

## PULMONARY GAS EMBOLISM IN RABBIT CAUSED BY HYDROGEN PEROXIDE - CASE REPORT

### *EMBOLIA GASOSA PULMONAR EM COELHO CAUSADA POR PERÓXIDO DE HIDROGÊNIO – RELATO DE CASO*

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#### SUMMARY

Gas embolism caused by the use of hydrogen peroxide is a phenomenon that is not usually described in clinical routine; therefore, this case report describes the occurrence of massive venous air embolism in a rabbit during cleaning and disinfection of an abscess in the masseter using 3% hydrogen peroxide. By injecting hydrogen peroxide under pressure in the abscess cavity, the animal had a sudden cardiac arrest and died. During necropsy, numerous air bubbles (gaseous emboli) were observed in the caudal vena cava and pulmonary artery, pulmonary emphysema and atelectasis. Histopathological examination showed air bubbles in the blood vessels and pulmonary arteries of the region adjacent to the abscess and interstitial lung hemorrhage as well. The kidneys, liver and brain showed congestion. The sudden cardiorespiratory collapse when using this product, associated with the necropsy and histopathological findings suggest that the venous air embolism occurred due to massive use of hydrogen peroxide. Due to the severity of the case presented and the possibility of the occurrence of this pathology, the use of hydrogen peroxide should be avoided in situations when accumulation in cavities may happen.

**KEY-WORDS:** Gas embolism. Hydrogen peroxide. Masseteric abscess. Microembolism.

#### RESUMO

A embolia gasosa provocada pela utilização do peróxido de hidrogênio é um fenômeno que não é descrito na rotina clínica veterinária, neste sentido descreve-se a ocorrência de embolia gasosa venosa massiva em coelho durante a limpeza e desinfecção de um abscesso no masseter utilizando peróxido de hidrogênio a 3%. Ao injetar peróxido de hidrogênio sobre pressão na cavidade do abscesso, o animal teve uma parada cardiorrespiratória súbita e veio a óbito. A necropsia foram observadas inúmeras bolhas de ar (êmbolos gasosos) na veia cava caudal e tronco pulmonar, atelectasia e enfisema pulmonar. No exame histopatológico observou-se a presença de bolhas de ar em vasos da região adjacente ao abscesso e em artérias pulmonares bem como hemorragia intersticial pulmonar. Os rins, o fígado e o encéfalo apresentavam congestão. O colapso cardiorrespiratório súbito durante a utilização deste produto, associado aos achados de necropsia e histopatológicos sugerem que a embolia gasosa venosa massiva ocorreu devido à utilização do peróxido de hidrogênio. Devido à gravidade do caso apresentado e à possibilidade de ocorrência desta patologia, seu uso deve ser evitado em condições que determinem seu acúmulo em cavidades.

**PALAVRAS-CHAVE:** Água oxigenada. Abscesso massetérico. Embolismo gasoso. Microembolismo.

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Hydrogen peroxide 3% is an oxidizing agent often used for cleaning and disinfecting wounds (RIDER et al., 2008; VIDIL et al., 2008), despite evidence that the side effects outweigh the benefits (PAULINO, 2006). When in contact with peroxidases and catalases (enzymes present in the blood) is oxidized and forms a dense foam due to the release of H<sub>2</sub>O and O<sub>2</sub> (YUN, 1969; HUSSAIN-KHAN et al., 2004).

This substance used in injury or subcutaneous tissue might be absorbed and release O<sub>2</sub> in the vascular bed (KUZNETSOV, 1993; VIDIL et al., 2008) and when the volume of gas formed exceeds the solubilization capacity of the blood, oxygen bubbles are formed leading to gas embolism (SUN et al., 2004). Depending on the type of blood vessel where the bubbles are formed, embolism can be classified as arterial or venous (BÁRBARA et al., 2004), when formed in veins, these emboli may coalesce in the right ventricle and obstruct the pulmonary trunk causing fatal circulatory blockage (MAXIE; ROBINSON, 2006), thus characterizing massive venous air embolism (LOCALI & ALMEIDA, 2006).

There are several reports about the occurrence of air embolism caused by hydrogen peroxide in human patients, either by ingestion or by use on open wounds (SASTRE et al., 2001) or in abscess disinfection (HARLLER et al., 2002), this combined to the fact that this product can impair healing has made its use increasingly restrictive (PAULINO, 2006). Experimentally, the use of 0.25% hydrogen peroxide in the small intestine of dogs led to methemoglobin generation and air bubble formation in the hepatic portal vein (YUN, 1969).

In veterinary medicine, although only on the first approach to the injury, this substance is still widely used to treat contaminated wounds (PAULINO, 2006). Another less frequent application is the induction of emesis, when it is used orally (ANDRADE et al., 2008).

Due to limited data regarding gas embolism in animals caused by hydrogen peroxide, the occurrence data is limited to experimental work. This case report describes a massive venous air embolism in a rabbit caused by the use of 3% hydrogen peroxide during the disinfection procedure of a facial abscess.

