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ORIGINAL RESEARCH

Scoping review exploring the evidence base on *Vitis vinifera* toxicity in dogs after ingestion: Clinical effects, treatments and types of *V. vinifera*

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Abstract

Background: Treatment of *Vitis vinifera* fruit (VVF) ingestion can be challenging due to no clear toxic dose, signalment factors and variable clinical signs. Current treatment guidance is generalised: decontamination, aggressive fluid therapy, monitoring and/or treatment of renal dysfunction. The objective of this study was to conduct a scoping review of scientific evidence regarding the ingestion of VVF in dogs. Three primary areas were reviewed: VVF types ingested, clinical signs reported and treatments given. The inclusion criterion was any paper presenting data on clinical signs or treatments of dogs that had ingested VVF (unprocessed VVF only).

Methods: The following databases were searched: CAB Abstracts, Medline, Embase and Scopus. No limits were placed on language or date. The review followed the Joanna Briggs Institute scoping review methodology.

Results: Twenty-four papers were identified. A wide range of VVF types were ingested, but the toxic dose was difficult to ascertain. The most commonly reported signs were gastrointestinal, renal, neurological and haematological. Treatment commonly consisted of fluid therapy, diuretics and antiemetics.

Limitations: This scoping review neither explored cases of processed VVF ingestion nor did it chart laboratory findings; therefore, potentially clinically significant findings in these areas may have been missed.

Conclusions: VVF ingestion typically causes gastrointestinal/renal dysfunction, with no clear toxicity attributable to VVF type. Treatments varied according to the presence/absence of clinical signs, and the prognosis was varied. Further research on current treatment efficacy is warranted, permitting an evidence-based, risk–benefit approach to be adopted by clinicians.

KEYWORDS
acute kidney injury, AKI, canine, dog, grape, grape toxicity, raisin toxicity, *Vitis vinifera*

INTRODUCTION

Vitis vinifera fruit toxicity in dogs

Multiple varieties of *Vitis vinifera* fruit (VVF) have been reported as toxic in dogs. This includes grapes ,[1] dried grapes (currants, raisins)[2,3] and processed grape products such as grape marc.[4] The toxic component of VVF is currently unknown; hypotheses include possible contamination of the grape with heavy metals, pesticides or other toxins (mycotoxins, ochratoxins)[5] or an intrinsic component of the grape itself (e.g., tartaric acid[6]). To date, studies screening for such toxins or contaminants have been unable to find evidence of any heavy metal or mycotoxin contamination.[5,7] Recently, a case series studying the toxic effects of cream of tartar, tamarind and grapes concluded that tartaric acid (an organic acid found

naturally at a relatively high level in grapes) could be a possible underpinning causal factor among the three.[6]

The clinical signs associated with VVF toxicity are varied but can include vomiting, diarrhoea and lethargy.[8] Acute kidney injury (AKI) is of particular interest in cases of VVF toxicity due to the mortality associated with end-organ damage.[9] Histopathology of the kidneys of VVF toxicity patients has shown necrosis of the proximal convoluted tubule epithelial cells, with some showing signs of nuclear regeneration, which is characteristic of tubules when neighbouring cells degenerate and are sloughed.[6,10] Nevertheless, to date, the mechanism of renal proximal tubule epithelial cell death, the cell type in the kidney primarily affected by VVF ingestion, remains unknown, although trapping of organic anions through inadequate apical transport is suspected.[11]

The toxic dose is unknown; thus, guidance for appropriate treatment has usually recommended that the consumption of any quantity of VVF warrants aggressive therapy, including induction of emesis, administration of activated charcoal and intensive fluid therapy for at least 48 hours. Renal parameter monitoring (i.e., creatinine) for at least 72 hours is also recommended.[12]

Rationale

Evidence-based medicine,[13] and later evidence-based veterinary medicine (EBVM),[14] considers an approach to treatment where 'examination of evidence from clinical research' has greater priority over 'intuition, unsystematic clinical experience and pathophysiological rationale' for decision making in clinical practice. EBVM is an iterative process; the best available evidence is used to form a conclusion, which is updated and re-evaluated as new sources of evidence emerge that could influence an outcome (i.e., patient health after treatment). An EBVM decision also considers owner, patient and other veterinary factors (such as access to treatments) in any final decision. Nonetheless, being able to use evidence in clinical decision making is reliant on having a strong evidence base.

Unfortunately, much of the current information regarding VVF toxicity (such as toxic dose or toxic mechanism) is unknown.[12] There are considerable gaps in knowledge in regard to canine VVF toxicity. For a common toxic event (197 cases of VVF ingestion in dogs were reported to the Dutch Poisons Information Centre in 2018[15]), this has implications for clinicians wanting to use an EBVM approach (e.g., What is the best evidence for treatment, are antiemetics or fluids indicated?). A scoping review will provide an overview of key reported features of toxicity and identify knowledge gaps.

A scoping review is a methodological exploration of the evidence base within a topic, drawing together the best available published evidence. Unlike a systematic review, the questions explored within the topic are broader, allowing for a more extensive charting of the evidence base. Critical appraisal of the evidence (an assessment of quality) is uncommonly executed in scoping reviews and therefore is a limitation of these types of reviews. However, scoping reviews can provide excellent starting points for further research and can be cautiously used by clinicians looking to utilise an evidence base to support their decision making.

To date, as far as the authors are aware, no such scoping review has been conducted on VVF toxicity in dogs. The authors have screened relevant databases of veterinary evidence (SYREAF: https://syreaf.org/protocols/; OSF: https://osf.io/registries; VetSRev: https://vetsrev.nottingham.ac.uk/), alongside four literature databases (CAB Abstracts, Medline, Embase and Scopus), for this review.

Objectives

The aim of this scoping review was to explore the evidence regarding VVF ingestion in dogs. Three objectives were specifically proposed with linked clinical outcomes:

- Objective 1: 'To what degree does the evidence show that the types of VVF ingested lead to a clinically significant outcome?'
- Objective 2: 'To what degree does the evidence show common clinical signs of toxicity due to VVF ingestion?'
- Objective 3: 'To what degree does the evidence show that similar treatments are given to patients that have ingested VVF?'

