Clinical signs associated with ingestion of black walnut tree (*Juglans nigra*) wood, nuts, and hulls in dogs: 93 cases (2001–2012)

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OBJECTIVE

To identify clinical signs associated with oral exposure to black walnut tree (*Juglans nigra*) wood, nuts, or nut hulls in dogs and to compare clinical syndromes between dogs that ingested wood and dogs that ingested the walnuts or nut hulls.

DESIGN

Retrospective case series.

ANIMALS

93 dogs.

PROCEDURES

Records of dogs with oral exposure to black walnut wood, nuts, or nut hulls between November 2001 and December 2012 were retrieved from the Animal Poison Control Center database. Records were reviewed, and data regarding signalment; exposure; time of onset, type, and duration of clinical signs; serum biochemical abnormalities; treatment; and response to treatment were collected. Results were compared statistically between dogs that ingested wood and those that ingested nut components.

RESULTS

28 cases involved exposure to wood, and 65 involved exposure to nuts or hulls. Spontaneous vomiting was commonly observed (13/28 [46%] and 31/65 [48%] dogs that ingested wood and nut components, respectively). Neurologic or musculoskeletal signs were significantly more common in dogs that ingested wood (26/28 [93%]) than in those that ingested nuts or hulls (15/65 [23%]). Relative risk of developing neurologic signs after ingestion of wood was approximately 4 times that after ingestion of nuts or hulls.

CONCLUSIONS AND CLINICAL RELEVANCE

Ingestion of black walnut wood by dogs resulted in a clinical syndrome in which neurologic or musculoskeletal signs were most frequently reported, whereas ingestion of black walnuts or their hulls was most commonly associated with vomiting. To our knowledge, this is the first report describing 2 different clinical syndromes associated with exposure to black walnut tree components in dogs. (*J Am Vet Med Assoc* 2016;248:195–200)

Black walnut trees (*Juglans nigra*) are commonly found in parks and forests throughout eastern North America.[1] This hardy heartwood tree is used in landscaping and is commonly grown for wood or the edible nuts.[1] The trees and their nuts are often found in private yards and may be available to dogs when nuts fall, branches break off, or trees are cut down.

Serious clinical signs of toxicosis have been described in a clinical report of a dog that ingested walnuts that were moldy and contaminated with tremorgenic mycotoxins.[2] These mycotoxins can cause muscle tremors, vomiting, hyperthermia, seizures, and tachycardia in dogs.[3] Black walnut shavings used as bedding have been associated with laminitis in horses,[4] but to the authors' knowledge, no illnesses attributed to exposure to black walnut wood or wood shavings have been reported in other species, and the literature contains no reports of clinical signs in dogs that have ingested black walnut wood or nuts.

Veterinary staff members at the ASPCA APCC, a 24-hour consultation service that receives calls from throughout the United States and Canada concerning accidental or environmental animal poisonings from animal owners and veterinarians, observed that dogs reported to ingest black walnut wood seemed to have different clinical signs of toxicosis than did dogs reported to ingest black walnuts or black walnut hulls. The purpose of the study reported here was to identify clinical signs associated with oral exposure to black walnut wood, nuts, or nut hulls in dogs and to compare clinical syndromes between dogs that ingested wood and those that ingested black walnuts or their

ABBREVIATIONS

APCC Animal Poison Control Center
ASPCA American Society for the Prevention of Cruelty to Animals

hulls. Our hypothesis was that there would be a difference in the types of signs seen following ingestion of these black walnut tree components.

Materials and Methods

Case selection

The electronic ASPCA APCC toxicology database was searched for records of dogs reported to have oral exposure to wood (including shavings, chips, branches, or sawdust), walnuts, or walnut hulls from black walnut trees between November 1, 2001, and December 31, 2012. Cases included in the study were all single-exposure events with witnessed oral exposure or strong evidence of exposure (such as a chewed up empty bag of nuts or evidence of walnuts or wood in a dog's vomitus). Cases were excluded from if there was any known or suspected additional exposure to any other toxicant, if they involved exposure to visibly moldy nuts (a risk for exposure to tremorgenic agents) or concurrent exposure to nuts and wood, if they involved ingestion of water in which black walnuts or black walnut wood had been soaking, or if the substance involved was a black walnut dietary supplement.

