Prevalence of seizures in cats after head trauma

Kristina S. Grohmann, DVM; Martin J. Schmidt, Dr med vet;
Andreas Moritz, Dr med vet; Martin Kramer, Dr med vet

Objective—To determine the prevalence of seizures in cats after head trauma.

Design—Retrospective cross-sectional study.

Animals—52 cats with head trauma.

Procedures—Information was obtained from medical records of cats with head trauma and via telephone interviews of owners at least 2 years after cats had head trauma. Severity of head trauma in cats was classified with the modified Glasgow coma scale (mGCS), and the association between scores and development of seizures was determined.

Results—9 cats had moderate head trauma (mGCS score, 9 to 14), and 43 cats had mild head trauma (mGCS score, 15 to 18). None of the cats developed seizures during the follow-up period (≥ 2 years after head injury). The calculated 95% confidence interval for prevalence of seizures in cats after head injury was 0% to 5.6%. There was no significant relationship between severity of head trauma and the risk of seizures in cats.

Conclusions and Clinical Relevance—Results indicated the probability that cats with mild to moderate head trauma would develop posttraumatic seizures was low. However, clinicians should monitor cats with a history of head trauma for development of secondary epilepsy. (*J Am Vet Med Assoc* 2012;241:1467–1470)

Seizures develop in 6% to 20% of humans with TBI.[1,2] Epileptogenesis can be induced via damage to vulnerable regions of the brain (eg, the hippocampus), intracerebral hemorrhage, or cerebrocortical damage.[3–5] The severity of brain lesions is directly associated with risk of seizures in humans.[6] Posttraumatic seizures (ie, seizures developing after head trauma) can result from brain damage attributable to a direct mechanical force to the head and brain (primary TBI) or nonmechanically induced brain damage not attributable to primary TBI (secondary TBI).[6]

Posttraumatic seizures can be classified on the basis of the time of onset relative to a brain injury; early PTSs develop within 1 week after injury, and late PTSs develop later than 1 week after injury. Early PTSs are typically associated with severe head injuries in humans, but such seizures may also develop after mild or moderate head injuries[7]; 50% to 80% of humans who develop early PTSs do so within 24 hours after head injury.[7–9] In children, early PTSs are more common than late PTSs; late PTSs develop in only 1% to 2% of children with head injuries.[8,9] Within 5 years after head trauma, PTSs develop in 0.5% of humans with mild injuries (loss of consciousness or posttraumatic amnesia for < 30 minutes and no skull fractures), 1.2% of humans with moderate injuries (loss of consciousness or posttraumatic amnesia for 30 minutes to 24 hours or presence of skull fractures), and 10.0% of humans with severe injuries (brain contusion, intracranial hematoma, or loss of consciousness or posttraumatic amnesia for > 24 hours).[10] The majority (65% to 86%) of humans who develop seizures after head trauma develop those seizures within 2 years after that trauma.[11–14]

Abbreviations

mGCS	Modified Glasgow coma scale
PTS	Posttraumatic seizure
TBI	Traumatic brain injury

Head trauma is common in dogs and cats and typically results from kicks, bites, or being struck by a motor vehicle or missile object.[15] Results of another study[16] indicated brain injuries in dogs were typically caused by blunt motor vehicle trauma and those in cats were typically caused by crush injuries. Severe brain injuries are associated with a high mortality rate for humans[17] and other animals[18]; such high mortality rates are attributable to high impact forces on the brain and adjacent organs. Traumatic brain injury reportedly causes secondary epilepsy in small animals.[15,19–21] However, to the authors' knowledge, no studies have been conducted in which the prevalence of PTSs in pet animals has been determined. In addition, seizures that develop as a result of TBI are often refractory to antiepileptic treatment[22] in humans, which worsens outcomes and long-term prognoses; similar findings for cats have not been reported, to the authors' knowledge. The objective of the study reported here was to determine the prevalence of PTSs in cats after TBI.

Materials and Methods

Medical records of the Small Animal Clinic of the Department of Veterinary Clinical Sciences, Justus-Liebig-University, Giessen, Germany, were searched to identify cats that had been hospitalized from January

From the Small Animal Clinic, Department of Veterinary Clinical Sciences, Justus-Liebig-University, 35392 Giessen, Germany.