METHODS

Protocol and registration

The conduct guidance from the Joanna Briggs Institute methodology[16] and Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) reporting guidance[17] on scoping reviews was followed. The protocol is available at SYREAF.org: https://syreaf.org/wp-content/uploads/2023/07/Exploring-the-evidence-base-on-Vitis-vinifera-toxicity-in-dogs-after-ingestion-clinical-effects-treatments-and-management-practices-A-scoping-review-protocol.pdf.

Eligibility criteria

This scoping review included all research-based papers, with no restriction on study type, publication date or location. Grey literature (e.g., non-peer reviewed conference abstracts) was included if the literature contained novel data. All non-English papers were included if the abstract was in English. All papers

focused on one or more aspects of VVF toxicity in dogs, including toxicological effects and/or treatment and/or prognosis.

For paper selection, the following definitions were used:

- Research-based papers: any publication presenting novel data (i.e., primary sources) or new analyses of existing data (e.g., systematic reviews). Excludes narrative reviews and opinion pieces.
- Toxicological effects: any clinical effect present in dogs that have ingested VVF.
- VVF: all fruit products from the *V. vinifera* plant and foodstuffs containing whole VVF (e.g., mince pies), excluding processed fruit (e.g., grape extract).
- Dogs: any animal from the species *Canis familiaris*, excluding unowned dogs (e.g., wild or feral animals).

Information sources

Searches were conducted using CAB Abstracts, Medline, Embase and Scopus. Citations were filtered as detailed below, and relevant citations backwards searched for relevant sources.

Search

Preliminary searches were developed by two authors and broadened to suit the research question. To capture the majority of VVF containing foodstuffs, searches included keywords from preliminary relevant papers and a manual search of food encyclopaedias via the UoN Library (https://nusearch.nottingham.ac.uk). Search strategy development was supported by an experienced academic librarian (Supporting Information S1).

Selection of sources of evidence

All the databases were searched on the same day (19 April 2023), the search results were transferred to Endnote Online (EndNote Team, Philadelphia, PA, USA) and duplicates were removed. The results were imported into Rayyan (https://www.rayyan.ai/)[17] for blinded screening and further manual deduplication. Citations were screened for eligibility (Supporting Information S2). The screening occurred in two steps, with two independent blinded reviewers; first the abstract and title (J.D./A.Z.), followed by the full text (J.D./T.H.). The reviewers were then unblinded and disagreements were discussed. When a consensus was not reached, a third reviewer (M.B.) was enlisted.

Data charting process

Microsoft Forms was used to capture data (Table 1), which were subsequently exported to Microsoft Excel. Similar to screening, a pilot exercise was conducted to ensure consistency across reviewers and ensure that no refinements were needed regarding the form. Two pairs of reviewers independently extracted data from the papers (J.D./D.G. and J.D./M.B.). Following charting, any disagreements were discussed, and when a consensus could not be reached, the topic was extended to the wider research group.

Non-English language papers were translated using DeepL (https://www.deepl.com/translator), and data charting was checked by individuals fluent in the relevant language.

TABLE 1 List of data items present on a Microsoft Form used to extract information regarding *Vitis vinifera* fruit (VVF) toxicity in dogs

Title of study
Author
Date of publishing
Study descriptors
Location of study
Source of data for the study
Type of study
Aims of study
Patient descriptors
Number of patients
VVF products ingested
Type of fruit consumed (fresh, dried, foodstuffs)
Quantity of VVF consumed
The clinical signs of intoxication shown
Definition of acute kidney injury
Number of patients with clinical signs
List of clinical signs categorised into body systems
Treatment
Treatments given
Duration of treatments
Study outcomes
Outcome of patients

Analysis of the charted data

Source of data for the study

The source of the information was categorised into three broad groups: practice data, poisons information service data and other. Practice data included information from clinics, including multicentre data, first opinion and referral. Poisons information services were considered external services that provide advice regarding cases of intoxication.

Type of study

The type of study was categorised into three groups: descriptive, observational and experimental. The definitions used were as follows.

- Descriptive studies include case series/case studies and were defined as 'a study concerned with, and designed only to, describe the existing distribution of variables without much regard to causal relationships or other hypotheses'.[18]
- Observational studies included case-control and cohort (both descriptive and population-based)[19] studies and could be defined as a study that contains the methodological rigor that allows for the testing of causal relationships and hypotheses.[18]
- Experimental studies were defined as studies where the investigator 'intentionally altered factors within the study'; therefore, conditions in the study were under the direct control of the investigator.[18]

Number of patients with clinical signs

Patients were recorded as asymptomatic or symptomatic after VVF ingestion. Patients recorded as developing kidney damage, acute renal failure or AKI were synthesised into a singular group using AKI as the preferred nomenclature.[20] Papers either used the international renal interest society (IRIS) guidelines or contained enough information to extrapolate the IRIS grade of AKI; these papers were recorded as IRIS and IRIS (extrapolated), respectively. Papers that contained a definition of AKI but not enough information to determine the IRIS grade were recorded as other; papers that did not give a definition of AKI were recorded as none.

List of clinical signs categorised into body systems

Clinical signs were recorded unless specifically stated as being due to an external factor (e.g., fluid overload). Laboratory findings (e.g., azotaemia) were not included as not all cases had laboratory analysis data. Papers including each clinical sign were tallied, with clinical signs categorised using the classification system of Robinson as modified by Nielsen et al.[21,22] Similar clinical signs were merged into larger umbrella terms (e.g., inappetence would come under the umbrella term of anorexia). A full list of merged related terms can be found in Supporting Information S3.

Treatments given

Treatments were recorded unless use was specifically for a clinical sign unrelated to VVF toxicity (e.g., trazadone for anxiety). Treatments were categorised according to indicated use. Treatments that did not fit a category or where indicated use was unable to be determined were categorised under miscellaneous. The number of papers reporting the use of each treatment was tallied.