Data collection and medical records review

The following information on each dog, collected by APCC staff members at the time of telephone consultation, was retrieved from the records: breed (or predominant breed if mixed), sex, age, body weight, number of animals exposed and at risk of exposure, number of animals with clinical signs, source of exposure, amount of the product ingested (if known), and assurance of exposure (ie, whether exposure was observed or evidenced by other findings). Information regarding time of onset, type, and duration of clinical signs; serum biochemical alterations; treatment; and response to treatment was also collected. Follow-up calls were made to update progression of clinical signs, response to treatment, and final outcome in cases (wherever available) included in this study. Dogs were considered to have developed neurologic or musculoskeletal signs if ≥ 1 of the following signs was reported: sedation or subdued behavior, lethargy, generalized weakness, hind limb weakness, ataxia, stiffness, apparent disorientation, or tremors or fasciculations. Hyperthermia was defined as rectal temperature > 38.9°C (102°F).

Statistical analysis

A Fisher exact test was used to assess differences in the frequency (proportion) of dogs that had neurologic or musculoskeletal signs after oral exposure to black walnuts or their hulls, compared with that of dogs with oral exposure to wood from the black walnut tree. Relative risk was calculated to provide an estimate of the magnitude of the relationship between exposure to black walnut wood and development of neurologic or musculoskeletal signs. A χ^2 test was used to determine if any breeds were significantly overrepresented among these cases, compared with the usual frequency of calls involving the same breed and mixes of the breed at the APCC over the same time period. A statistical software package[a] was used for all comparisons. Values of $P < 0.05$ were considered significant.

Results

A total of 93 dogs were identified that met the case criteria for study inclusion during the 11-year study period. Of these, 28 (30%) dogs had ingested black walnut wood or wood shavings, and 65 (70%) had ingested the walnuts or hulls.

Cases involving black walnut wood

Of the 28 dogs that ingested wood, 14 (50%) were observed to have ingested shavings from black walnut trees or otherwise had evidence of consuming the shavings (in 1 case, the wood shavings were observed in vomitus); 14 (50%) dogs were seen chewing on wood or branches. Cases occurred throughout the year but were most commonly reported in January, February, and April (12/28 cases). Calls about black walnut wood exposure came primarily from eastern North America (the United States [n = 12] and Ontario, Canada [1]). A few cases (3) from the west coast involved shavings created during furniture making. Of the 11 cases involving wood consumption where it ascertained the tree grew (ie, it was stated that the tree was on the premises), 3 each were in Pennsylvania and Michigan, 2 in New York, and 1 each in Ontario, Iowa, and Maryland.

The dogs in this group ranged in age from 0.25 to 5.5 years (mean ± SD, 2.2 ± 2.17 years; median, 1.75 years); 20 of 28 (71%) dogs were ≤ 2 years old. Body weight ranged from 8.2 to 45.1 kg (18 to 99.2 lb; mean ± SD, 30.9 ± 9.4 kg [68.0 ± 20.7 lb]; median, 31.7 kg [69.7 lb]), with most (22 [79%]) dogs weighing > 25 kg (55.1 lb). Fifteen (54%) were male (11 neutered and 4 sexually intact) and 13 (46%) were female (7 spayed and 6 sexually intact). The breeds most commonly represented were Labrador Retriever (8/28 [29%]), Golden Retriever (6 [21%]), and Boxer or Boxer mix (5 [18%]).

