Acta Scientiae Veterinariae, 2023. 51(Suppl 1): 906.

CASE REPORT
Pub. 906

ISSN 1679-9216

# Calcium Carbide Poisoning in a Dog - Ultrasound Findings

**Denise Jaques Ramos[1], Diana Salazar[2], Camila Lie Yamauchi[3], Tamiris Disselli[2], Kairo Adriano Ribeiro de Carvalho[4], Giovanna Serpa Maciel Feliciano[2], Stéfany Tagliatela Tinto[2], Ricardo Pozzobon[3] & Marcus Antônio Rossi Feliciano[2]**

## ABSTRACT

***Background*:** Calcium carbide has been used not only in industry, but it is also present in the domestic environment. This substance goes through chemical reactions that lead to the release of acetylene gas and heat being capable of causing burns, damage to mucous membranes, eyes and skin, central nervous system depression and also described as an anesthetic agent. It is lethal in concentrations of 35 % for humans and 80 % for dogs. Although reports that involve this substance in animals as scarce, emergency calls related to intoxication are part of the small animal clinical routine. This report aims to describe laboratory and ultrasonography findings in a case of carbide poisoning in a dog.

***Case*:** A 8-year-old American Staffordshire Terrier with a history of emesis, bloody diarrhea, hiporexy, oligodipsy and ingestion of calcium carbide for 3 days was admitted for medical care at the University Veterinary Hospital of Universidade Federal de Santa Maria (UFSM). The patient had a previous diagnosis of hypoadrenocorticism which was treated with sodium liothyronine 800 mg prescribed by another professional and whose dose was reduced to 600 mg by the owner without veterinary indication. Physical examination showed a high body score, evident lameness of the hind limbs, hyperemic mucosa with elevated time of capillary perfusion and an increase of mandibular and popliteal lymph nodes. Dehydration, fever, elevated heart and respiratory rates and skin lesions of the front limbs were also noted. The main possible differentiate diagnosis based on these clinical signs and history presented by the patient were poisoning calcium carbide intoxication, diabetes mellitus and hypoadrenocorticism. The dog was hospitalized to receive initial treatment and undergo further examination The first treatment instituted was dipyrone [Dipyrone Ibasa 50%® - 15 mg/kg, i.v, TID, for 3 days], tramadol hydrochloride [3 mL/kg, i.v, TID, for 2 days], sucralfate [Sucrafilm® - 5 mg/mL, v.o, TID, for 2 days], maropitant citrate [Cerenia® - 0.1 mg/kg, sc, SID, for 3 days], omeprazole [1 mg/kg, i.v, SID, for 3 days], levothyroxine [300 mg, ½ (half) tablet, v.o, BID, for 2 days]. Blood results have shown leukocytosis (19,600; reference 5,700-14,200) e lymphopenia (196/µL; reference 900/µL-4,700/µL) and in the biochemical tests, there was an increase in triglycerides (175 mg/L; reference 23 mg/L-102 mg/L), without signs of renal or hepatic lesions. In the ultrasonography exam, hepatomegaly and splenomegaly with a heterogeneous parenchyma were noticed. Alteration of the gastrointestinal tract with a thickened wall was also noticed associated with gas in the digestive tract and hyperechoic area in mucous layer. These findings are compatible with reports of mucosal irritation associated with this substance. The pancreas parenchyma was slightly heterogeneous and hyperechoic with enlargement of the body and small disperse hyperechoic focuses. Left adrenal images were compatible with clinical suspicions of hyperadrenocorticism.

***Discussion*:** All the alterations found in the imaging exam were compatible with the clinical signs due to direct contact with the substance in question. Dystrophic mineralization focuses found in spleen and pancreas can be due to the alterations of phosphorus and calcium even if not described in literature associated with calcium carbide poisoning. In conclusion, the clinical signs presented, and the image findings of this patient confirmed the danger and potentially lethal effects of the contact and ingestion of calcium carbide. The literature about this subject is scarce and deeply in need of more attention associated with new measures for prevention, instruction to owners and development of diagnose and treatment emergency protocols of these animals.