An adult male rabbit weighing 1.1 kg underwent drainage of an abscess in the masseter. The rabbit was pre-anesthetized with acepromazine (0.1mg/kg, IM) associated with morphine (0.1mg/kg, IM), followed by the association tiletamine/zolazepan (10mg/kg, IM) after 15 minutes.

Initially, the site was punctured for a diagnosis evaluation of the wound contents, which confirmed the presence of caseous material. An incision was then made in the skin in order to drain the abscess. After the excess pus was removed, we started the administration of hydrogen peroxide through the incision using a 20 mL syringe coupled to a urethral probe. After successive washing (using approximately 15 mL), a final application of 5 mL of peroxide under pressure caused an increase of the abscess volume due to

foaming characteristic of the oxidation of hydrogen peroxide, while excess product came out through the incision.

Immediately after this last application, the animal had a cardiopulmonary arrest and died. Subsequently, the rabbit was then taken to the Animal Pathology Laboratory where necropsy was performed. During the necropsy, the presence of numerous bubbles (gaseous emboli) in the vena cava (Figure 1A) and pulmonary artery (Figure 1B) was observed. Pulmonary emphysema was also observed, affecting mainly the left cranial lobe and the ventral edges of both lungs (Figure 1C) and the same lesion could be observed to a lesser extent through the serosal surface of both lungs. In the ventral medial left lobe, there was striking atelectasis (Figure 1D) and the dorsal region of lungs appeared to be slightly ischemic.

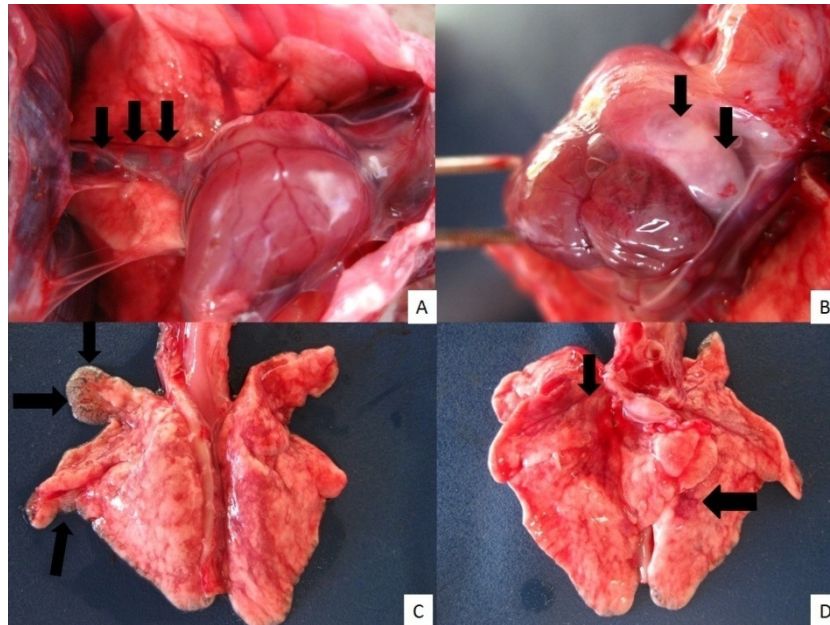
Histopathological examination showed the presence of air bubbles (gaseous emboli) in the lumen of blood vessels of the masseter (Figure 2A) and lungs (Figure 2B), as well as emphysema and interstitial pulmonary hemorrhagic foci (Figure 2C); moderate congestion in the brain (Figure 2D), liver and kidney was also observed. In the lung, the air bubbles partially blocked the diameter of the blood vessel and pressed the blood cells and adjacent endothelium.

Due to hydrogen peroxide related sudden death, the necropsy and histopathological lesions, we concluded that animal died of a massive pulmonary embolism by oxygen.

