Seizures following head trauma in dogs: 259 cases (1999–2009)

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Objective—To determine whether dogs with head trauma have a greater incidence of seizures than the general canine patient population.

Design—Retrospective case series.

Animals—259 client-owned dogs.

Procedures—Medical records of dogs evaluated for head trauma at The Ohio State University Veterinary Medical Center from 1999 to 2009 were reviewed. Data were collected regarding the cause of the head trauma, physical examination and neurologic examination findings, comorbidities, and the development of seizures during hospitalization. A telephone survey was conducted to question owners regarding the development of seizures after discharge. Relationships between the nature of the head trauma and the development of seizures were then examined.

Results—3.5% of dogs with head trauma developed in-hospital seizures, and 6.8% of dogs with head trauma for which follow-up information was available developed seizures after hospital discharge, compared with an epilepsy rate of 1.4% in our hospital. Dogs that developed in-hospital seizures were significantly more likely to have been hit by a car or experienced acceleration-deceleration injury. Additionally, 10% of dogs with traumatic brain injury had in-hospital seizures. No visit or patient characteristics were significantly associated with the development of out-of-hospital seizures.

Conclusions and Clinical Relevance—Dogs with head trauma may develop seizures at a greater rate than dogs in the general canine patient population. Particularly in the immediate to early posttraumatic period, clinicians should remain vigilant for the development of posttraumatic seizures and treat patients accordingly. (*J Am Vet Med Assoc* 2012;241:1479–1483)

In veterinary patients with head trauma, the potential for development of seizures is an important concern. Seizures following head trauma have been well described in human patients.[1,2] Presently, there are no studies examining the link between seizures and head trauma in dogs. However, several studies have examined seizure etiology in dogs more broadly. One study[3] of 50 dogs referred to general practitioners for seizures over a 2-year period identified primary idiopathic epilepsy as the most common cause; only 1 dog developed seizures secondary to a traumatic event. In a study[4] that examined the relationship between epileptic seizures and status epilepticus in dogs, approximately 6% of dogs were evaluated for epilepsy secondary to other causes, which included trauma.

Traumatic brain injury occurs in a subset of patients with head trauma. In the human literature, head injury is defined as an event causing clinical evidence of damage to the head, with the exception of injuries confined to the face or foreign bodies of the nose or ears.[5] In contrast, patients with TBI by definition must have some evidence of neurologic dysfunction (loss of consciousness, amnesia, and neurologic deficit) or an intracranial lesion secondary to head injury on imaging studies.[6] For this reason, most studies[7–11] in the human literature that describe seizures following head trauma focus on patients with TBI. In the veterinary literature, there are no specific definitions for either head injury or TBI, although studies of TBI in dogs have characterized patients by their degree of neurologic dysfunction.[12–14]

Abbreviations

CI	Confidence interval
PTS	Posttraumatic seizures
TBI	Traumatic brain injury

Various studies[1,2] have been conducted in human patients that demonstrate an unequivocal relationship between the incidence of developing seizures following TBI and the severity of the inciting injury. One study[1] examined a group of over 4,500 adults and children and found that individuals treated for TBI are between 1.5 and 17.0 times as likely to develop seizures, depending upon the severity and cause of the TBI. A more recent study[2] found that the incidence of developing seizures within 3 years following TBI in human patients ranged from 4.4% to 13.6%, again depending primarily on the severity of the underlying injury. For comparison, a recent meta-analysis showed the 1-year incidence rate of seizures in humans from nontraumatic causes to be 48/100,000 cases, with a prevalence of 0.71%.[15]

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The authors thank Celia Ruckel, Bobbi Schmidt, and Krystal Phillips for their assistance in searching the medical records database and obtaining files for review.