**Keywords:** ultrasonography, dog, intoxication, toxicity.

DOI: 10.22456/1679-9216.130030

Received: 10 June 2003 Accepted: 6 October 2023 Published: 3 November 2023

[1]Department of Veterinary Surgery, UNESP, Jaboticabal, SP, Brazil. [2]Department of Veterinary Medicine, USP, Pirassununga, SP, Brazil. [3]Department of Large Animals Clinic & [4]Department of Small Animals Clinic, Federal University of Santa Maria (UFSM), Santa Maria, RS, Brazil. CORRESPONDENCE: M.A.R. Feliciano [marcusfeliciano@yahoo.com]. FZEA - Universidade de São Paulo (USP). Rua Duque de Caxias n. 225. Jardim Elite. CEP 13635-900 Pirassununga, SP, Brazil.

## INTRODUCTION

Calcium carbide has space in industry and domestic use for fruit ripening, despite its negative and harmful effects [14]. Its ability to undergo chemical reactions leads to the release of acetylene gas and heat capable of causing burns and damage to mucous membranes, skin and eyes [14,15]. The gas has the characteristic of depressing the central nervous system and has already been described as an anaesthetic agent [1,13], when accidentally inhaled, it can lead to headaches, dizziness, mood disturbances, sleep, mental confusion, memory loss, edema cerebral palsy and seizures [15,16,19].

Lethal effects are described at a concentration of 9% for rats, 35% for humans and 80% for dogs [8]. Traces of arsenic and phosphorus are associated with dry mouth, fissured tongue, skin ulcerations, weakness, dizziness, thirst, dysphagia and vomiting when there is chronic exposure. Symptoms of acute cases range from severe esophageal and gastric ulcerations to perforation and convulsions [8,14,17,19]. There are also indications of teratogenic, mutagenic effects and effects that affect reproductive capacity [3].

Reports involving this substance in animals are scarce, but intoxications are routine in the care of small animals, whether intentional or accidental. In a 6-year survey, 15,568 cases of intoxication were seen, both in dogs and cats, in just one institution [12]. The importance of the reports, identification of the agents and their effects aims at the dissemination of information and agility in the service. This report aims to present laboratory, ultrasonographic and radiographic findings in a case of calcium carbide poisoning in a dog.

## CASE

A 8-year-old American Staffordshire Terrier arrived at the Veterinary Hospital of Universidade Federal de Santa Maria (UFSM) with the main complaint of emesis and diarrhea since the day before the consultation, which evolved into bloody diarrhea with a fetid odor. During the anamnesis, the owner stated that the patient had hyporexia and olygodipsia for 3 days when she ingested calcium carbide that was stored in a mechanical workshop. The animal had a previous diagnosis of hypothyroidism which was treated with sodium liothyronine[1] at a dose of 800 mg prescribed by a colleague and whose dose was reduced to 600 mg by the owner without veterinary indication.

On physical examination, the patient presented an alert state of consciousness, high body score (obese), pelvic limb claudication, hyperaemic mucous membranes with elevated time of capillary perfusion time of 3 s and an increase of mandibular and popliteal lymph nodes bilaterally palpable. Other parameters measured during the physical examination include rectal temperature of 37.1ºC, percentage of dehydration of 7, respiratory rate of 24 movements/min and heart rate of 180 beats/min. The presence of brownish skin lesions on the forelimbs was also verified, and it was not possible to state whether they were correlated with the intoxication condition.

Based on the clinical findings and animal's history, clinical suspicions of calcium carbide intoxication, "diabetes mellitus" and hyperadrenocorticism were raised. After consultation, the patient was hospitalized for follow-up and complementary imaging (ultrasound and radiography) and haematological exams, with blood count and complete biochemical profile (albumin, ALT, AST, cholesterol, creatinine, creatine kinase, alkaline phosphatase, fructosamine, glucose, total proteins, triglycerides and urea).