Outcome of patients

For papers that stated outcome, the number of patients that survived until discharge, or died before discharge, was recorded. Of the subset that died, the number that were euthanased was recorded if the data were present.

Critical appraisal

A critical appraisal process was not undertaken for this scoping review.

RESULTS

Selection of sources of evidence

From the four databases searched, 977 records were identified using the specified search terms (Supporting Information S1), which were reduced to 559 after deduplication. Title and abstract screening excluded 515 records. During full-text screening, 19 papers were excluded, with 25 studies meeting the inclusion criteria. Of these 25 studies, one was subsequently excluded as the full text was unobtainable.[23] Full details of the screening results can be found in the PRISMA flow diagram (Figure 1). Backward citation searching was performed on the 24 included studies, which identified 356 additional papers, all of which were excluded after deduplication and screening.

Characteristics of sources of evidence

The publication dates ranged from 2001 to 2022. The majority were descriptive studies (16/24; 66.7%), with seven observational studies (29.2%) and one experimental study (4.2%; Figure 2). The median publication date of all descriptive studies was 2009 (interquartile range [IQR]: 10 years), and the median date of all observational studies was 2020 (IQR: 8.5 years).

Eleven studies were conducted in Europe (UK: 6, Germany: 2, Czech Republic: 1, Netherlands: 1 and Switzerland: 1), seven studies were conducted in North America (USA: 6 and Canada: 1) and five studies were conducted in Asia (South Korea: 3, India: 1 and Japan: 1). One study location was unable to be determined.

Among the 24 relevant studies, 17 (70.8%) papers were from practice data, six (25.0%) were from poisons information services data and one (4.2%) was categorised as other (experimental data). Of the seven observational studies, three (42.9%) were from practice data and four (57.1%) were from poisons information services data. The poisons information services were available worldwide and consisted of the AnTox database from the U.S. Animal Poisons Control Centre, the UK's Veterinary Poisons Information Service and the Dutch Poisons Information Centre.

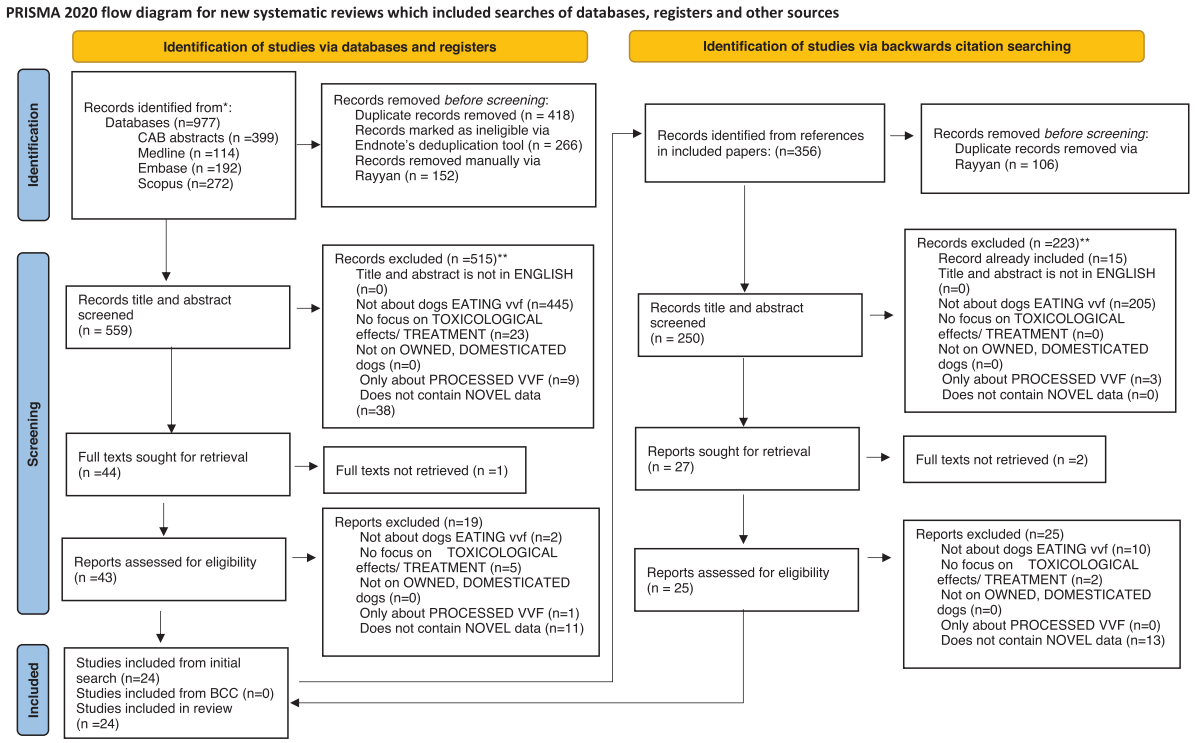

FIGURE 1 PRISMA flowchart showing the screening process for relevant papers in this scoping review of *Vitis vinifera* fruit (VVF) toxicity in dogs