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The primary objective of the study reported here was to investigate whether dogs with head trauma develop seizures at a greater rate than other dogs examined at our hospital. Secondarily, our goal was to characterize canine patients that are referred for medical care following head trauma in terms of their injuries, clinical signs, neurologic status, and outcomes. We hypothesized that the seizure rate among dogs with TBI, but not among dogs with head injury alone, would be significantly higher than the background rate of epilepsy at our hospital. Furthermore, we hypothesized that the seizure rate would be correlated with the severity of the TBI and degree of neurologic impairment.

Materials and Methods

Case selection—The medical records database at The Ohio State University Veterinary Medical Center was searched between March 1999 and September 2009 for dogs with a diagnosis code of injury of the head and also all related categories. Diagnoses related to injury to the eye only or injury to the soft tissues of the mouth only were excluded, with the exception of orbital fractures. The database was also searched for unique medical records of dogs that had a diagnosis of epilepsy at our hospital during this same time period.

Data collection—From each medical record, information was collected regarding the following: patient signalment and history, vital signs on initial examination, evidence of trauma to the head, neurologic examination, initial treatment, number of days in the hospital, comorbidities, laboratory data, and in-hospital seizure activity. All information was recorded by a single author (SGF).

Not all information was available for every medical record. For most data categories, when no information was reported in a medical record, that information was omitted from further analysis. For selected data categories, however, we assumed a normal value when no abnormality was noted in the medical record. These data categories included skull fractures, jaw fractures, bite wounds, external soft tissue injuries to the head, and limb fractures.

Patient classification—Patients with evidence of head injury and patients with evidence of TBI were included. Patients with head injury were defined by physical examination findings consistent with trauma to the head, including skull fractures, jaw fractures, bite wounds, or external soft tissue injuries to the head. Patients with TBI were defined as those with altered mentation or CT and MRI findings supportive of TBI (eg, subdural hematoma, midline shift, and focal edema). We excluded patients from TBI designation if they had signs of depressed mentation and were hypotensive on initial examination (systolic blood pressure ≤ 100 mm Hg) because of the difficulty in distinguishing mentation changes caused by TBI from those caused by cardiovascular instability.

Patients with TBI were assigned a neurologic grade on the basis of the severity of their injuries. Mild TBI was defined by either signs of depressed mentation with normal pupils and normal systolic blood pressure or a normal mentation with CT and MRI findings supportive of intracranial trauma. Moderate TBI was defined by signs of depressed mentation with anisocoria or miosis or by an obtunded or stuporous mentation with normal pupil size, anisocoria, or miosis. Severe TBI was defined by either a comatose mentation or mydriasis.

Telephone survey—We attempted to contact all owners of patients with either a head injury or TBI that survived to discharge. Owners were asked about any seizure activity after discharge from the hospital or prior to admission. If any seizure activity was noted, the date of seizure onset, frequency and character of the seizures, and any treatment provided were recorded.

Exclusion criteria—Patients that had an incomplete medical record were excluded from the study. A complete medical record was defined as including the following: signalment, initial injury, initial vital signs, and mental status at the time of examination. Patients with no physical examination findings supportive of head injury nor any neurologic findings consistent with TBI were excluded. Finally, we excluded patients with a history of prior episodes of either seizures or head trauma from our analysis of PTS.

Statistical analysis—Summary statistics (median and range for continuous variables, frequency, and 95% CI for categorical variables) were determined. Proportions were analyzed with the Fisher exact or χ^2 test, whichever was appropriate on the basis of sample size. Continuous variables were compared by means of a 1-way ANOVA. Bonferroni adjustment was used for multiplicity. Values of $P < 0.05$ were considered significant. All analyses were performed with a commercial software package.[a]

Results

In-hospital records—Two hundred seventy-two dogs were diagnosed with head trauma between 1999 and 2009. Thirteen of these dogs were removed on the basis of our exclusion criteria: 4 had an incomplete medical record, and 9 had no evidence of either head injury or TBI. The remaining patient population of 259 dogs was 53% female. The most common breeds represented were mixed (27.4%), Labrador Retriever (5.4%), Golden Retriever (5.0%), Yorkshire Terrier (4.3%), Chihuahua (3.9%), Jack Russell Terrier (3.5%), and Boxer (3.0%). All other breeds represented < 3% of the patient population. The median age at initial examination was 3.0 years (range, 1 to 16; mean, 4.2 years).