Clinical signs were noted in all 28 (100%) cases in which dogs consumed wood or wood shavings. The time to onset of clinical signs ranged from 0.17 to 19 hours after the exposure was observed (n = 27 dogs). Vomiting, when present, developed ≤ 1 hour after exposure. Some type of neurologic or musculoskeletal signs developed in 26 of these 28 dogs (93%), including ≥ 1 of the following: generalized weakness, hind limb weakness, lethargy or subdued behavior, ataxia, evidence of disorientation, reluctance to move, stiffness, or tremors **(Table 1)**. The most commonly reported clinical signs for this group of dogs included lethargy or subdued behavior (14/28 [50%] dogs), generalized or hind limb weakness (13 [46%]), vomiting (13 [46%]), stiffness (8 [29%]), ataxia (7 [25%]), and tremors or fasciculations (7 [25%]). The duration of clinical signs ranged from 1 to 33.25 hours (mean ± SD, 14.4 ± 2 hours; median, 9 hours).

Table 1—Clinical signs in 93 dogs following ingestion of wood (n = 28) versus nuts or nut hulls (65) from black walnut trees (*Juglans nigra*).

Observation	Wood ingestion (No. [%] with sign)	Nut ingestion (No. [%] with sign)
Lethargy or subdued behavior	14 (50)	6 (9)
Generalized or hind limb weakness	13 (46)	1 (2)
Tremors or fasciculations	7 (25)	2 (3)
Stiffness	8 (29)	0 (0)
Ataxia	7 (25)	1 (1.5)
Anorexia	5 (18)	4 (6)
Hesitancy to move	4 (14)	0 (0)
Vomiting	13 (46)	31 (48)
Diarrhea	1 (4)	5 (8)
Hyperthermia*	3 (11)	2 (3)
Disorientation	2 (7)	2 (3)
Generalized erythema or erythema of the pinnae	2 (7)	0 (0)
Signs of pain	2 (7)	0 (0)
Difficulty walking	1 (4)	0 (0)
Lameness	1 (4)	0 (0)
Drooling	2 (7)	3 (5)
High liver enzyme activities†	1 (4)	2 (3)
Seizures	0 (0)	3 (5)

Data were retrospectively collected from electronic records of the ASPCA APCC toxicology database between November 2001 and December 2012. Cases included in the study were single-exposure events with witnessed oral exposure or strong evidence of exposure (such as a chewed-up empty bag of nuts or evidence of walnuts or wood in a dog's vomitus). Cases were excluded if there was any known or suspected additional exposure to any other toxicant, if they involved exposure to visibly moldy nuts (a risk for exposure to tremorgenic agents) or concurrent ingestion of nuts and wood, if they involved ingestion of water in which black walnuts or black walnut wood had been soaking, or if the substance ingested was a black walnut dietary supplement. At least 1 clinical sign was observed in 28 of 28 (100%) dogs that ingested wood and 40 of 65 (62%) dogs that ingested nuts or nut hulls; some dogs had multiple signs.

*Defined as rectal temperature > 38.9°C (102.0°F). †Includes serum alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, and γ-glutamyltransferase activity exceeding the upper limit of the reference range used by the reporting veterinarian or laboratory.

Twenty of the 28 dogs went to a veterinary hospital for treatment and received supportive care with various treatments, most frequently including IV fluid administration in 4 dogs and fluids by an unspecified route in 5 dogs. Other treatments included methocarbamol (1), and antiemetics (3). None of the dogs were induced to vomit, but 13 (46%) did vomit spontaneously. Of the 13 dogs that vomited, 11 developed neurologic or musculoskeletal signs and the other 2 were the only dogs with wood exposure that did not develop such signs. Outcome information was obtained in 13 cases; all 13 of these dogs (8 of which were treated at a veterinary hospital and 5 of which were not) had a full recovery. There were no reported deaths.

Cases involving black walnut nut or hull ingestion

The 65 cases involving ingestion of black walnuts or nut hulls occurred throughout the year, but most commonly in September (n = 11), October (16), and December (8). Exposures were reported in all regions (north, south, east and west) of the United States and in Ontario, Canada.