The authors thank Dr. Klaus Failling for assistance with statistical analysis.

Address correspondence to Ms. Grohmann (ksgrohmann@gmx.de).

2000 through December 2007 for treatment of head injuries. Records for all cats treated for head wounds, head fractures of unknown origin, or injuries attributable to being struck by a motor vehicle were reviewed. Information obtained from medical records of cats included age, sex, type of injury, clinical condition at the time of admission to the clinic, and short-term and long-term outcomes. Severity of neurologic abnormalities at the time of admission to the clinic had been assessed with the mGCS.[23–25] The mGCS included 3 assessment categories (motor activity, brainstem reflexes, and level of consciousness), each of which had been assigned a score of 1 to 6 (**Appendix**). For determination of mGCS scores, the sum of the scores for each of those categories was calculated. The mGCS scores indicated either severe head trauma with a grave prognosis attributable to extensive neurologic dysfunction (mGCS score, 3 to 8), moderate head trauma with a poor to guarded prognosis (mGCS score, 9 to 14), or mild head trauma with a good prognosis (mGCS score, 15 to 18).

Two years or more after cats had been treated at the clinic, owners were interviewed via telephone by use of a standardized questionnaire[a] to obtain information regarding the clinical condition and neurologic status of each cat. Statistical analysis was performed with software.[b,c] For estimation of prevalence of PTSs in cats, an exact 95% confidence interval was calculated. For determination of the relationship between development of PTSs and other factors (ie, mGCS score, age, and sex of cat), an exact logistic regression was performed.

Results

From January 2000 through December 2007, 110 cats had been hospitalized at the clinic for treatment of head wounds, head fractures of unknown origin, or injuries attributable to being struck by a motor vehicle. Of those 110 cats, 52 for which a neurologic examination had been performed by a veterinary neurologist (MJS) were included in the study. Breeds included domestic shorthair (n = 51) and Maine Coon (1). Thirty-one (59.6%) cats were male (29 neutered and 2 sexually intact), and 21 (40.4%) were neutered females. Median age of cats at the time of admission to the clinic was 3 years (mean, 4 years; range, 4 months to 15 years).

At the time of admission to the clinic, 9 of the 52 (17.3%) cats had moderate head trauma (mGCS score, 9 to 14) and 43 (82.7%) cats had mild head trauma (mGCS score, 15 to 18). Diagnostic procedures performed for cats included radiography of the head (for cats with a history of head trauma) and CT of the head (for cats with neurologic deficits and signs of increased intracranial pressure). Thirty-seven cats underwent radiography of the head during anesthesia; 37 cats had facial bone fractures, 30 cats had separation of the mandibular symphysis, 8 cats had fractures of the maxilla, and 3 cats had fractures of the body of a mandible (some of the cats had more than 1 type of fracture). Permission for anesthesia was denied by owners of the other 15 cats. Six of the 37 cats underwent CT; depressed skull fractures or intracranial hemorrhage was not detected in any of the cats.

All 52 cats included in the study had received crystalloid fluids and oxygen. The cats for which the mGCS score (≤ 14) indicated they likely had increased intracranial pressure were treated with mannitol[d] (1 g/kg [0.45 g/lb], IV, slowly during 20 minutes). Additionally, several animals received further treatment such as surgical fracture fixation, esophageal feeding tube placement, treatments for repair of dental injuries, and wound debridement. All cats received treatments (eg, buprenorphine[e] [0.005 mg/kg {0.002 mg/lb}, IV, q 6 h] or meloxicam[f] [0.1 mg/kg {0.045 mg/lb}, PO, the first day followed by 0.05 mg/kg {0.023 mg/lb} once daily]) intended for management of pain.

The minimum hospitalization time for the cats was 7 days. During telephone interviews (from 2 to 9 years after cats had head trauma), owners reported that all 52 cats had recovered uneventfully. Neurologic signs or other clinical signs attributable to head trauma had not been observed in any of the cats after discharge from the clinic. None of the cats developed seizures of any type after hospitalization. The calculated 95% confidence interval for prevalence of PTSs in cats was 0% to 5.6%. Exact logistic regression for determination of the relationship between PTSs and other variables could not be performed because none of the cats developed PTSs.