We grouped patients into 3 categories on the basis of their initial injuries: those with head injury and TBI (n = 63), those with head injury only (169), and those with TBI only (27). The medical records review with patients grouped by these injury categories was summarized (**Table 1**).

Both the initial injuries and discharge status of all dogs were significantly ($P < 0.001$) related to injury category. Dogs examined for acceleration and deceleration injuries (ie, thrown, dropped, or fell) were significantly ($P = 0.003$) more likely to have TBI only than a head injury only. Dogs that were discharged from the hospital were significantly ($P < 0.002$) more likely to have a

Table 1—Summary of visit information for dogs with head trauma (n = 259) examined at The Ohio State University Veterinary Medical Center from 1999 to 2009.

Variable	Total No.	Head injury and TBI	Head injury only	TBI only
Total patients	**259**	24%	65%	11%
Initial injury				
Hit by a car	99	42%	45%	12%
Bite wounds	101	9%	89%	2%
Crushed, kicked, or hit	18	11%	72%	17%
Other	16	25%	56%	19%
Acceleration or deceleration	16	38%	19%	44%
Gunshot	9	0%	100%	0%
Head injury evidence				
Skull fractures	42	43%	57%	NA
Jaw fractures	60	22%	78%	NA
Bite wounds	92	9%	91%	NA
Soft tissue injury	117	41%	59%	NA
TBI severity				
Mild	32	81%	NA	19%
Moderate	36	67%	NA	33%
Severe	22	59%	NA	41%
Discharge status				
Alive	227	22%	70%	7%
Euthanized	25	48%	24%	28%
Died	7	14%	43%	43%
Hospitalization duration				
Median	NA	2 d	< 1 d	< 1 d
Range	NA	0–11 d	0–24 d	0–7 d

NA = Not applicable.

head injury only than TBI only. Additionally, there was a significant ($P = 0.049$) difference in hospitalization duration among injury categories. Median hospitalization duration was < 1 day for dogs with only a head injury (range, 0 to 24; mean, 1.2 days), < 1 day for dogs with only TBI (range, 0 to 7; mean, 1.6 days), and 2 days for dogs with a head injury and TBI (range, 0 to 11; mean, 2.1 days). No other data were significantly associated with injury category.

Physical examination findings consistent with head injury were observed in 232 (89.5%) patients; these included 42 patients with skull fractures, 60 patients with jaw fractures, 92 patients with bite wounds to the head, and 117 patients with external soft tissue injuries to the head. One hundred sixty-nine (72.8%) of these dogs had head injury only without TBI. Compared with dogs with head injury and TBI, dogs with head injury only were significantly ($P = 0.021$) older and had a significantly ($P = 0.027$) shorter hospitalization duration.

Neurologic examination findings consistent with TBI were observed in 90 (34.7%) patients. Of these, 32 (35.6%) had mild TBI, 36 (40.0%) had moderate TBI, and 22 (24.4%) had severe TBI. No patients required surgery to alleviate neurologic signs. Severity of TBI was significantly ($P = 0.017$) associated with survival to discharge; 94% of patients with mild TBI were discharged, compared with 72% of patients with moderate TBI and 55% of patients with severe TBI. Patients with TBI (median age, 1.4 years; range, 0 to 15.1 years; mean, 3.3 years) were significantly ($P = 0.005$) younger than patients without TBI (median age, 3.8 years; range, 0.1 to 16 years; mean, 4.7 years) and were significantly ($P = 0.025$) more likely to have had an extended duration of hospitalization (median, 2 days; range, 0 to 11 days; mean, 2.0 days for patients with TBI vs median, < 1 day; range, 0 to 24 days; mean, 1.2 days for patients without TBI). Twenty-seven (30.0%) patients had TBI only without head injury. There were no significant differences in initial injury, comorbidities, hospital duration, or survival time between patients with TBI only and patients with head injury and TBI.