The ages of dogs in this group ranged from 0.1 to 14 years (mean ± SD, 3.5 ± 3.4 years; median, 2 years). Body weight ranged from 1.6 to 68 kg (3.5 to 149.6 lb; mean ± SD, 21.5 ± 16.5 kg [47.4 ± 36.3 lb]; median, 20.2 kg [44.4 lb]). Thirty-nine (60%) dogs were female (28 spayed, 9 sexually intact, and 2 of unknown reproductive status) and 26 (40%) were males (20 neutered and 6 sexually intact). The breeds most commonly involved were Labrador Retriever (13/65 [20%]), Golden Retriever (6 [9%]), and Jack Russell or Parson Russell Terrier (4 [6%]).

Clinical signs were observed in 40 of 65 (62%) cases. The time to onset of clinical signs after exposure was observed (n = 37 dogs) ranged from 0.02 to 192 hours. The most commonly reported clinical signs included vomiting (31 of 65 [48%] dogs), lethargy or subdued behavior (6 [9%]), diarrhea (5 [8%]), and anorexia (4 [6%]; **Table 1**). Fifteen of 65 (23%) dogs developed ≥ 1 of the following neurologic or musculoskeletal signs: lethargy, apparent disorientation, tremors or fasciculations, ataxia, seizures, and generalized or hind limb weakness.

Seventeen of the 65 (26%) dogs in this group were treated at a veterinary hospital, most commonly by IV or SC fluid administration (n = 6) and antiemetics (2). Two (3%) dogs had vomiting induced at the hospital, and of these, 1 dog developed diarrhea and the other developed ataxia and tremors. Of the 31 dogs that vomited spontaneously, 10 developed ≥ 1 neurologic or musculoskeletal sign. Further follow-up information for these dogs was not available.

Comparisons of dogs that ingested black walnut wood with those that ingested nuts or nut hulls

Evaluation of the frequency of neurologic or musculoskeletal signs in each group (26/28 [93%] dogs that ingested wood vs 15/65 [23%] that ingested tree nut components) revealed that these signs were significantly ($P < 0.001$ [2 tailed]) more common in dogs that ingested wood. The relative risk of developing neurologic or musculoskeletal signs after ingestion of black walnut wood in dogs was 4.02 times that for dogs that consumed nuts or nut hulls (95% confidence interval, 2.55 to 6.34; $P < 0.001$).

Results of statistical analysis also showed that some breeds were overrepresented in these cases. During the study period, 123,759 of 822,647 (15%) calls placed to the APCC regarding dogs involved Labrador Retrievers, 41,916 (5%) involved Golden Retrievers, 23,941 (2.9%) involved Boxers, and 18,763 (2.3%) involved Jack Russell or Parson Russell Terriers (or mixes of these breeds). For cases involving consumption of black walnut wood, the frequency of involvement for the 3 most commonly involved breeds (Labrador Retrievers, Golden Retrievers, and Boxers [including mixes of these breeds]) was significantly ($P < 0.05$ for all comparisons) higher than expected. Similarly, Jack Russell or Parson Russell terriers were more frequently represented among cases of nut or nut hull consumption than expected ($P < 0.05$).

Discussion

The results of the present retrospective case series provided evidence that, in dogs, ingestion of black walnut wood is associated with a clinical syndrome of toxicosis different from that which develops following ingestion of black walnuts or their hulls. Dogs that ingested wood were approximately 4 times as likely to develop neurologic or musculoskeletal signs as were those that ingested the black walnuts or their components. For one of the dogs that developed neurologic signs after consuming the nuts, the signs were not evident until 192 hours (approx 8 days) after exposure. The authors do not believe that it is likely that these signs were related to the observed ingestion of walnuts (considering that, once the study was completed, it was inconsistent with the other cases), but because some toxicants have a delayed effect, cases were not excluded on the basis of time to onset of clinical signs. If these signs were not attributable to the witnessed exposure to nuts, this indicates that dogs ingesting the wood would have an even higher relative risk of developing neurologic or musculoskeletal signs, compared with that of dogs ingesting the nuts or nut hulls, than was calculated.