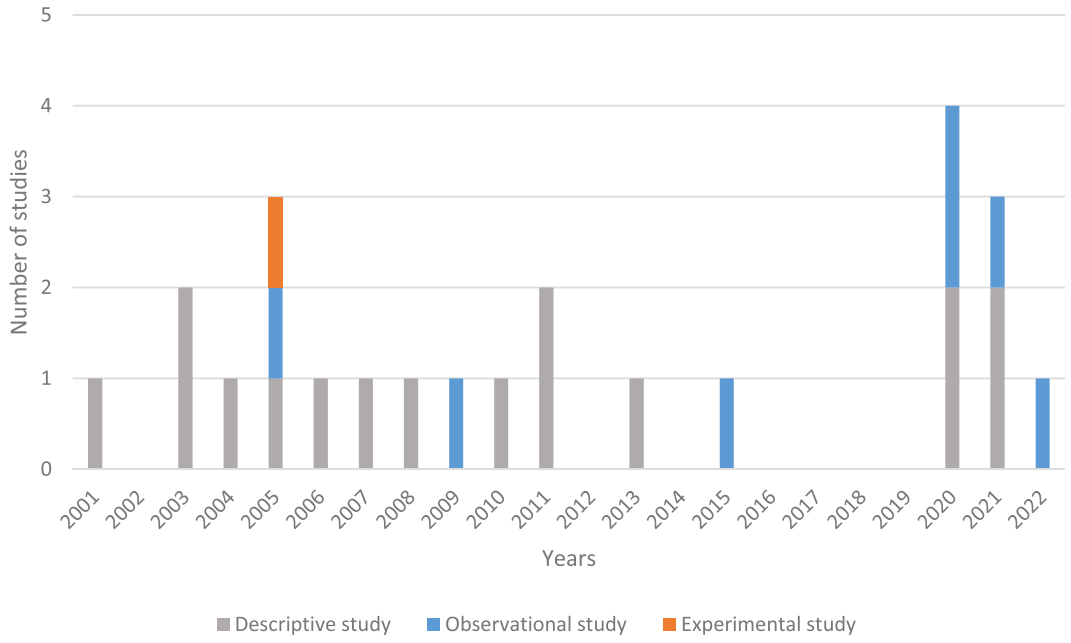

FIGURE 2 Number of published studies, by year and type of study design, found during a scoping review of *Vitis vinifera* fruit toxicity in dogs

Ten of the 24 studies (41.7%) stated a clear aim. Of these, two were descriptive studies, one was experimental and seven were observational (Table 2). Six observational studies stipulated clinical signs, clinical course, clinical features or presenting signs within the aims of their studies.[4,7,8,15,24,25] Three aimed to calculate the prevalence or incidence of AKI.[8,15,25] No observational studies included the type of VVF as a comparative parameter.

Synthesis of results

Types of VVF ingested

Twenty-two studies specified the type of VVF ingested in each patient, which was unclear in the remaining two papers. Among the 17 studies that specified the consumption of grapes, only five (29.4%) specified the colour of the grapes. Among the 15 studies that

TABLE 2 Aims of studies found during a scoping review of literature focused on *Vitis vinifera* fruit (VVF) ingestion in dogs

Author	Study type	Aim of study
Eubig et al.[7]	Observational	To summarise signalment, clinical signs, laboratory results and response to treatment in 43 dogs that developed azotemia after ingestion of grapes or raisins
Yuk et al.[26]	Experimental	To determine the dose of grape poisoning in dogs and to use it for future diagnosis and treatment of acute renal failure
Sutton et al.[24]	Observational	To describe factors that influenced the clinical course and outcome of 169 cases of *Vitis* intoxication in dogs reported to the VPIS, London
Pak[27]	Descriptive	To characterise the diseases and clinicopathological findings associated with acute renal failure caused by ingestion of grapes or raisins in dogs that were presented to Kangwon National University Veterinary Teaching Hospital
Bates and Edwards[28]	Observational	To establish the common causes of death in cats and dogs with suspected poisoning reported to the VPIS in the UK
Arnold et al.[29]	Descriptive	To describe a case of suspected hepatotoxicity in a dog secondary to administration of trazodone
Reich et al.[25]	Observational	To describe the prevalence of AKI, clinical course, gastrointestinal decontamination procedures and outcome in dogs following grape or raisin ingestion
Schweighauser et al.[4]	Observational	First, to evaluate the clinical, laboratory, pathological and outcome features of dogs diagnosed with grape or raisin toxicosis compared to dogs diagnosed with AKI of other origin, with emphasis on renal and neurological manifestations; and second, to investigate potential risk factors for development of neurological signs by comparing dogs with and without central nervous system deficits
Croft et al.[8]	Observational	To assess key presenting signs in dogs following VVF ingestion (grapes, raisins, currants and sultanas), outcome and the incidence of AKI
Dijkman et al.[15]	Observational	To determine the incidence of clinical signs and *Vitis* fruit-induced AKI in dogs and cats with a *Vitis* fruit ingestion reported to the Dutch Poisons Information Center, and a description of the therapies instituted by the veterinarians

Abbreviations: AKI, acute kidney injury; VPIS, Veterinary Poisons Information Service.

specified the consumption of dried VVF, 13 (86.7%) specified the type of dried fruit (e.g., raisin, currant or sultana). Among the three studies that specified the consumption of VVF in foodstuffs, only one study specified the type for each individual patient (Figure 3).

A total of 2073 patients were recorded as consuming VVF across the studies (two patients not included in this total consumed only grape marc[4]). The type of VVF was recorded for 1119 (54.0%) of these patients; 410 (36.6%) patients consumed grapes, of which 12 (2.9%) ingested red grapes and six (1.5%) ingested white grapes, with the remainder consuming an unspecified colour of grape. Four hundred and fifty (40.2%) patients consumed dried VVF, of which 176 (39.1%) consumed raisins, 21 (4.7%) consumed sultanas and two (0.4%) consumed currants, with the remainder consuming an unspecified type of dried VVF. Eight patients consumed both grapes and dried VVF. Two hundred and sixty-seven (23.9%) consumed VVF in foodstuffs.

Clinical signs

The distinction between asymptomatic and symptomatic patients was made in 23 studies (95.8%), with a total of 1163 patients. Eight hundred and sixty (73.9%) patients remained asymptomatic, whereas 303 (26.1%) patients developed clincal signs. Removing the studies that had AKI in their inclusion criteria (n = 2), the percentage of symptomatic patients reduced to 24.4%. Five hundred and fifty-four patients had the required data to diagnose AKI, of these 109 (19.7%) patients developed AKI. Again, removing the studies that had AKI in their inclusion criteria (n = 2), the percentage of patients developing AKI reduced to 15.7% (Table 3).

Thirteen (54.2%) studies either used the IRIS definition for AKI or contained enough data to extrapolate the IRIS grading for the cases of AKI. These cases are reported in Table 4.