During hospitalization, the patient was medicated with dipyrone[2] [Dipirona Ibasa 50%® - 15 mg/kg, i.v, TID, for 3 days], tramadol hydrochloride[3] [Tramal® - 3 mL/kg, i.v, TID, for 2 days], sucralfate[4] [Sucrafilm® - 5 mg/mL, v.o, TID, for 2 days], maropitant citrate[5] [Cerenia® - 0.1 mg/kg, sc, SID, for 3 days], omeprazole[6] [Gaviz® V - 1 mg/kg, i.v, SID, for 3 days], levothyroxine[7] [Tyrox® 300mg - ½ (half) tablet, p.o., BID, for 2 days].

The result of the patient's haematological profile examination showed alterations in cells such as: total leukocytes in the amount of 19,600, 196/µL of rods, 18,032/µL of segmented neutrophils, 196/µL of lymphocytes. In addition, neutrophils were hyposegmented in the evaluation of the blood smear slide. In the biochemical test, there were alterations in the values of albumin (2.6 g/dL), alkaline phosphatase (2,162 IU/L) and triglycerides (175 mg/dL).

For the abdominal ultrasound examination[8], a trichotomy was performed to remove hair in the region of interest and the patient was positioned in dorsal decubitus. Afterwards, ultrasound gel was applied to favour the contact of the transducer with the animal's skin and the scan was performed with the micro convex and linear transducers.

