Case Series

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Use of orally administered dexmedetomidine to induce emesis in cats

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Abstract

Case series summary This case series describes the use of orally administered dexmedetomidine at a dose of 20 µg/kg to induce emesis in six cats. Emesis was successfully induced in 5/6 cats, with each of the cats vomiting once. The reasons for inducing vomiting included known or suspected ingestion of lilies, onions, acetaminophen (paracetamol) or acetylsalicylic acid. Four of the five cats in which emesis induction was successful did not develop any clinical signs of toxicity associated with the toxin ingested; the fifth cat developed clinicopathological changes consistent with acetaminophen toxicity. All six cats exhibited moderate to profound sedation, as expected, but no other adverse effects were documented.

Relevance and novel information Induction of emesis in cats is notoriously difficult. This case series describes a novel route of administration of dexmedetomidine, a commonly available medication, with a high success rate observed for inducing emesis in this group of cats.

Plain language summary

Cats are notoriously more difficult to elicit vomiting in than dogs. This case series describes the use of a novel way of giving cats a commonly available veterinary medication to cause vomiting. The medication, dexmedetomidine, was given by mouth to six cats, of which five vomited. All six cats had eaten toxins: lilies, acetaminophen (paracetamol), aspirin or onions. Four of the five cats that vomited did not develop any signs of toxicity. All six cats that received the medication became sedated, but no other side effects were noted.

Keywords: Emesis; transmucosal; dexmedetomidine; α_2-agonists

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Introduction

The intentional induction of emesis in cats is generally accepted to be challenging.[1,2] Reported success rates have been described in the range of 43–75%, compared with success rates above 90% in dogs given a variety of emetics.[1,3,4] Although not as often as dogs, cats frequently ingest toxic substances or foreign objects that may potentially be life-threatening or produce significant morbidity.[5] Some reported toxins ingested by cats include onions, garlic, acetaminophen (paracetamol) and lilies.[6,7] Emetic agents that have been used in cats include oral hydrogen peroxide, xylazine hydrochloride administered intravenously (IV) or intramuscularly (IM), dexmedetomidine hydrochloride administered IM or IV, and hydromorphone administered subcutaneously (SC).[2] However, the use of hydrogen peroxide is not recommended due to the risk of necroulcerative hemorrhagic gastritis.[8] All other emetic agents have had variable success rates in

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inducing emesis in cats, which is often problematic when gastrointestinal decontamination is desired. Recently, the use of transmucosal detomidine was evaluated for sedation in cats.[9] In that study, all cats displayed signs of nausea and 100% (7/7) of cats vomited within 2 mins of drug administration. Although not specifically studied, it was hypothesized that oral administration of alpha$_2$ (α_2)-agonists in cats may induce emesis more reliably than IV or IM administration. This case series describes six cats in which emesis induction was attempted with oral administration of dexmedetomidine.

Case series description

Case selection

Medical records of cats administered dexmedetomidine orally were collected by one author (KM), who was present for all cases at a satellite emergency clinic of the University of Florida (UF Pet Emergency Treatment Services – PETS) over a 5-month period between March 2023 and August 2023. Records were reviewed and were included if cats had been given dexmedetomidine orally in an attempt to induce emesis.

Medical record review

From each cat's medical record, the following data were recorded in an Excel spreadsheet (Microsoft Corp): signalment; weight; reason for emesis induction; dose of dexmedetomidine; whether emesis was successfully induced; when noted in the record, elapsed time from administration until emesis; when noted in the record, degree of sedation; and dose and route of administration of the reversal agent atipamezole. Time until emesis was defined as the interval, in minutes, from administration of the emetic agent until vomiting occurred. If the exact time of vomiting was not noted, then time to administration of reversal agent, defined as the time interval of the emetic agent and when the reversal drug was administered to counter the sedative effect of the emetic agent, was noted instead of time until emesis.

Drug dose and administration

All cats were weighed and given 20 µg/kg dexmedetomidine orally. The syringe containing the dose was positioned over the back of the tongue for administration, but no efforts were made (such as holding the mouth shut or giving a small amount of water) to encourage swallowing.

Results

The induction of emesis with oral dexmedetomidine was attempted in six cats. The six cats included four neutered males and two intact males. The median age was 1 year (range 14 weeks–5 years) and the median weight was 4.35 kg (range 2.27–6.06) All cats were domestic shorthair cats. Five of the cats were reported to be healthy with no comorbidities. One cat was evaluated for unknown trauma, suspected to be vehicular. Acetaminophen (52 mg/kg) and acetylsalicylic acid (34 mg/kg) had been administered for analgesia by a neighbor who observed the injured cat. It had multiple pelvic fractures but was hemodynamically stable without any life-threatening consequences of trauma or alterations in other organ systems.