The amount of wood or shavings consumed in most of the cases could not be determined, but in some dogs, it appeared that even small exposures resulted in clinical effects. For example, 1 case involved a 2.5-year-old 27.3-kg (60-lb) Golden Retriever and another involved a 6-year-old 29.5-kg (65-lb) Labrador Retriever, both of which had licked sawdust off of a pair of boots; in a different case, a 3-year-old 31.8-kg (70-lb) Golden Retriever consumed what was estimated as 1 tablespoon of sawdust; and in yet another, a 1-year-old 43.2-kg (95-lb) Bernese Mountain Dog had been chewing on a bone after it was dropped into sawdust. All of these large-breed dogs developed clinical signs after ingestion of relatively small amounts of wood. This may indicate that dogs are sensitive to small amounts of black walnut wood and that any exposure warrants observation for clinical signs. Labrador Retrievers, Golden Retrievers, and Boxers or mixes of these breeds had reported black walnut wood exposure more frequently than would be expected on the basis of the typical percentage of calls related to these breeds that were received by the APCC annually during the study. It is not known if there is any clinical importance to this, or whether there is any true breed predisposition to development of clinical signs following ingestion of black walnut tree wood; it may merely indicate that the retrieving breeds are more likely to find and chew on sticks or branches.

The range of the black walnut tree is wide in the eastern half of North America; it extends from southern Ontario to the Gulf coast in some states and from Nebraska and Kansas to the east coast.[1] Of cases involving wood consumption where it is certain that the tree grew locally, 3 each occurred in Pennsylvania and Michigan, 2 in New York, and 1 each in Iowa, Maryland, and Ontario. Overall, cases involving wood consumption were noticeably lacking from the southern regions where the black walnut tree is distributed. Only 1 case (in North Carolina) was considered as being from the South, but this case involved a dog that ingested shavings while the owner was making black walnut wood furniture, and the origin of the wood was unknown. It might be that trees in the northern part of the range produce a toxicant that is not present throughout the range, or it might be that the tree grows less frequently in the South. This would be an interesting avenue for further research. Cases involving wood consumption occurred throughout the year and in every month except for December, and there did not appear to be any single season in which a preponderance of cases occurred.

The mechanism of action by which black walnut wood affects dogs is not known. Juglone is a component of black walnuts (found in highest concentrations in the buds, nut hulls, and roots) that is known to cause clinical effects in several species. Juglone extract has been reported to cause sedative effects in fish (concentrations > 0.15 mg/L), mice (1 to 7.5 mg/kg [0.45 to 3.41 mg/lb], IP), rats (2.5 to 15 mg/kg [1.14 to 6.82 mg/lb], IV or IP), and rabbits (unspecified dose, IV).[5] It was found that when mice received juglone IP and rats received juglone IV, they became very sedate and developed hind limb paresis or paralysis (described as dragging of the hind limbs). The same research group found that IV (marginal ear vein)

administration of juglone to rabbits resulted in sedation and vasodilation of the vasculature in the ear.[5] In anesthetized dogs given juglone extract by constant-rate infusion (2 mg/kg/h [0.9 mg/lb/h]) IV, no change in heart rate, blood pressure, or depth of anesthesia was observed.[5] In a similar study,[6] when juglone (5.0 mg/kg [2.3 mg/lb] at a rate of 1.0 mL/1.5 minutes) was administered to anesthetized dogs IV, no changes in heart rate or blood pressure were detected, but increases in respiratory rate and Hct were identified, and cyanosis was seen. Necropsy findings included gross hemorrhage and congestion of the lungs, and histopathologic evaluation revealed interstitial and alveolar edema of the lungs with a leukocytic exudate and focal atelectasis as well as focal hepatic necrosis.[6]

Juglone can act as a CNS depressant in some species,[5] so it is possible that juglone played a role in the development of the clinical signs reported in the present retrospective study of dogs. Although when given IV to anesthetized dogs under experimental conditions, no further depressant effect was observed,[5] it is possible that subtle effects could have been undetectable because of anesthesia. This could be consistent with observations for some dogs of the present report, considering that the signs were generally mild, but at this point there is not enough information to make such a determination. Some differences may be attributable to the dose of juglone (which would be unknown in this study) or route of exposure.