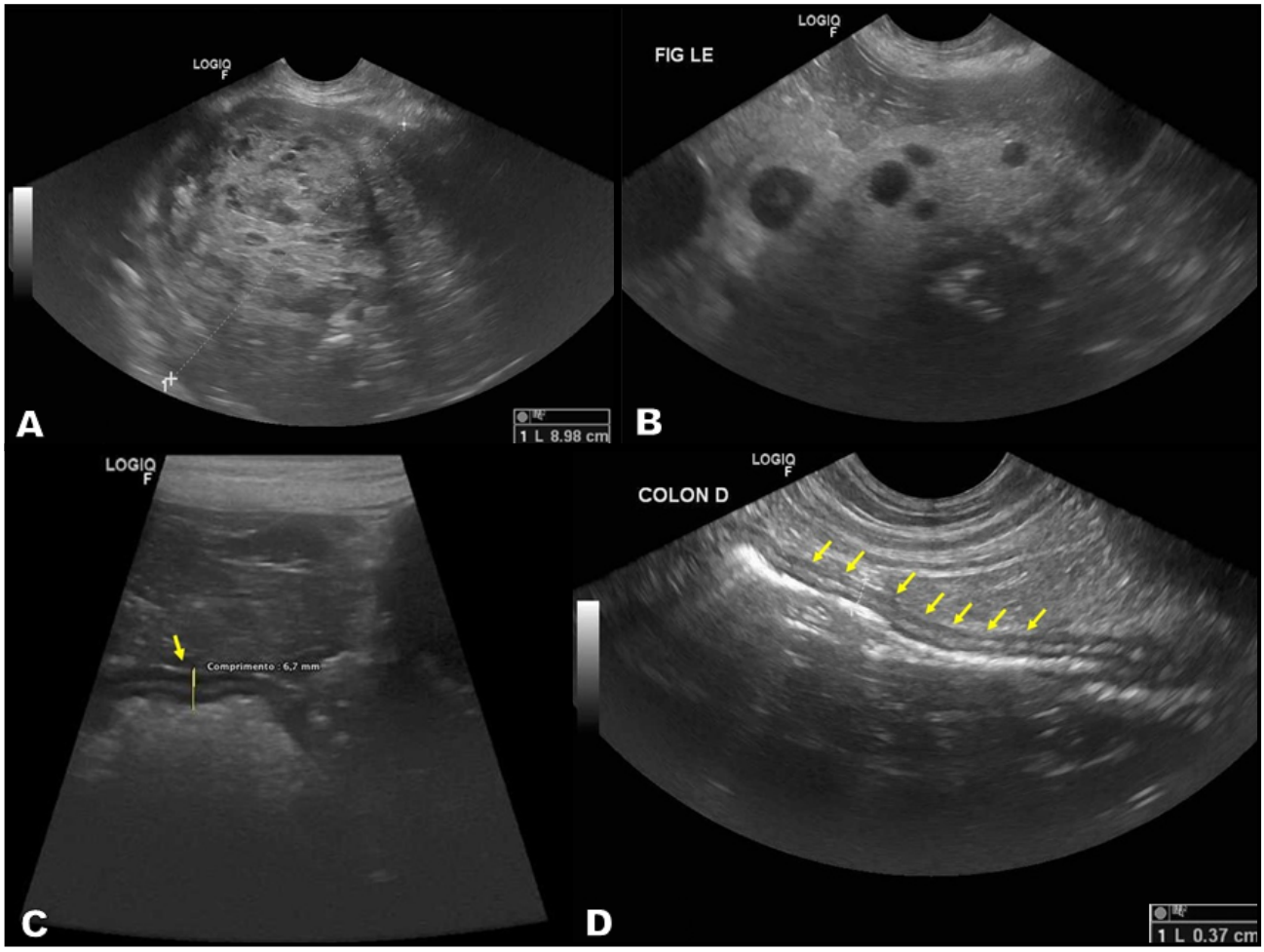


**Figure 1.** Calcium carbide poisoned canine abdominal ultrasonographic image A- In spleen tissue it is possible to verify circumscribed formation in splenic body measuring 8.98 cm of height, with irregular contours, heterogenous echogenicity and hyperechoic areas. B- Left hepatic lobe it is possible to verify increase in size, hyperechoic and heterogenous circumscribed areas (asterisk) of regular and hyperechoic contours. C- Thickened stomach wall in area of gastric fundus, measuring 0.67 cm of thickness and with muscular and mucosal layers more evident. D- Descending colon, thickened wall (measuring 0.37 cm) and evident stratification (arrows).

Ultrasonographic findings included splenomegaly associated with a circumscribed mass located in the body region, measuring approximately 9.3 cm long x 8.98 cm high, characterized by irregular contours and heterogeneous echogenicity with hyperechogenic areas forming a discreet acoustic shadow (Figure 1A). The liver showed increased dimensions, smooth contours and hyperechogenic and heterogeneous parenchyma, with countless circumscribed foci with regular and diffusely distributed hypoechogenic contours (Figure 1B). Some of these foci had mixed echogenicity, with the largest measuring approximately 1.71 cm long x 1.63 cm high.

The stomach was partially visualized, with the presence of intraluminal gaseous content in the portions where it was possible to perform the evaluation. The gastric wall showed thickening in the gastric fundus region, measuring approximately 0.67 cm (Figure 1C). The stratification of the layers was preserved, but the muscle and mucosal layers were evident.

As for the descending colon, a portion had a thickened wall (measuring approximately 0.37 cm - Figure 1D) and evident stratification, with the submucosal layer appearing to be more noticeable and thicker than the others, associated with the presence of a hyperechogenic area measuring approximately 0.21 cm in length (Figure 2A). The cecum was visualized and evaluated, showing intraluminal gaseous content and an evident stratified wall.

The pancreas presented a slightly heterogeneous and hypoechogenic parenchyma in the visualized portions, with thickening in a 1.04 cm body region associated with small hyperechogenic foci scattered throughout the parenchyma (Figure 2B). In addition, there was a larger area in the body region that measured approximately 0.87 cm in diameter.