In all cats, emesis induction was attempted using oral administration of dexmedetomidine subsequent to known or suspected toxin ingestion. Toxins included lily petals (n = 2), acetaminophen and acetylsalicylic acid (n = 1), and onions (n = 3). The two cats presented for possible lily ingestion were housemates. No ingestion by either cat was observed, but the lily petals from a bouquet on a table had bite marks with pieces missing. Two of the three cats presented for onion ingestion were housemates that were observed eating an unknown amount of cooked onion in pasta sauce with meat. The third cat presented for onion ingestion had possibly ingested a slice of raw onion. None of the cats had vomited spontaneously before the administration of dexmedetomidine. Emesis was successfully induced in 5/6 (83%) cats. In all cats in which emesis was successful, there was only one episode of emesis. In the single cat in which emesis was unsuccessful, hydromorphone (0.1 mg/kg) was given SC after reversal of dexmedetomidine, but this did not cause emesis either.

Time to emesis was recorded in three cats, with times of 12, 20 and 20 mins. Time from ingestion of toxin to presentation was in the range of 0.5–3 h (median 1 h). Five cats received atipamezole hydrochloride (0.2 mg/kg IM) for reversal. The cat that did not receive atipamezole had the comorbidity of pelvic fractures and was not reversed due to recovery from sedation after diagnostic imaging was completed. The time to administration of reversal agent was in the range of 58–71 mins (median 68 mins). Depth of sedation was not recorded for any cat, but subjectively, sedation was moderate to profound. No adverse events were recorded or witnessed.

In one of the two cats suspected to have ingested lily petals, there were pieces of lily petals and cat food in the vomitus. In the vomitus from the other cat that had possibly ingested lily petals, there was only cat food present. No pills were recovered from the cat that was given acetaminophen and acetylsalicylic acid, as only cat food was present in the vomitus. Onions were present in the vomitus of two cats that had eaten onions. Emesis was unsuccessful in the third cat that had ingested onions. None of the cats were administered activated charcoal.

Four of the five cats in which emesis was successful had no reported clinical signs consistent with ingestion of toxins. The exact dose of the toxins, other than the cat given acetaminophen and acetylsalicylic acid, was not

known. The cats that had possibly ingested lily petals were hospitalized and received IV fluids for 72 h after ingestion, and no changes in serum creatinine were noted during that time. Two of the cats that had ingested onions had a recheck packed cell volume (PCV) 48 h after ingestion, and no changes were noted from baseline values recorded at presentation. The cat that was suspected to have ingested onion but did not vomit after orally administered dexmedetomidine and hydromorphone SC was monitored for 48 h. Acanthocytes and echinocytes were noted on a recheck blood smear 24 h after possible ingestion. The cat was treated with *N*-acetylcysteine for 24 h after the red blood cell morphology changes were noted. The PCV was normal 48 h later, with no evidence of anemia. The cat that was administered acetaminophen and acetylsalicylic acid was given these medications approximately 3 h before presentation and was then fed a meal before presenting to the emergency service. The cat vomited once after administration of oral dexmedetomidine but developed a methemoglobin level of 13.7% (reference interval 0–1) 3.5 h after presentation (6 h after administration of acetaminophen), consistent with acetaminophen poisoning. No clinical signs of methemoglobinemia were noted, but due to financial constraints and concurrent pelvic fractures, humane euthanasia was elected approximately 6.5 h after ingestion of acetaminophen and aspirin.

Discussion

The results of the present small case series indicate that oral dexmedetomidine was effective for the induction of emesis in 5/6 cats. Although randomized controlled studies are needed to assess the efficacy of oral administration of dexmedetomidine compared with other previously described emetic agents and routes, this case series suggests that in cats with no known cardiovascular comorbidities, orally administered dexmedetomidine at 20 µg/kg can be an effective and safe method of inducing emesis. This is consistent with prior studies performed to evaluate the sedative, cardiorespiratory and anti-nociceptive effects of oral transmucosal dexmedetomidine in combination with buprenorphine, in which 70% (28/40) and 67% (4/6) of cats vomited. Doses administered in those studies were 20 µg/kg and 40 µg/kg, respectively.[10,11]

The mechanism of action of α_2-agonists as an emetic agent is via stimulation of the chemoreceptor trigger zone of the area postrema.[12] The α-adrenergic stimulation centrally also causes sedation. Peripheral effects of α_2-agonists include bradycardia, hypotension, hypertension and atrioventricular block. Due to these complications, these medications may not be safe for cats with comorbidities of cardiac disease or hypotension, although adverse effects may occur even in healthy cats.[13] No complications were reported or observed in the six cats treated with oral dexmedetomidine in this case series, but heart rate and blood pressure measurements after administration of the medication were not recorded. The authors recognize that bradycardia, hypertension and hypotension are important complications that may develop with the use of α_2-agonists. Monitoring of these vital parameters is encouraged when α_2-agonists are used for emesis induction in cats.