Most papers (21/24; 87.5%) recorded at least one clinical sign present in patients that had ingested VVF. There was a wide range of clinical signs attributed to VVF toxicity; those most often reported were vomiting (19/21; 90.5%), lethargy (18/21; 85.7%) and diarrhoea (14/21; 66.7%). While some neurological signs were reported in multiple papers (tremor, ataxia, seizures and obtundation),[1,3,7,9,15,25,30] the majority were only reported in one paper, which specifically looked at the neurological signs of VVF toxicity[4] (Table 5).

Treatments

Most studies (19/24; 79.2%) described at least one treatment administered to patients with VVF toxicity. The most common groups of treatments reported to be given were fluid therapy (19/19; 100.0%), diuretics (12/19; 63.2%) and antiemetics (11/19; 57.9%) (Table 6).

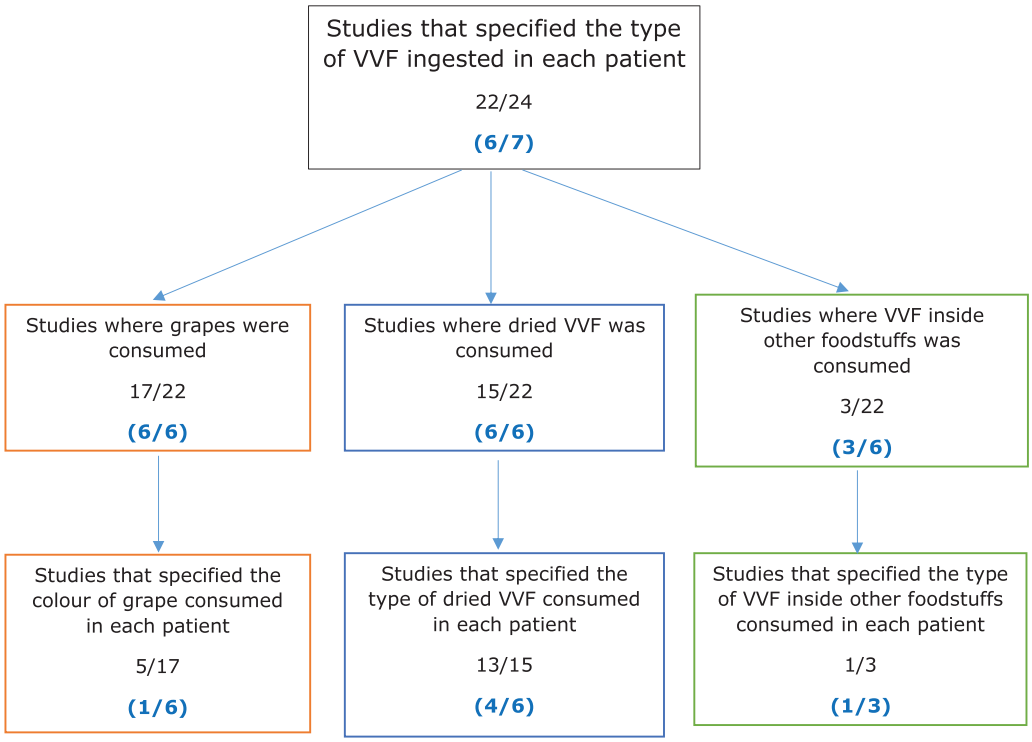

FIGURE 3 Types of *Vitis vinifera* fruit (VVF) ingested by dogs in 24 studies found during a scoping review of VVF toxicity in dogs. Numbers in dark blue are observational studies, and numbers in black are descriptive studies. Orange box surround—fresh VVF ingestion; blue box surround—dried VVF ingestion (raisins/sultanas/currants); green box surround—VVF inside other foodstuffs ingestion (e.g., fruit loaf, mince pies)

Outcome

Across the 24 papers, 94.2% (1890/2006) of the patients survived. Of the patients that died, and where the cause of death was recorded, 63.6% (68/107) were euthanased. The cause of death was unable to be ascertained in 9/116 (7.8%) of the patients that died. Papers that provided extra details regarding the outcome of the patients have been added to the footnotes of Table 7.

DISCUSSION

Summary of evidence

General study descriptions

This scoping review found 24 relevant papers, with the majority being descriptive studies. More recently, the ratio of observational to descriptive studies has increased, and the median publication date for descriptive studies is 2009 compared to 2020 for observational studies. This may reflect how relatively recent the discovery of VVF toxicity is, but also identifies how the understanding of the toxicity has changed. Case studies and case series appear first as examples of a novel disease not yet described, with the earliest paper found being a short descriptive study from 2001.[5] This is generally then followed by observational studies attempting to identify risk factors for the disease. Due to the inherent biases in descriptive studies caused by small sample size and non-random sample selection, the certainty of the conclusions drawn from these studies is weak and can be difficult to extrapolate. However, with the number of observational studies increasing, it is expected that the amount and strength of the evidence will increase.

Types of VVF ingested that led to clinical signs

One potentially overlooked risk factor is the type of VVF consumed. None of the observational studies included the type of VVF as a variable. While some observational studies did index the types of VVF ingested by patients in the initial results, the different types were combined during the statistical analysis. This loss of detail makes it difficult to draw conclusions regarding any significant difference between types of VVF. This complements work performed previously,[39] which found inadequate evidence to compare the toxicity of fresh to dried grapes. One paper[24] suggested that dogs appeared more likely to be ill after ingestion of dried, compared to fresh fruit. Nevertheless, a significant knowledge gap remains in this regard.