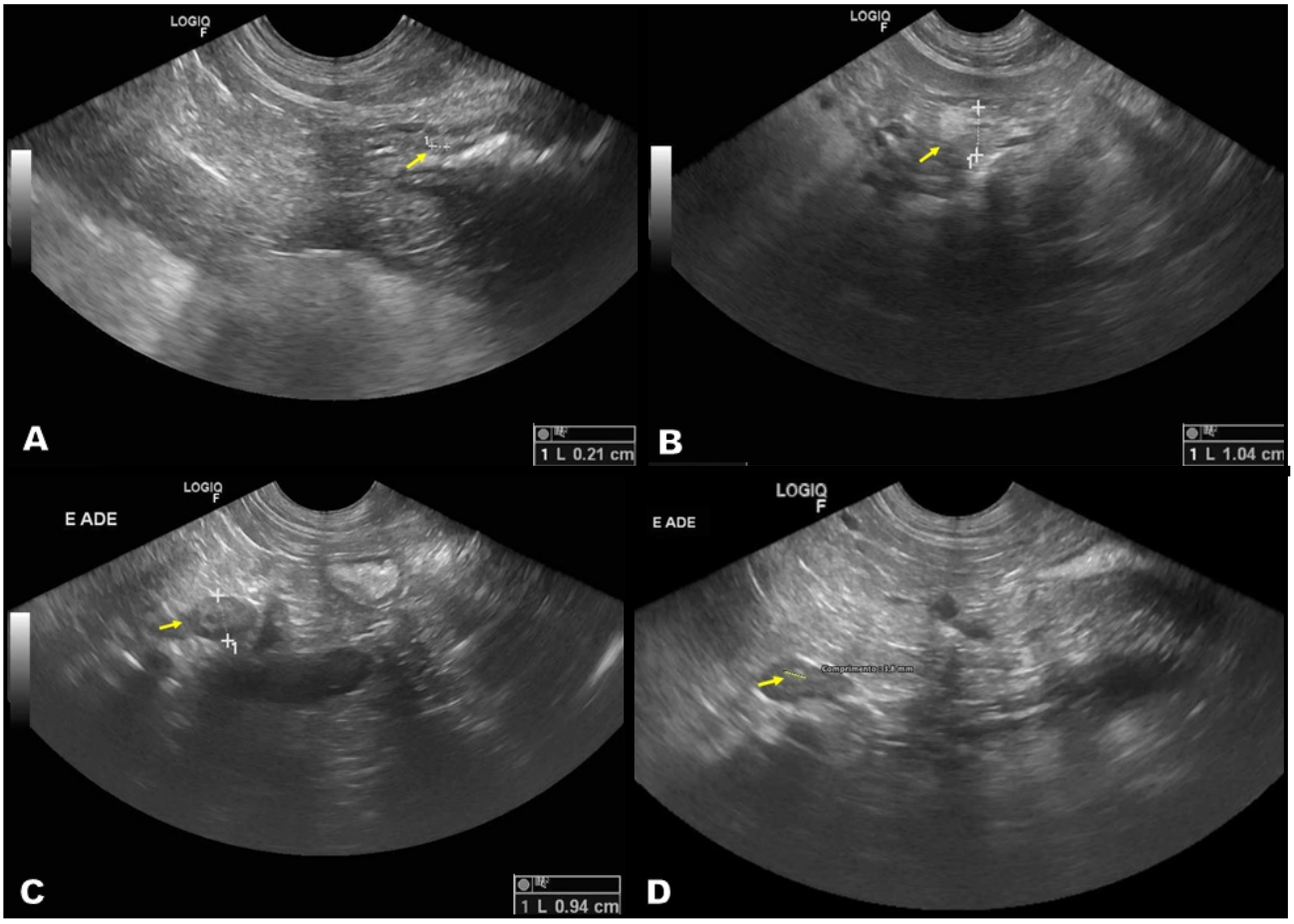


**Figure 2.** Calcium carbide poisoned canine abdominal ultrasonographic image. A-Hyperechoic areas in descending colon, measuring 0.21 cm of thickness (arrow). B- Slightly heterogenous and hypoechoic spleen parenchyma (arrow), with thickness of the body area of spleen (measuring 1.04 cm) associated to small hyperechoic and disperse focuses on parenchyma. C- Left adrenal with increase in dimensions of the posterior pole (arrow), measuring 0.94 cm of thickness. D- In anterior pole of the left adrenal gland, presence of circumscribed hyperechogenic areas, measuring 0.38 cm length (arrow).

The left adrenal was identified and had the caudal pole with enlarged dimensions, measuring approximately 0.94 cm in diameter (Figure 2C) and the cranial pole presented a circumscribed and hyperechogenic area that measured approximately 0.38 cm in diameter (Figure 2D). The right adrenal was not visualized and, therefore, it was not possible to perform its evaluation.

After the consultation and carrying out the complementary tests mentioned above, the animal owner did not return for reassessment of the patient's condition.