In 59 (22.8%) of the included dogs, at least 1 comorbidity was noted. The vast majority (83.0%) of these dogs were examined for injuries resulting from being hit by a car. These comorbidities included the following: pulmonary contusions (29/101 patients evaluated), pneumothorax (23/103), limb fractures (20/259), pelvic fractures (6/20), rib fractures (8/103), and vertebral fractures (2/14). No comorbidities were significantly associated with any injury category.

A total of 9 patients suffered in-hospital seizures; this corresponds to an incidence rate of 3.5% of the study population (95% CI, 1.6% to 6.5%) and 10% of patients with TBI (95% CI, 4.7% to 18.1%). Two dogs had 3 seizures each, 1 dog had 2 seizures, and the remaining 6 dogs had 1 seizure each. Seven dogs had generalized seizures only, 1 dog had focal seizures only, and 1 dog had both generalized and focal seizures. The initial injury of the dogs at the time of hospital admission was significantly ($P = 0.008$) associated with the development of in-hospital seizures. The initial injuries of these dogs were being hit by a car (n = 5), acceleration-deceleration injury (3), and unknown (1). The presence of TBI was also significantly ($P < 0.001$) associated with the development of in-hospital seizures. All dogs with in-hospital seizures had either moderate or severe TBI. Five of the 9 dogs were actively seizing at the initial examination. Only one of the dogs with

seizure activity on initial examination was hospitalized, and that dog did not develop any further seizures in the hospital. Among the 4 dogs that had seizure activity after hospital admission, 2 had seizures shortly after admission (3 to 6 hours) and 2 had seizures several days after admission (> 48 hours). Six of the 9 patients with in-hospital seizures survived to discharge; the remainder were euthanized. There was no association between in-hospital seizures and survival to discharge.

In addition to our study patients, we found 827 dogs that had a diagnosis of epilepsy during the study period, compared with 57,626 canine patients examined at The Ohio State University Veterinary Medical Center. This corresponded with an epilepsy rate of 1.4% at our hospital.

Survey—We were able to contact the owners of 74 of 227 (32.6%) dogs that survived to discharge. All contacted owners agreed to participate in our survey. There were significant (P = 0.014) differences in the initial injuries among dogs for which owners were contacted and were not contacted, but no differences were noted for any other visit or patient characteristics. We were only able to contact the owners of 1 of the 6 dogs with in-hospital seizures that survived to discharge, and that patient did not have any subsequent seizures.

Of the 74 dog owners contacted, 5 reported their dogs had seizures after hospital discharge. This corresponded to a prevalence of 6.8% (95% CI, 2.2% to 15.1%). All dogs with out-of-hospital seizures had at least 2 seizure events, and all were generalized seizures. One dog had seizures within 1 month after discharge, and 3 dogs had seizures > 4 years after discharge; data were unavailable for 1 dog. The dogs ranged in age from 4 months to 16 years at the time of seizure onset. Two of the 5 dogs with out-of-hospital seizures had mild TBI on inital examination, and 3 of these dogs had head injury only on initial examination. Two of the 5 dogs with out-of-hospital seizures were treated with antiepileptic drugs. The development out-of-hospital seizures was not significantly related to the initial reasons for hospitalization or to neurologic grade.

Discussion

The results of the present study suggest that dogs with head trauma may develop seizures at a greater rate than dogs in the general patient population. When compared with the prevalence of epilepsy in the canine patient population at our hospital, dogs with head injury, TBI, or both may be at increased risk for developing seizures.