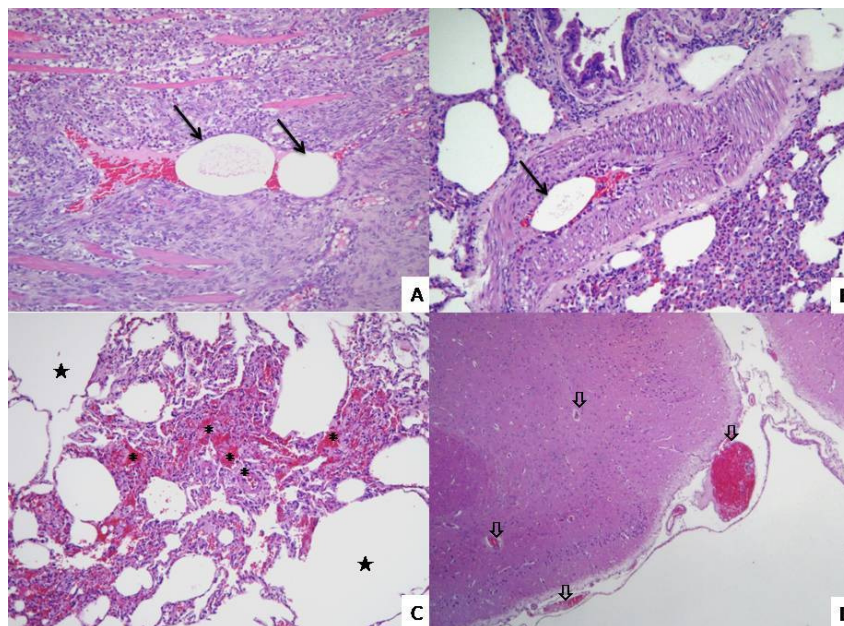
In humans, embolism has been documented from the ingestion of hydrogen peroxide (RIDER et al., 2008), its use in open wounds, irrigation of the thoracic cavity (SASTRE et al., 2001), irrigation of fracture (SUN et al., 2004) and abscesses (HARLLER et al., 2002), being fatal in some cases. Due to this, Hussain-Khan et al. (2004) warn that this product use should be restricted to cases of extreme necessity. In animals, venous gas embolism is a rare complication related to pneumocystography, laparoscopy and cryosurgery (MAXIE & ROBINSON, 2006), however, there are no reports correlating its occurrence with the use of this substance in the clinical routine.

The use of hydrogen peroxide in closed or semi-closed vascularized cavities under pressure may favor the occurrence of gas embolism (SASTRE et al., 2001; HALLER et al., 2002; VIDIL et al., 2008). This was probably one of the predisposing factors in the case reported, since the peroxide generated high pressure in the adjacent tissues of the abscess due to the release of large amounts of oxygen in a confined space; 1 mL of hydrogen peroxide releases about 9.8 mL of oxygen (HALLER et al., 2002).

The air bubbles observed during histopathological examination can be attributed to the passage of hydrogen peroxide through the intraepithelial spaces and capillary endothelium, when in contact with blood catalases the substance is oxidized releasing H<sub>2</sub>O and O<sub>2</sub> in the blood stream (KUSNETZOV, 1993). However, there is also the possibility that these bubbles were formed by the absorption of the O<sub>2</sub> released in the reaction (SASTRE



**Figure 1** - Images of Necropsy findings (arrows). A. numerous air bubbles in the caudal vena cava. B. Bubbles in the pulmonary trunk. C. Pulmonary emphysema. D. Atelectasis.



**Figure 2.** A - Histological section of abscess in the rabbit masseteric region showing air bubbles (arrows) in blood vessel amid the inflammatory infiltrate. B. Air bubble in the lumen of the pulmonary artery (arrow). C. multifocal interstitial hemorrhage (asterisks) and emphysema (stars) in the lung parenchyma. D. congestion of the brain and leptomeninges (arrows). Hematoxylin eosin 10x.

et al., 2001). Yun (1969) showed that, if the substance is absorbed unchanged from the intestinal mucosa it may form bubbles in the bloodstream. In whatever form O<sub>2</sub> reaches the blood stream, the bubbles are formed only when the gas volume exceeds the blood solubilization capacity leading to gas embolism (SUN et al., 2004).

Venous gas pulmonary embolism is characterized by microvascular obstruction by the small bubbles present in the lumen of the blood vessels. When the number of bubbles is large enough to obstruct the pulmonary trunk, the right ventricular overload occurs accompanied by acute congestive heart failure, often fatal (MAXIE & ROBINSON, 2006), thus characterizing the massive venous air embolism. Thus, there is increased pulmonary artery pressure, central venous pressure and reduced cardiac output culminating with signs of circulatory shock (LOCALI & ALMEIDA, 2006), which certainly happened to this animal. The alveolar hypoperfusion leads to blood gas changes such as the reduction of the CO<sub>2</sub> tidal volume, hypercapnia and hypoxemia (LOCALI & ALMEIDA, 2006; NELSON & COUTO, 2010).

The increase in vagal tone, mediated by the action of hydrogen peroxide in vascular chemo and mechanoreceptors in vagal terminations may have contributed to the massive embolism that caused the death of the animal, since it can lead to bronchospasm, respiratory arrest, hypotension and arrhythmias (KUSNETZOV, 1993). In humans, it leads to tachyarrhythmia and atrial ventricular blockage, while the degree of symptoms observed is determined by the ability of the right ventricle to overcome the blockade imposed by the emboli (LOCALI & ALMEIDA, 2006).

The fact that the animal had voluntary movements (sometimes forceful) during the procedures led us to exclude the possibility of complications related to anesthesia protocol and, on the other hand, cardiac arrest happened immediately after a relatively large volume (5 mL) of hydrogen peroxide was infused, which led us to clinically suspect pulmonary embolism. Cardiac arrest occurred without any noticeable signs.

The situation described in this case report as well as proven damages that the indiscriminate use of hydrogen peroxide has caused, warns against its use in procedures to clean and disinfect wounds in animals; moreover, it should be avoided altogether when there is a possibility that it can accumulate in cavities.

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