Discussion

Trauma can cause potentially epileptogenic brain damage via various mechanisms. Traumatic brain injury acutely causes breakdown of the blood-brain barrier, changes in brain perfusion (attributable to disturbed autoregulation of intracranial circulation), increased intracranial pressure, and local release of neuroexcitatory amino acids, cytokines, bioactive lipids, and other mediators.[25] An increase in the extracellular concentration of potassium can cause depolarization and hyperexcitability of neurons.[25] Hyperexcitability of neurons may be potentiated via a trauma-related increase in extracellular glutamate concentrations in rats.[26] Damage to neuronal connections causes alterations in intrinsic membrane properties of neurons and increases responsiveness of neurons to excitatory stimuli, as has been determined for chronically axonotomized cerebrocortical areas in cats.[27]

Trauma secondary to being struck by a motor vehicle is a common cause of death for domestic small animals[28,29] because of high impact forces. Brain damage in humans can be classified with the Glasgow coma scale,[30] and brain damage in animals can be classified with the mGCS.[23,24] These scoring systems can be used to determine probability of survival within 48 hours after head trauma. However, the scores determined with those scales indicate impairment of neurologic function and do not indicate structural damage of the brain and cannot be used to predict long-term neurologic sequelae. The present study was conducted to determine prevalence of seizures after head trauma in cats. Because none of the cats in this study developed seizures, the mGCS may be of limited value for prediction of PTSs.

Head trauma secondary to various causes (eg, experimentally induced head trauma or head trauma secondary to accidents, falls, or gunshot wounds) can cause secondary epilepsy in humans[15] and domestic small animals.[9–21] The risk for epileptogenesis in humans and rodents is dependent on severity of injury.[31–33]

In the present study, all cats recovered uneventfully from head trauma, and owners did not report seizure activity for any of the cats. Therefore, the relationship between severity of neurologic deficits after head trauma and probability of development of PTSs in cats could not be determined.

Cats in the present study may not have developed PTSs because severity of brain trauma is dependent on several physical and mechanical factors. The body size and head position of an animal may influence transmission of mechanical energy to the brain. Cats with severe head trauma may die before they can return home; injuries of such cats would not be evaluated, whereas humans with severe brain trauma are likely to be examined and treated immediately. If cats with severe head trauma had been brought to the clinic of the present study for intensive treatment and survived, some of those cats may have developed PTSs. The size and shape of the brains of cats could influence the nature and severity of the effects of head trauma. Brain damage can be caused by direct compressive pressure (which causes cell damage) or shearing forces that develop in localized regions in the brain (primarily in brain tissue adjacent to the ventricles). High shear forces develop in large brains more often than they develop in small brains during an impact, which can cause severe axonal damage that requires a prolonged time for healing.[33] Other authors[34] have suggested that brains of small animals are more tolerant to injury than those of large animals because brains of small animals are more compact and less deformable under load than those of large animals. That conclusion[34] is supported by results of other studies that suggest the number of neurons per cubic millimeter of brain decreases as brain weight increases[35] and that the ratio of the number of glial cells to the number of neurons in the cerebral cortex of large brains is higher than that in small brains.[36,37] However, rodents are susceptible to PTSs after head trauma, even though they have small brains.[6] Therefore, brain size may not be an important factor in development of PTSs.

Differences between cats and humans regarding development of PTSs may be attributable to differences in biological reactions of brain tissue to trauma in cats and humans. However, to the authors' knowledge, no data regarding such differences between cats and humans have been published. The time during which animals with experimentally induced head trauma develop epileptic seizures varies depending on the nature and severity of trauma.[38] Most human patients that develop epilepsy after TBI do so within 2 years after that injury.[11–14] Although all cats in the present study had head trauma at least 2 years prior to the date follow-up information was obtained, some of the cats had head trauma up to 9 years prior to telephone interviews of owners. Therefore, it was considered unlikely that PTSs would develop in cats more than 2 years after head trauma.