Juglone is known to uncouple oxidative phosphorylation.[7] The signs seen in the dogs of the present report could fit with an exposure to an uncoupler of oxidative phosphorylation. Only 3 of 28 (11%) dogs were reportedly hyperthermic after wood exposure, which would be expected with uncoupling of oxidative phosphorylation. However, many of these dogs were not examined by a veterinarian and it is likely body temperature was not measured in many cases. Thus, the proportion of dogs reported as having hyperthermia may have been artificially low.

Anecdotally, in many of the cases in this study, there were comments that the shavings or sawdust were from a freshly cut tree or a recently cut piece of lumber. Hydrojuglone is rapidly converted to juglone (the active agent) when exposed to air.[8] If an association can be identified between clinical signs and exposure to freshly produced shavings or recently chewed branches, this could suggest that juglone is involved; however, the retrospective design of the present study did not allow for this.

In addition to juglone, there may be other pharmacologically active constituents in black walnut that are responsible for the signs seen in the present study. Wood shavings from these trees are known to cause laminitis when used as bedding for horses, but juglone is not considered the causative agent. The syndrome has been reproduced when aqueous extracts of the heartwood, which does not contain juglone, were given to horses via stomach tube.[4] Hardwoods, including *Juglans* spp, have been shown to contain biologically active compounds that interact with neurotransmitter receptors such as γ-aminobutyric acid type A, N-methyl-D aspartate, acetylcholinesterase, and dopamine in fish,[9] and it is possible that one of these compounds affects mammalian neurotransmitters as well.

Black walnuts are also a known substrate for growth of tremorgenic mycotoxins, which can cause severe muscle tremors and hyperthermia when ingested by dogs.[2] It is not thought that the signs seen following wood exposure in the present study were consistent with exposure to tremorgens, because the signs seen were much milder than those previously described in dogs exposed to tremorgenic mycotoxins.[2,3] Although mold contamination was not observed by callers, it is possible that some of the dogs reported to have tremors had ingested products with tremorgenic mycotoxin contamination. The callers may not have inspected the wood or nuts closely, or fungal contamination may not have been visible. Although the authors believe that the signs were generally not consistent with the severity of signs seen with tremorgenic mycotoxins, it was not possible to completely rule out their involvement.

The present study had important limitations, and the results should be considered with these in mind. This type of epidemiological study cannot provide definitive conclusions in many instances, but the information is helpful in trying to identify associations between black walnut exposure and the clinical signs and outcomes that may be seen. It is also useful to help guide further research, possibly including a controlled prospective study to attempt to reproduce this syndrome. Other limitations include the inability to obtain follow-up information in some cases, which limited available data. Another limitation is that, because of the nature of calls to the APCC, we are reliant on other observers to describe the clinical signs seen and to identify the agent involved, and these include pet owners as well as veterinarians. Thus, it is possible that errors could occur because of inaccurate perceptions or faulty recall by the callers.

The results of the present study showed that the clinical syndrome associated with ingestion of black walnut wood in dogs is distinctly different from that which develops after ingestion of black walnuts or their hulls. Dogs that ingested black walnut wood were approximately 4 times as likely to develop neurologic or musculoskeletal signs (including lethargy, generalized or hind limb weakness, stiffness, ataxia, apparent disorientation, and tremors or fasciculations) than were dogs that ingested black walnuts or hulls. All dogs with follow-up information had a complete recovery with or without veterinary care. Dogs appear to have a good prognosis following oral exposures to the wood of black walnut trees, and life-threatening effects are not anticipated, but it is important for clinicians to be aware of the potential clinical effects identified in this report. Follow-up for dogs that consumed nuts or nut hulls was insufficient to suggest prognosis. The cause of the reported signs remains unknown, and further studies are warranted.

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Footnotes

a. Stata Statistical Software, Release 11, Statacorp LP, College Station, Tex.

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