MR is often inadequate during the early phase, as the brain morphology at this point in time appears normal or with only slight changes (Arbelaez et al., 1999; Choi et al., 2008). In contrast to this, diffusion-weighted MR (DWI) is capable of depicting acute and subacute changes after a global cerebral hypoxia (Arbelaez et al., 1999; González et al., 1999). DWI uses the movement of protons within the tissue. During hypoxia, the membrane-bound Na-K pump malfunctions leading to an influx of water from the extracellular into the intracellular space, inhibiting the intracellular proton movement. Areas with limited diffusion appear markedly hyperintense in the DWI (Lövblad and Bassetti, 2000; Schaefer et al., 2000). DWI and the corresponding ADC can show the restricted diffusion associated with acute ischemia about 30 minutes after the ictus in patients with acute stroke (Choi et al., 2010). Furthermore, the degree of the changes of the DWI signal intensity correlates with the severity of neuronal injury (Rojas et al., 2006). Although the MR was performed only a few hours after the strangulation in this case, the T2 images were of diagnostic quality. In human strangulation cases, MRI frequently includes the neck area to evaluate the integrity of the soft tissue structures. Hemorrhage into the lymph nodes is a specific diagnostic sign indicating strangulation as the pathomechanism (Yen et al., 2005). Further findings may be subcutaneous or glandular hemorrhage and fractures of the larynx, thyroid, and hyoid cartilage (Yen et al., 2005).

Because of the clinical history and unremarkable clinical examination, strangulation by the owner is considered the most probable cause. Furthermore, up to this point in time, the dog had not shown any neurological abnormalities. The main limitation of this study is the lack of necropsy and histopathological examination of the damaged brain tissue, but the owners declined further investigations.

To the author's knowledge, this is the first case description of a dog with suspected hypoxia after strangulation by the owner. Considering that disciplining a dog by holding it off the ground by its collar appears to be frequently applied practice, it is important to recognize that even a short period is sufficient to produce life-threatening damage to the brain. Because this technique of punishment is not uncommon, further cases of ischemic brain damage resulting in the death of the dog may have gone unreported. It is therefore important to inform veterinary personnel, dog trainers and owners that hanging or "helicoptering" dogs as a form of discipline is a severe health risk that can be fatal.

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