In human patients, PTS are typically categorized into immediate (< 24 hours after injury), early (< 7 days after injury), and late (> 8 days after injury) onset.[9] Both immediate and early seizures are considered to be direct reactions to brain damage, whereas late seizures are believed to occur as a result of secondary effects such as bleeding, cortical scarring, and reperfusion injury.[9,16] We found the rate of out-of-hospital (late) seizures in the dogs of the present study to be greater than the rate of in-hospital (early) seizures (6.8% vs 3.5%). The increased occurrence of late-onset seizures we observed is consistent with what has been reported in the human literature.[2,16]

However, it is critical to note that 90% of late-onset seizures in humans occur by 18 to 24 months following TBI and that human patients with late-onset PTS generally have severe neurologic impairment on examination.[9] In contrast, only 1 of the 5 patients in our study with late PTS had seizures within this time frame, and the patients we reported with late PTS had either absent or mild neurologic signs on examination. Two of these 5 dogs also had seizure onset late in life (ages 13 and 16), which could be consistent with other seizure etiologies. It is therefore likely that seizures in these patients were unrelated to their prior head trauma and thus were not truly related to PTS. Given these caveats, it is difficult to effectively interpret the prevalence of out-of-hospital seizures reported in the present study. A prospective investigation with follow-up advanced imaging is warranted to differentiate canine patients with PTS from those that develop late-onset seizures for other reasons.

Despite the low likelihood that patients with head injury without TBI would develop PTS, we chose to review all patients with head trauma in our study. The incidence of PTS in dogs has not been previously described, making it difficult to completely exclude the possibility that patients with head injury alone could develop PTS. Additionally, one of our objectives was to characterize the population of dogs that was referred to our emergency department for head trauma. The inclusion of patients with head injury alone was pertinent to this objective.

In the present study, all patients with in-hospital (early) PTS were administered antiepileptic drugs; however, only 2 of 5 patients with out-of-hospital (late) PTS were treated with anticonvulsants. Current guidelines for human patients recommend intervention for early PTS with antiepileptic drugs for at least 1 week.[9,17] However, no consensus currently exists among human neurologists regarding PTS prophylaxis.[17–19] A recent review of both observational and prospective studies examining the usefulness of prophylactic antiepileptic drugs in humans described only mixed benefits.[19] Management strategies for PTS in dogs have yet to be critically evaluated, and future studies examining specific antiepileptic drug protocols for TBI and PTS are needed. In the absence of these studies, current guidelines recommend the use of diazepam for active seizure control and phenobarbital for further seizure prevention.[20]

There are several limitations to the present study. First, the retrospective nature of this study prevented us from obtaining a complete, standardized neurologic evaluation or using the modified Glasgow Coma Scale to categorize our patients. There was likely some variability in the clinicians' assessment of subjective parameters such as mental status. This may have led to misclassification of the neurologic grade for some patients. Second, we assumed certain physical examination findings to be normal in the absence of reported abnormal signs (skull fractures, jaw fractures, bite wounds, external soft tissue injuries to the head, and limb fractures). Although we believe that this was a reasonable assumption because these abnormalities were likely to be detected on even a cursory physical

examination, this may have led to a misclassification of certain patients with head injury or other comorbidities. Finally, we succeeded in contacting owners of only 74 of the 227 discharged patients with head injury, TBI, or both in our follow-up survey, and there was a significant difference in the initial injuries among these groups. On the basis of recent studies[21–23] of recruitment and contact rates in telephone surveys, our 33% response rate is in the range of what is typically expected by epidemiologists. However, this response rate may have led to an under- or overrepresentation of the incidence of late PTS.

This study represents the first attempt to characterize the prevalence of PTSs in dogs. Relative to the prevalence of epilepsy in the general canine population at our hospital, dogs with a head injury, TBI, or both may be at increased risk for developing seizures. Further prospective studies are warranted to collect additional data and to determine the optimal long-term management of affected patients.

a. SAS for Windows, version 9.2, SAS Institute Inc, Cary, NC.