Limitations of the present study included lack of reevaluation of cats by a veterinarian; therefore, follow-up information was obtained via telephone interviews of owners. Because follow-up diagnostic imaging (eg, MRI or CT) of brains of cats was not performed, evaluation of PTSs was restricted to information obtained from owners regarding clinical signs of cats. Furthermore, the number of cats included in the study was low; therefore, further studies including a larger number of cats are warranted to confirm results of the present study. Because follow-up veterinary examinations of cats were not performed, subtle neurologic abnormalities attributable to brain damage may not have been detected. The standardized questionnaire used in this study did not include information regarding types of seizure activity (eg, focal seizures vs generalized seizures) and did not refer to epilepsy; this was intended to prevent bias of owners. Despite use of these methods, some owners may have been biased regarding development of seizures in the cats. However, because behavioral changes of cats attributable to brain damage were not reported by owners, bias was unlikely. In addition, seizures in cats may not have been detected by owners. In particular, focal seizures of cats may not have been detected or may have been falsely interpreted as normal behavior by owners. Similarly, seizures of any severity (eg, generalized tonic-clonic seizures) in cats housed outdoors may not have been detected by owners. However, despite the possibility that owners may not have detected seizures in some cats, we concluded that the cats included in this study maintained a good quality of life after discharge from the clinic.

Determination of the prevalence of PTSs in pet animals after head trauma may require performance of a prospective study. Such a study should include grading of the severity of trauma sustained by animals, standardized treatment protocols, and a long follow-up period with video recording of animals for detection of mild focal seizures or behavioral changes.

Results of the present study indicated that cats with a medical history of mild or moderate head trauma had ≤ 5.6% probability of developing PTSs. However, clinicians should monitor cats with a history of head trauma for development of secondary epilepsy.

a. The questionnaire is available from the corresponding author upon request.
b. LogXact-9, Cytel Inc, Cambridge, Mass.
c. BiAS for Windows, version 9.08, Epsilon-Verlag, Hochheim, Germany.
d. Mannitol-Infusionslösung, Serumwerke Bernburg AG, Bernburg, Germany.
e. Buprenovet, Bayer Vital GmbH, Leverkusen, Germany.
f. Metacam für Katzen, Boehringer Ingelheim GmbH, Ingelheim, Germany.

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Appendix

Modified Glasgow coma scale for determination of head trauma severity in dogs and cats.

Neurologic examination findings	Score
Motor activity	
Clinically normal gait and spinal reflexes	6
Hemiparesis or tetraparesis	5
Recumbent; intermittent extensor rigidity	4
Recumbent; constant extensor rigidity	3
Recumbent; constant extensor rigidity and opisthotonus	2
Recumbent; decreased or absent spinal reflexes and muscle tone	1
Brainstem reflexes	
Clinically normal pupillary and oculocephalic reflexes	6
Slow pupillary reflexes; clinically normal to decreased oculocephalic reflex	5
Bilateral miosis; clinically normal to decreased oculocephalic reflex	4
Pinpoint pupils; decreased or absent oculocephalic reflex	3
Unilateral unresponsive mydriasis; decreased or absent oculocephalic reflex	2
Bilateral unresponsive mydriasis; decreased or absent oculocephalic reflex	1
Level of consciousness	
Occasional periods of alertness; responsive to environment	6
Signs of depression or delirium; capable of responding to environment but response inappropriate	5
Semicomatose; responsive to visual stimuli	4
Semicomatose; responsive to auditory stimuli	3
Semicomatose; responsive only to repeated noxious stimuli	2
Comatose; unresponsive to repeated noxious stimuli	1

For determination of mGCS scores, the sum of the scores for each of the categories (motor activity, brainstem reflexes, and level of consciousness) is calculated. The mGCS scores indicate either severe head trauma with a grave prognosis attributable to extensive neurologic dysfunction (mGCS score, 3 to 8), moderate head trauma with a poor to guarded prognosis (mGCS score, 9 to 14), or mild head trauma with a good prognosis (mGCS score, 15 to 18).