## DISCUSSION

Calcium carbide is a light grey powder, and it has been used in the industry for maturation of fruits since they can't be transported and distributed when mature. This substance is an alkali and when in contact with moisture it goes through chemical reactions that lead to the release of acetylene gas that is also present in the natural process of fruit maturation [8]. Although acetylene gas is forbidden, it is also highly used in the welding process and cutting of metals [4,19]. As a powder it is found in domestic use as a potent way to unclog drains [8].

When inhaled it is capable of causing depression in the central nervous system, can also lead to other serious damage like mucous burns and eyes and skin irritation. When accidental ingestion of the powder occurs it leads to an acute toxicity of variable degree, that can be light and manageable or develop to a mucositis of III-degree, esophageal erosion and gastric ulcers [8].

This substance is extremely dangerous to the human body because it contains arsenic and phosphorus hydrate traces with potential cytotoxic effects [4]. The first arsenic and phosphorus intoxication symptoms include vomit, diarrhea with or without blood, chest and abdomen burn sensation, thirst, weakness and difficulty to swallow [20]. It is potentially related to cancer and neurologic effects [3].

Regarding the treatment there is no specific antidote, when direct contact occurs with $CaCO_2$ it is indicated to wash the affected area with a lot of water and when ingested, the orientation is to increase water consumption to minimize the irritation. It is not recommended to proceed with a gastric lavage procedure or to induce emesis. In case there is compromised central nervous system or pulmonary it is necessary to start support treatment [8].

Although diagnose of poisoning cases are made by clinical findings and possible contact with the substance, to aid the diagnosis and in understanding the possible lesions is a patient, using the ultrasound becomes a valuable tool. With it, it`s possible to make a better treatment strategy based on the gravity of lesions and to prevent further complications. In this case, gastrointestinal alterations found are compatible with clinical signs previously associated with calcium carbide intoxication. Some of the findings indicate inflammation like the thickening of the gastric fundus wall, colon. Also, the hyperechoic area in the mucosal layer described in the colon is an indication of an ulceration process in progress [2].

Hepatomegaly and diffuse alterations in the echogenicity and texture are described. These can be related with a process of a diffuse, acute reaction. This reaction may have a systemic origin but yet without alterations of the organ wall which is compatible with the reactions described for the carbide intoxication [9]. Due to the proximity between the organs and the intimate vascular communication, many hepatic alterations can reflect in the spleen and leas to diffuse alterations [9].

In this case report, spleen and pancreas showed mineralized areas, this type of alteration is usually related with a chronic process and, in this case, may be related with the clinical signs if he had a long exposure to this chemical agent. This type of alteration is not described in the literature, but dystrophic mineralization, as visualized in the exam, can be related with an elevation of calcium and phosphorus in the blood which leads to a deposition of the same in soft tissue [6].

Like the liver, the pancreas had its dimensions bigger than normal values. This organ is closely related to the digestive process of proteins, fat and carbohydrates, it also regulates blood glycose through insulin production [18]. Its vascular supply is connected with liver, spleen and small intestine especially duodenal portion [18]. The thickening of its wall is compatible with the inflammatory process seen in its adjacent organs, that may be a direct consequence of the substance ingestion, or an indirect response caused by the reaction in the area that is mostly in contact to it.

In a general way, it's possible to affirm that the image findings described suggest an inflammation of the digestive tract (specially the final portion) with signs of imminent ulceration of the descending colon [2]. All that was described in this case report can be similarly found in the literature regarding the effects of calcium carbide intoxication in humans. These indicate that there are signs of gastroenterocolitis with possible ulcer formation associated.

The ultrasonographic alteration found in the liver are reinforced by laboratorial findings of increased alkaline phosphatase. This enzyme is synthetized in the liver, osteoblasts, intestinal and renal epithelium and in the placenta. Due to its short half-life and low production levels in other tissues, the main responsible for blood increased levels of this enzyme. It can occur in cases of cholestasis, induced by drugs or chronic disease and neoplasia specially in dogs. Inflammatory conditions can contribute to the increase of alkaline phosphatase in the blood also by the obstruction of small biliary vessels [10].