In addition to cardiovascular side effects, emesis that is preceded by sedation could increase the risk of aspiration pneumonia, although aspiration pneumonia happens very uncommonly in cats.[14] While recent studies suggest aspiration pneumonia is a rare complication of emesis induction in dogs, emetic agents used in dogs do not cause sedation as profound as dexmedetomidine does in cats.[15,16] However, the findings of this case series also suggest that orally administered dexmedetomidine can be successfully reversed with IM atipamezole, as all cats recovered uneventfully once reversed. Although profound sedation was observed in this study, the study by Santos et al,[10] investigating 20 µg/kg dexmedetomidine administered orally, reported much less sedation. Future studies should utilize objective sedation scores to elucidate the sedative effects of transmucosally absorbed dexmedetomidine in cats.

While a recent paper evaluating transmucosal detomidine showed very promising results for emesis, with 7/7 cats vomiting after administration, this agent may not be readily available in the majority of veterinary clinics.[9] The more widely available α_2-agonists, xylazine and dexmedetomidine, have been more extensively studied. Early studies reported a 91–100% emesis efficacy with IM xylazine, but this high success rate has failed to be duplicated in subsequent studies.[1,12,13,17] Success rates of other studies utilizing dexmedetomidine and xylazine vary from 0% (0/6 cats) to 43% (9/21 cats) for IM xylazine to 58% (15/26 cats and 7/12 cats) for IM dexmedetomidine.[1,4,13] The wide range of rates of successful emesis induction in cats highlights the need for additional prospective studies to directly compare the route of administration and dose of various α_2-agonists. It is important to emphasize that successful emesis induction does not suggest that all potentially toxic material has been removed from the stomach. Studies in humans and dogs report a recovery rate of 10–77% of material ingested after emesis is induced.[18–20] A study in cats demonstrated that 79% (11/14) of cats that vomited produced foreign material they had ingested, although in four of those cats, less than 75% of the foreign material ingested was recovered.[5] This is further highlighted by a cat in the present case series that developed clinicopathological evidence of acetaminophen toxicity even after emesis occurred. The routine use of emetic agents in cats after ingestion of a potentially toxic substance is not recommended and should be considered on a case-by-case basis based on the substance ingested and time the substance was ingested, as the efficacy of emesis reduces over time. In addition,

emesis should not be induced in patients that are already exhibiting clinical signs; other means of gastrointestinal decontamination, such as gastric lavage, may be considered on a case-by-case basis in those circumstances.

Dosing dexmedetomidine orally may be easier than an IM injection, particularly in cats that are stressed. The medication can easily be administered while the cat's mouth is open and only minimally restrained. In addition, the sedative effects of α_2-agonists may be helpful for decreasing stress and facilitating additional diagnostics, such as bloodwork, or interventions, such as IV catheter placement. It is difficult to say whether dexmedetomidine was swallowed by the cats in this case series or absorbed transmucosally to cause the desired physiologic effect of emesis. The transmucosal absorption of dexmedetomidine has been studied extensively in cats. Slingsby et al[21] also demonstrated effective transmucosal absorption of 40 µg/kg dexmedetomidine in a group of cats, while evaluating its effect on nociception. Out of 12 cats in the transmucosal group, 11 (91.6%) vomited, compared with 9/12 (75%) cats in the IM group. Time to emesis was less than 10 mins.[21] Other studies have evaluated transmucosal absorption of dexmedetomidine, with mixed results in terms of its sedative efficacy; however, vomiting was a consistent adverse effect noted, demonstrating absorption.[10,22] While the authors cannot specifically state whether the route of absorption in this study was oral or transmucosal after oral administration, there was an excellent response in terms of the desired end goal of emesis. Due to the small volume of the drug instilled in each cat's oral cavity (median 0.18 ml, range 0.09–0.24), the authors believe the mechanism of absorption is likely transmucosal.

The dose of 20 µg/kg dexmedetomidine used in this group of cats bears further exploration. This was an arbitrary dose based on previous use by one of the authors in the past. It is possible that a lower dose would be as effective and lead to less profound sedation and other adverse effects (eg, hypertension, hypotension). It is also possible that a higher dose could lead to faster emesis, which may be helpful for rapidly acting toxins, without changing adverse effects. This assumption is based on the prior study by Slingsby et al[21] in which emesis occurred more rapidly with a 40 µg/kg dose of oral dexmedetomidine.

Conclusions

Dexmedetomidine, administered at 20 µg/kg orally, was observed to reliably induce emesis in a small group of cats. Larger studies comparing various routes of administration of dexmedetomidine, as well as determination of the ideal doses, should be considered.

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Ethical approval The work described in this manuscript involved the use of non-experimental (owned or unowned) animals. Established internationally recognized high standards ('best practice') of veterinary clinical care for the individual patient were always followed and/or this work involved the use of cadavers. Ethical approval from a committee was therefore not specifically required for publication in *JFMS*. Although not required, where ethical approval was still obtained, it is stated in the manuscript.

Informed consent Informed consent (verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (experimental or non-experimental animals, including cadavers) for all procedure(s) undertaken (prospective or retrospective studies). For any animals or people individually identifiable within this publication, informed consent (verbal or written) for their use in the publication was obtained from the people involved.

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