Clinical signs of toxicity due to ingestion of VVF

The most frequently recorded clinical signs were vomiting, diarrhoea and lethargy. This aligns with existing observational studies, which ranked the

TABLE 3 Breakdown of patients found in published papers identified during a scoping review of *Vitis vinifera* fruit toxicity in dogs

Year published	Author	No. of patients asymptomatic (%)	No. of patients with clinical signs (%)	No. of patients diagnosed with AKI (%)
2001	Gwaltney-Brant et al[5]	0/10 (0.0)	10/10 (100.0)	Not stated
2003	Penny et al[2]	0/1 (0.0)	1/1 (100.0)	1/1 (100.0)
2003	Campbell and Bates[31]	1/4 (25.0)	3/4 (75.0)	Not stated
2004	Mazzaferro et al[9]	0/4 (0.0)	4/4 (100.0)	4/4 (100.0)
2005	Koch et al[32]	0/1 (0.0)	1/1 (100.0)	1/1 (100.0)
2005	Yuk et al[26]	3/5 (60.0)	2/5 (40.0)	1/5 (20.0)
2005	Eubig et al[7]	33/90 (36.7)[a]	57/90 (63.3)[a]	43/90 (47.8)[a]
2006	Elwood and Whatling[33]	1/1 (100.0)	0/1 (0.0)	0/1 (0.0)
2007	Usselmann[34]	2/3 (66.7)	1/3 (33.3)	1/3 (33.3)
2008	Stanley and Langston[3]	0/1 (0.0)	1/1 (100.0)	1/1 (100.0)
2009	Sutton et al[24]	101/169 (59.8)	68/169 (40.2)	17/169 (10.1)
2010	Itoh at al[30]	0/1 (0.0)	1/1 (100.0)	1/1 (100.0)
2011	Yoon et al[1]	0/2 (0.0)	2/2 (100.0)	Not stated
2011	Kralova-Kovarikova et al[35]	0/1 (0.0)	1/1 (0.0)	1/1 (0.0)
2013	Pak[27]	0/11 (0.0)[b]	11/11 (0.0)[b]	11/11 (0.0)[b]
2015	Bates and Edwards[28]	Not stated	Not stated	Not stated
2020	Arnold et al[29]	1/1 (100.0)	0/1 (0.0)	0/1 (0.0)
2020	Reich et al[25]	105/139 (75.5)[c]	34/139 (24.5)[c]	8/120 (6.7)
2020	Schweighauser et al[4]	0/15 (0.0)[b]	15/15 (100.0)[b]	15/15 (100.0)[b]
2020	Jayaraj et al[36]	0/1 (0.0)	1/1 (100.0)	Not stated
2021	Croft et al[8]	532/606 (87.8)	74/606 (12.2)	1/33 (3.0)
2021	Mitropoulou et al[37]	0/1 (0.0)	1/1 (100.0)	1/1 (100.0)
2021	Takada and Loewen[38]	0/1 (0.0)	1/1 (100.0)	1/1 (100.0)
2022	Dijkman et al[15]	81/95 (85.3)	14/95 (14.7)	1/95 (1.1)
Total		860/1163 (73.9)	303/1163 (26.1)	109/554 (19.7)
Total (excluding AKI only studies)		860/1137 (75.6)	277/1137 (24.4)	83/528 (15.7)

Note: The rows shaded blue are observational studies, and the rows shaded orange are experimental studies.
Abbreviation: AKI, acute kidney injury.
[a]Patient breakdown extrapolated from exclusion breakdown.
[b]AKI was an inclusion criterion for the study.
[c]Breakdown based on whether clinical signs were shown before presentation.

most common clinical signs, and with articles on VVF toxicity, where vomiting, diarrhoea and lethargy were described as possible diagnostic indicators.[40–42] This scoping review highlights that other commonly reported clinical signs were neurological and/or haematological.

The majority of neurological signs reported were from a single paper describing 15 dogs, with a specific focus on the neurological aspects of VVF toxicity.[4] While the mechanism of neurotoxicity is unclear, it was found that 'neurological manifestations may even dominate the early clinical picture and confuse the initial diagnostic evaluation'. It was reported in this paper that the neurological signs were reversible in the patients that survived and were unlikely to be caused by uraemia.

Multiple studies have reported clinical signs, such as haematochezia, melaena or petechiation, that could suggest abnormal primary haemostasis.[9,32,35] An experimental study in dogs showed an inhibitory effect of grape juice on platelet aggregation[43]; thus, toxin-mediated thrombocytopathia may contribute to these clinical signs. However, AKI itself may be associated with hypocoagulability, based on two small prospective studies,[44,45] further complicating the causality of haematological clinical signs. Additionally, non-haematological complications of AKI, such as uraemic gastropathy, could cause melaena. Prolonged clotting times were also reported in one study,[9] suggesting abnormal secondary haemostasis. The cause of this is unknown, and the severe haemorrhage that would usually be associated with secondary haemostatic disorders was not reported. Further research (e.g., viscoelastic testing) should be undertaken before determining an association or causal link between VVF and the haematological abnormalities seen in some patients.

The incidence of AKI differs greatly between studies, with the overall incidence (removing papers where AKI was an inclusion criterion) being 15.7%. It is worth noting that many of the newer observational studies[8,15,25] had much lower incidences of

TABLE 4 Breakdown of acute kidney injury (AKI) severity found in published papers identified during a scoping review of *Vitis vinifera* fruit toxicity in dogs