The leucocytosis and lymphopenia described indicate and infectious process associated that can be related with the gastrointestinal inflammation which allows bacterial passage due to lesions of the first protective barrier [10]. None of the laboratory alterations observed are specific for calcium carbide intoxication but they are compatible with the pathophysiological process initiated by the ingestion of this substance [7].

Due to the easy access to this substance and its high potential damage to the health of animals and people, it would be of public interest to create means to educate people about the commercial products that are related with this chemical. Also, some biological measures should be stablished to avoid direct and indirect contact with this substance. An example would be the use of natural food maturation process by putting mature fruits close, so they can liberate acetylene gas and accelerate the process of maturation of other fruits without health risks [8]. There are already some laws that forbidden buying, selling and the industrial use of calcium carbide in food [5]. Also, in some areas of the country its domestic use is forbidden, like happens in Manaus by Municipal Law 1945/2014 [11].

Diagnostic image, clinical and pathological information are poor in veterinary medicine, when refereeing to specific findings of accidental or intentional calcium carbide intoxication in animas. By these data described in this case report, we can conclude that ultrasonography had and important hole in identifying and evaluating the presence and extension of lesions caused by the ingestion of the substance in a dog. Besides, the images were of great relevance to develop a correct treatment strategy and evaluate progress of the patent. Even with this report contribution to literature, its believed that further studies are necessary to define with more precision the damage caused in an animal body by the ingestion of this substance, and with that, develop fast and efficient treatment protocols.

MANUFACTURES

[1]Laboratório Enila Indústria e Comércio de Produtos Químicos e Farmacêuticos S.A. Rio de Janeiro, RJ, Brazil.
[2]Laboratório Ibasa Ltda. Porto Alegre, RS, Brazil.
[3]Grünenthal do Brasil Farmacêutica Ltda. São Paulo, SP, Brazil.
[4]EMS - Pharma Farmacêutica. Hortolândia, SP, Brazil.
[5]Zoetis Inc. Campinas, SP, Brazil.
[6]Agener União Saúde Animal - Grupo União Química Farmacêutica Nacional S.A. São Paulo, SP, Brazil.
[7]Cepav Pharma Ltda. São Paulo, SP, Brazil.
[8]GE HealthCare Technologies Inc. Chicago, IL, USA.

***Acknowledgements.*** The authors would like to thank the Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) (n. process - 305182/2020-0).

***Declaration of interest.*** The authors report no conflicts of interest. The authors alone are responsible for the content and writing of paper.

## REFERENCES

1 **Adriani J. 1962.** The Chemistry and Physics of Anesthesiology. *Anestesiology*. 23(4): 592. DOI: 10.1097/00000542-196207000-00025.

2 **Amado L.V. & Assis A.R. 2019.** Estômago e Intestinos. In: Feliciano M.A.R., Assis A.R. & Vicente W.R.R. (Eds). *Ultrassonografia em Cães e Gatos*. São Paulo: MedVet. pp.236-264.

3 **Bafor E.E., Greg-Egor E., Omoruyi O., Ochoyama E. & Omogiade G.U. 2019.** Disruptions in the female reproductive system on consumpiton of calcioum carbide ripened fruit in mouse models. *Heliyon*. 5(9): e02397. DOI: 10.1016/j.heliyon.2019.e02397.

4 **Bini M., Rajesh B. & Babu T.D. 2020.** Acute and subacute toxicity evaluation of calcium carbide and ethylene glycol in Wistar albino rats. *Journal of Basic and Clinical Physiology and Pharmacology*. 31(1): 20190126. DOI: 10.1515/jbcpp-2019-0126.

5 **Food Safety and Standards Authority of India (FSSAI). 2011.** Food Safety and Standards (Prohibition and Restrictions on Sales) Regulations, 2011. New Delhi: FSSAI, 7p.

6 **Górniak S.L. 2020.** Raticidas. In: Spinosa H.S., Górniak S.L. & Palermo Neto J. (Eds). *Toxicologia Aplicada à Medicina Veterinária*. 2.ed. Barueri: Manole, pp.215-224.