References

1. Annegers JF, Hauser WA, Coan SP, et al. A population-based study of seizures after traumatic brain injuries. *N Engl J Med* 1998;338:20–24.
2. Ferguson PL, Smith GM, Wannamaker BB, et al. A population-based study of risk of epilepsy after hospitalization for traumatic brain injury. *Epilepsia* 2010;51:891–898.
3. Podell M, Fenner WR, Powers JD. Seizure classification in dogs from a nonreferral-based population. *J Am Vet Med Assoc* 1995;206:1721–1728.
4. Zimmermann R, Hülsmeyer V-I, Sauter-Louis C, et al. Status epilepticus and epileptic seizures in dogs. *J Vet Intern Med* 2009;23:970–976.
5. Jennett B. Epidemiology of head injury. *J Neurol Neurosurg Psychiatry* 1996;60:362–369.
6. Blyth BJ, Bazarian JJ. Traumatic alterations in consciousness: traumatic brain injury. *Emerg Med Clin North Am* 2010;28:571–594.
7. Lowenstein DH. Epilepsy after head injury: an overview. *Epilepsia* 2009;50(suppl 2):4–9.
8. Pitkänen A, Immonen RJ, Gröhn OHJ, et al. From traumatic brain injury to posttraumatic epilepsy: what animal models tell us about the process and treatment options. *Epilepsia* 2009;50(suppl 2):21–29.
9. Agrawal A, Timothy J, Pandit L, et al. Post-traumatic epilepsy: an overview. *Clin Neurol Neurosurg* 2006;108:433–439.
10. Frey LC. Epidemiology of posttraumatic epilepsy: a critical review. *Epilepsia* 2003;44(suppl 10):11–17.
11. Temkin NR. Risk factors for posttraumatic seizures in adults. *Epilepsia* 2003;44(suppl 10):18–20.
12. Platt SR, Radaelli ST, McDonnell JJ. The prognostic value of the modified Glasgow Coma Scale in head trauma in dogs. *J Vet Intern Med* 2001;15:581–584.
13. Platt SR, Radaelli ST, McDonnell JJ. Computed tomography after mild head trauma in dogs. *Vet Rec* 2002;151:243.
14. Platt SR, Adams V, McConnell F, et al. Magnetic resonance imaging evaluation of head trauma in 32 dogs; associations with modified Glasgow coma score and patient outcome. *J Vet Intern Med* 2007;21:1145.
15. Hirtz D, Thurman DJ, Gwinn-Hardy K, et al. How common are the "common" neurologic disorders? *Neurology* 2007;68:326–337.
16. Asikainen I, Kaste M, Sarna S. Early and late posttraumatic seizures in traumatic brain injury rehabilitation patients: brain injury factors causing late seizures and influence of seizures on long-term outcome. *Epilepsia* 1999;40:584–589.
17. Bratton SL, Chestnut RM, Ghajar J, et al. Guidelines for the management of severe traumatic brain injury. XIII. Antiseizure prophylaxis. *J Neurotrauma* 2007;24(suppl 1):S83–S86.
18. Iudice A, Murri L. Pharmacological prophylaxis of post-traumatic epilepsy. *Drugs* 2000;59:1091–1099.
19. Chen JWY, Ruff RL, Eavey R, et al. Posttraumatic epilepsy and treatment. *J Rehabil Res Dev* 2009;46:685–696.
20. Sande A, West C. Traumatic brain injury: a review of pathophysiology and management. *J Vet Emerg Crit Care* 2010;20:177–190.
21. Hocking JS, Lim MS, Read T, et al. Postal surveys of physicians gave superior response rates over telephone interviews in a randomized trial. *J Clin Epidemiol* 2006;59:521–524.
22. O'Toole J, Sinclair M, Leder K. Maximising response rates in household telephone surveys. *BMC Med Res Methodol* 2008;8:71–75.
23. Kennedy C, Dimock M, Best J, et al. Gauging the impact of growing nonresponse on estimates from a national RDD telephone survey. *Public Opin* 2006;70:759–779.