Year published	Author	AKI definition	No. of patients diagnosed with AKI (%)	IRIS grade I (%)	IRIS grade II (%)	IRIS grade III (%)	IRIS grade IV (%)	IRIS grade V (%)
2003	Penny et al[2]	IRIS (extrapolated)	1/1 (100.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	1/1 (100.0)
2004	Mazzaferro et al[9]	IRIS (extrapolated)	4/4 (100.0)	0/4 (0.0)	0/4 (0.0)	0/4 (0.0)	1/4 (25.0)	3/4 (75.0)
2005	Koch et al[32]	IRIS (extrapolated)	1/1 (100.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	1/1 (100.0)
2005	Yuk et al[26]	IRIS (extrapolated)	1/5 (20.0)	0/5 (0.0)	0/5 (0.0)	0/5 (0.0)	1/5 (20.0)	0/5 (0.0)
2005	Eubig et al[7]	Other	43/90 (47.8)[a]	–	–	–	–	–
2006	Elwood and Whatling[33]	None	0/1 (0.0)	–	–	–	–	–
2007	Usselmann[34]	IRIS (extrapolated)	1/3 (33.3)	0/3 (0.0)	0/3 (0.0)	0/3 (0.0)	1/3 (33.3)	0/3 (0.0)
2008	Stanley and Langston[3]	IRIS (extrapolated)	1/1 (100.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	1/1 (100.0)
2009	Sutton et al[24]	None	17/169 (10.1)	–	–	–	–	–
2010	Itoh et al[30]	IRIS (extrapolated)	1/1 (100.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	1/1 (100.0)	0/1 (0.0)
2011	Kralova-Kovarikova et al[35]	IRIS (extrapolated)	1/1 (0.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	1/1 (0.0)
2013	Pak[27]	Other	11/11 (0.0)[b]	–	–	–	–	–
2020	Arnold et al[29]	None	0/1 (0.0)	–	–	–	–	–
2020	Reich et al[25]	IRIS	8/120 (6.7)	4/120 (3.3)	0/120 (0.0)	1/120 (0.8)	2/120 (1.7)	1/120 (0.8)
2020	Schweighauser et al[4]	IRIS	15/15 (100.0)[b]	0/15 (0.0)	0/15 (0.0)	0/15 (0.0)	5/15 (33.3)	10/15 (66.6)
2021	Croft et al[8]	IRIS	1/33 (3.0)	1/33 (100.0)	0/33 (0.0)	0/33 (0.0)	0/33 (0.0)	0/33 (0.0)
2021	Mitropoulou et al[37]	IRIS	1/1 (100.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	1/1 (100.0)
2021	Takada and Loewen[38]	IRIS (extrapolated)	1/1 (100.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	0/1 (0.0)	1/1 (100.0)
2022	Dijkman et al[15]	IRIS (extrapolated)	1/95 (1.1)	1/95 (1.1)	0/95 (0.0)	0/95 (0.0)	0/95 (0.0)	0/95 (0.0)
Total			109/554 (19.7)	6/282 (2.1)	0/282 (0.0)	1/282 (0.4)	11/282 (3.9)	20/282 (7.1)

Note: The rows shaded blue are observational studies, and the rows shaded orange are experimental studies.
Abbreviation: AKI, acute kidney injury; IRIS, international renal interest society.
[a]Patient breakdown extrapolated from exclusion breakdown.
[b]AKI was an inclusion criterion for the study.

TABLE 5 Number of papers reporting each clinical sign found during a scoping review of *Vitis vinifera* fruit toxicity in dogs

Gastrointestinal	20	Renal	18	Neurological	8	Haematological	5	Non-specific		Other clinical sign categories[a]	
Vomiting	19	Oliguria	10	Tremor	6	Haematochezia	3	Lethargy	18	Cardiovascular	4
Diarrhoea	14	Anuria	8	Ataxia	5	Melaena	1	Anorexia	9	Respiratory	1
Hypersalivation (ptyalism)	5	Polyuria	4	Seizures	3	Petechia	2	Polydipsia	7	Ophthalmological	1
Other	2	Decreased urine output[b]	2	Obtundation	2	Other	2	Abdominal pain	5	Musculoskeletal	1
		Dysuria	2	Other	11			Dehydration	6		
		Pollakiuria	1					Hypothermia	3		
								Oedema	2		
								Ascites	2		
								Other	5		

[a]Further breakdown of clinical sign categories can be found in Supporting Information S4.
[b]Decreased urine output has been defined as an unmeasured decrease in urine output and so encompasses both oliguria and anuria.

TABLE 6 Number of papers reporting each type of treatment administered to dogs with *Vitis vinifera* fruit ingestion identified during a scoping review

Fluids	19	Diuretics	12	Antiemetics	11	Gastric protectors	9	Miscellaneous	11	Other treatment categories[a]	
Intravenous fluid therapy	18	Furosemide	12	Metoclopramide	6	Ranitidine	5	Continuous renal replacement therapy	7	Decontamination	8
Subcutaneous fluids	4	Mannitol	8	Maropitant	4	Omeprazole	3	Peritoneal dialysis	6	Blood pressure modifiers	8
Oral electrolyte therapy	1			Other	5	Cimetidine	2	Blood transfusions (whole and fresh frozen plasma)	2	Antibiotics	4
						Famotidine	2	Other	3	Antithrombotics	3
						Sucralfate	2			Phosphate binders	2
						Unspecified	1			Corticosteroids	2
										Analgesics	2
										Gastrointestinal motility modifiers	2
										Erythropoietin analogues	1
										Antihypocalcaemia	1
										Neurological drugs	1

[a]Further breakdown of treatment categories can be found in Supporting Information S4.

AKI (6.7%–1.1%) than older observational studies[7,24] (10.1%–47.8%). It is difficult to draw conclusions due to differences between AKI definitions across studies; however, the lower incidence in newer studies could reflect either improving preventative treatment strategies or a more accurate prevalence based on better case recruitment and representative sampling.

Description of treatments given to patients that have ingested VVF

As stated by one paper,[15] there is a research gap in the efficacy of treatments recommended, particularly regarding prevention strategies for AKI in cases of VVF ingestion. The preventative treatment regime of aggressive fluid therapy and activated charcoal carries risk. Activated charcoal is contraindicated in multiple scenarios[46] due to the risk of death, long-term pulmonary disease, charcoal peritoneum and corneal abrasion. Potential overtreatment incurs unnecessary costs to owners, as well as the risk of iatrogenic complications. Activated charcoal functions by binding to potential toxins, preventing the toxins from being absorbed by the gastrointestinal system; however, this only works for specific compounds.[47] Due to the unknown toxin within VVF, it is unclear if activated charcoal works as a decontamination agent in cases of VVF toxicity.