7 **Igbinaduwa P.O. & Aikpitanyi-Iduitua R.O. 2016.** Calcium carbide induced alterations of some haematological and serum biochemical parameters of wistar rats. *Asian Journal Pharmaceutical and Health Sciences*. 6(1): 1396-1400.

8 **Jindal T., Agrawal N. & Sangwan S. 2013.** Accidental Poisoning with Calcium Carbide. *Journal of Clinical Toxicology*. 3: 159. DOI: 10.4172/2161-0495.1000159.

9 **Lacreta Jr. A.C.C. & Guimarães B.L.L. 2019.** Fígado. In: Feliciano M.A.R., Assis A.R. & Vicente W.R.R. (Eds). *Ultrassonografia em Cães e Gatos*. São Paulo: MedVet, pp.265-324.

10 **Lassen E.D. 2007.** Avaliação laboratorial do fígado. In: Thrall M.A., Baker D.C., Campbell T.W. (Eds). *Hematologia e Bioquímica Clínica Veterinária*. São Paulo: Roca, pp.335-354.

11 **MANAUS. 2014.** Lei Municipal Nº 1945, de 12 de dezembro de 2014: Proíbe o uso de substâncias químicas para amadurecimento de frutas, e dá outras providências. Manaus: Diário Oficial do Município de Manaus (DOM-MANAUS), 1p.

12 **Medeiros R.J., Monteiro F.O., Silva G.C. & Nascimento Jr. A. 2009.** Casos de intoxicações exógenas em cães e gatos atendidos na Faculdade de Veterinária da Universidade Federal Fluminense durante o período de 2002 a 2008. *Ciência Rural*. 39: 2105-2110. DOI: 10.1590/S0103-84782009005000151.

13 **Miller S.A. 1965.** Properties of Acetylene. In: *Acetylene: its properties, manufacture, and uses*. London: Ernest Benn, pp.1-11.

14 **Olisah M.C., Ilechukwu O.U., Ifemeje J.C. & Ofor C.C. 2017.** Effect of vitamin C against calcium carbide induced hepatotoxicity. *Tropical Journal of Applied Natural Sciences*. 2(1): 74-77. DOI: 10.25240/TJANS.2017.2.1.12.

**15 Okeke E.S., Okagu I.U., Okoye C.O. & Ezeorba T.P.C. 2022.** The use of calcium carbide in food and fruit ripening: Potential mechanisms of toxicity to humans and future prospects. *Toxicology*. 468: 153112. DOI: 10.1016/j.tox.2022.153112.

**16 Pässler P., Hefner W., Buckl K., Meinass H., Meiswinkel A., Wernicke H.J., Ebersberg G., Müller R., Bässler J., Behringer H. & Mayer D. 2011.** Acetylene. *Ullmann's Encyclopedia of Industrial Chemistry*. 1: 277-326. DOI: 10.1002/14356007.a01_097.pub4.

**17 Pedreschi F. & Murkovic M. 2022.** Possible health problems associated with the use of calcium carbide as a source of the ripening gas acetylene. *Global Harmonization Initiative*. 4. Disponível em: https://www.globalharmonization.net/library/reports-from-working-groups.

**18 Pereira E.A.S. & Paula G.N. 2019.** Glândulas Adrenais e Pâncreas. In: Feliciano M. A.R., Assis A.R. & Vicente W.R.R. (Eds). *Ultrassonografia em Cães e Gatos*. São Paulo: MedVet, pp.368-393.

**19 Per H., Kurto lu S., Ya mur F., Gümü H., Kumanda S. & Poyrazo lu H. 2007.** Calcium carbide poisoning via food in childhood. *The Journal of Emergency Medicine*. 32(2): 179-180. DOI: 10.1016/j.jemermed.2006.05.049.

**20 Ur-Rahman A., Chowdhury F.R. & Alam M.B. 2008.** Artificial Ripening: What We are Eating. *Journal of Medicine*. 9(1): 42-44. DOI: 10.3329/jom.v9i1.1425.