TABLE 7 Patient outcomes in studies identified from a scoping review on *Vitis vinifera* fruit toxicity in dogs

Year published	Author	No. survived (%)	No. died (%)	No. of dead that were euthanased (%)
2001	Gwaltney-Brant et al[5]	5/10 (50.0)	5/10 (50.0)	3/5 (60.0)
2003	Penny et al[2]	0/1 (0.0)	1/1 (100.0)	1/1 (100.0)
2003	Campbell and Bates[31]	1/4 (25.0)	3/4 (75.0)	2/3 (66.6)
2004	Mazzaferro et al[9]	2/4 (50.0)	2/4 (50.0)[a]	1/2 (50.0)
2005	Koch et al[32]	0/1 (0.0)	1/1 (100.0)	1/1 (100.0)
2005	Yuk et al[26]	Does not state	Does not state	Does not state
2005	Eubig et al[7]	23/43 (53.5)	20/43 (46.5)	15/20 (75.0)
2006	Elwood and Whatling[33]	1/1 (100.0)	0/1 (0.0)	N/A
2007	Usselmann[34]	2/3 (66.7)	1/3 (33.3)	1/1 (100.0)
2008	Stanley and Langston[3]	1/1 (100.0)	0/1 (0.0)	N/A
2009	Sutton et al[24]	151/168 (89.9)	17/168 (10.1)	4/17 (23.5)[b]
2010	Itoh [30]	0/1 (0.0)	1/1 (100.0)	1/1 (100.0)
2011	Yoon et al[1]	0/2 (0.0)	2/2 (100.0)	Does not state
2011	Kralova-Kovarikova et al[35]	0/1 (0.0)	1/1 (100.0)[c]	1/1 (100.0)
2013	Pak[27]	4/11 (36.4)	7/11 (63.6)	4/7 (57.1)[d]
2015	Bates and Edwards[28]	849/896 (94.8)	47/896 (5.2)	36/47 (76.6)
2020	Arnold e[29]	1/1 (100.0)	0/1 (0.0)	N/A
2020	Reich et al[25]	138/139 (99.3)	1/139 (0.7)[e]	0/1 (0.0)
2020	Schweighauser et al[4]	8/15 (53.3)	7/15 (46.7)	Does not state
2020	Jayaraj et al[36]	1/1 (100.0)	0/1 (0.0)	N/A
2021	Croft et al[8]	606/606 (100.0)	0/606 (0.0)	N/A
2021	Mitropoulou et al[37]	1/1 (100.0)	0/1 (0.0)	N/A
2021	Takada and Loewen[38]	1/1 (100.0)	0/1 (0.0)	N/A
2022	Dijkman et al[15]	95/95 (100.0)	0/95 (0.0)	N/A
Total		1890/2006 (94.2)	116/2006 (5.8)	68/107 (63.6)

Note: The rows shaded blue are observational studies, and the rows shaded orange are experimental studies.
[a]One patient sustained cardiopulmonary arrest.
[b]Of the four patients that were euthanased, two were for reasons other than grape toxicity.
[c]The development of disseminated intravascular coagulation was considered the reason for the fatality.
[d]Of the four patients that were euthanased, two were due to poor response to treatments while two were due to economic reasons.
[e]Patient died due to complications with continuous renal replacement therapy.

The potentially prolonged emptying time of VVF in the stomach of the dog, as shown by the presence of VVF in the vomitus up to 12 hours post-ingestion,[8,15] provides key information that contrasts with some commonly available recommendations regarding the time period for inducing emesis.[48] This finding contributes to the risk–benefit analysis that clinicians need to make when considering the treatment plan for patients with VVF ingestion, although caution should be taken as the evidence is anecdotal and further research is needed.

The most common treatment categories recorded were fluids, diuretics, antiemetics and gastric protectors. The majority of papers were case studies describing more severe cases of VVF toxicity, and so describe the supportive care provided once clinical signs are present. Antiemetics and gastric protectors were administered for animals presenting with prolonged vomiting, diuretics for oliguric/anuric patients, and fluid therapy for preventative strategies and deficit restoration in dehydrated patients. Similar to preventative treatments, the efficacy of these treatments has not been thoroughly studied, so further research would be useful in this area, particularly for diuretics, the efficacy of which is already debated.[49] This review found several reports of patients beginning to show signs of fluid overload, such as tissue oedema, that could be due to aggressive fluid therapy in patients with decreased renal function and AKI.[3,32,34,38]

Limitations

While efforts have been made to include as much relevant terminology in the search terms for this scoping review as possible, it is acknowledged that the foodstuff search terms used may not necessarily be exhaustive and could have an Anglocentric skew.

Due to resource limitations, this scoping review neither investigates the ingestion of processed VVF nor does it chart the clinical findings diagnosed from laboratory analysis. This means that some potentially clinically significant information may have been missed.

While the IRIS grading of AKI has been attempted to be determined for all patients, serial biochemistry measurements were not present within all papers, indicating that grade I cases may have been missed.

Clinical signs and treatments were unable to be charted per patient and were instead charted per paper, leading to a higher proportion of the rarer and more extreme clinical signs and subsequent treatments due to the large percentage of case studies within this scoping review.

Since no critical appraisal of the papers found was performed, any clinical conclusions should be interpreted with caution.

CONCLUSION

This scoping review identified several clinically important gaps in the literature reporting VVF toxicity cases. VVF in the vomitus of dogs past the expected timeframe of gastric emptying presents an important avenue for research that could allow for a more successful decontamination protocol, with further research into the efficacy of treatments warranted. The reporting of neurological and haematological clinical signs possibly indicates overlooked secondary features of VVF toxicity, adding VVF toxicity as a potential differential in patients displaying unexplained neurological or haematological clinical signs. Some of the gaps identified could potentially be filled by the recording of a small amount of additional case information (e.g., VVF types and volumes ingested and outcomes) in studies focused on VVF toxicity.

AUTHOR CONTRIBUTIONS

Study design by Joshua Downs, Agnieszka Zoltowska, Thomas Hackney, David Gardner and Marnie Brennan. Search strategy created by Joshua Downs, Alison Ashmore and Marnie Brennan. Selection of sources of evidence by Joshua Downs, Agnieszka Zoltowska, Thomas Hackney and Marnie Brennan. Data charting by Joshua Downs, David Gardner and Marnie Brennan. The lead on data analysis and writing of the draft manuscript was taken by Joshua Downs. All authors contributed to the writing of the final manuscript.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

All data charted from the papers are freely available and can be found in Supporting Information S4.

ETHICS STATEMENT

The protocol was ethically approved by the Committee for Animal Research and Ethics at the University of Nottingham School of Veterinary Medicine and Science, University of Nottingham (REC: 3975 231017).

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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