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# White glue intoxication in a plain parakeet (*Brotogeris tirica*) – Case report

## Intoxicação por cola branca em periquito-verde (*Brotogeris tirica*) – Relato de caso

## Intoxicación por cola blanca en periquito-verde (*Brotogeris tirica*) – Relato de caso

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**Abstract**
Despite the lack of information on avian sensitivity to toxic agents, it is known that several products that are relatively harmless to humans may have profound effects on birds. Thus, the objective of this work is to report the case of a plain parakeet (Brotogeris tirica) that was intoxicated after ingesting white glue and to discuss the pathophysiological aspects involved in intoxication. For this, the case study methodology was used, with a qualitative character, describing the case in detail and posteriorly seeking subsidies in the literature centered on the subject under study to complement the knowledge necessary to carry out this study. Twelve hours after ingesting white glue, the plain parakeet (Brotogeris tirica) began to experience recurrent vomiting episodes, which rapidly progressed to a critical condition of dyspnea, prostration and death. Although Polyvinyl Acetate (PVAc), the main component of that type of glue, is described as a non-hazardous substance, necroscopic and histopathological findings, consisting of pulmonary and hepatic congestion and hemorrhage, were very similar to that seen in some avian toxicoses. A thorough review on in vivo and in vitro mechanisms of PVAc degradation suggests that an association of high-dose exposition and B-esterase insufficiency in birds was the probable cause of PVAc poisoning, serving as an alert to veterinarians and bird owners.
**Keywords:** Plain parakeet; *Brotogeris tirica*; Avian; Parakeets; Hemorrhage; Congest; Dyspnea; Poisoning.

**Resumo**
Apesar da falta de informações sobre a sensibilidade das aves a agentes tóxicos, sabe-se que diversos produtos relativamente inofensivos ao homem podem ter efeitos profundos nas aves. Dessa forma, o objetivo deste trabalho é relatar o caso de um Periquito-verde (Brotogeris tirica) que foi intoxicado após a ingestão de cola branca e discutir os aspectos fisiopatológicos envolvidos na intoxicação. Para isso, utilizou-se a metodologia do tipo estudo de caso, de caráter qualitativo, descrevendo o caso de forma detalhada e posteriormente buscando subsídios na literatura centrados na temática em estudo para complementar o saber necessário para a realização deste estudo. Doze horas após a ingestão de cola branca, o Periquito-verde (Brotogeris tirica) começou a apresentar episódios recorrentes de vômitos, que rapidamente evoluíram para um quadro crítico de dispneia, prostração e óbito. Embora o Acetato de Polivinila (PVAc), principal componente desse tipo de cola, seja descrito como uma substância não perigosa, os achados necroscópicos e histopatológicos, consistindo em congestão e hemorragia pulmonar e hepática, foram muito semelhantes aos observados em algumas toxicoses aviárias. Uma revisão minuciosa dos mecanismos in vivo e in vitro da degradação do PVAc sugere que uma associação de exposição a altas doses e insuficiência de B-esterase em aves foi a provável causa da intoxicação por PVAc, servindo de alerta para veterinários e proprietários de aves.
**Palavras-chave:** Periquito-verde; *Brotogeris tirica*; Ave; Periquitos; Hemorragia; Congestão; Dispneia; Intoxicação.

**Resumen**
A pesar de la falta de información sobre la sensibilidad de las aves a los agentes tóxicos, se sabe que varios productos que son relativamente inofensivos para los humanos pueden tener efectos profundos en las aves. Así, el objetivo de este trabajo es reportar el caso de un Periquito Verde (Brotogeris tirica) que se intoxicó después de ingerir cola blanca y discutir los aspectos fisiopatológicos involucrados en la intoxicación. Para ello se utilizó la metodología del tipo

estudio de caso, con carácter cualitativo, describiendo el caso en detalle y posteriormente buscando subsidios en la literatura centrada en el tema objeto de estudio para complementar los conocimientos necesarios para la realización de este estudio. Doce horas después de ingerir cola blanca, el Periquito Verde (Brotogeris tirica) comenzó a presentar episodios recurrentes de vómitos, que rápidamente evolucionaron a un estado crítico de disnea, postración y muerte. Aunque el Acetato de Polivinilo (PVAc), el componente principal de este tipo de pegamento, se describe como una sustancia no peligrosa, los hallazgos necroscópicos e histopatológicos, consistentes en congestión y hemorragia pulmonar y hepática, fueron muy similares a los observados en algunas toxicosis aviares. Una revisión minuciosa de los mecanismos in vivo e in vitro de la degradación de PVAc sugiere que una asociación de exposición a dosis altas e insuficiencia de B-esterasa en aves fue la causa probable de intoxicación por PVAc, lo que sirve como advertencia para veterinarios y propietarios de aves.
**Palabras clave:** Periquito verde; *Brotogeris tirica*; Pájaro; Periquitos; Hemorragia; Congestión; Disnea; Intoxicación.

## 1. Introduction

Psittaciformes is an order of brightly colored zygodactyl birds, with short, hooked beaks, which comprise about 375 species of parrots, including macaws, amazons, parakeets, cockatoos, cockatiels, and lorikeets, among others. Because of their sociable nature, exuberant coloring and ability to imitate sounds, many species became popular as pets (Heatley & Conejo, 2012). However, captivity is often related to a myriad of health problems in wild animals, which also include involuntary exposure and poisoning by toxic substances. Although illness caused by toxic agents are relatively rare in pet birds, captivity leads to their unwanted exposure to various man-made and natural products that can cause intoxication, especially parrots, because of their natural chewing and exploring behavior (Bauck & Labounde, 1997).

This report will describe the case of a plain parakeet (Brotogeris tirica), that got poisoned by the ingestion of white glue. Although Polyvinyl Acetate (PVAc), the main component of this kind of glue, is described to be non-toxic, the signs and symptoms presented by the animal were very similar to that seen in some toxicosis, including hepatic and pulmonary congestion, diffuse hemorrhage, and sudden death.

## 2. Methodology

The present work is a case study, which aims to make a description and analysis as detailed and complete as possible of a case that presents some particularity or characteristic that makes it special and differential. (Pereira, Shitsuka, Parreira & Shitsuka, 2018). As Pereira et al., (2018) guide, before carrying out the case study, we searched the literature to see if there were similar reports, to attest to the relevance of the case. Subsequently, we searched for subsidies in the literature, using PubMed, Google Scholar, and Scielo databases, focused on the subject under study to complement the knowledge necessary to carry out the case study.

The present case study meets the methodological requirements for this type of research described above, since the case is fully detailed, and has scientific relevance since there are no previously reported cases of white glue poisoning in birds in the literature. The publication of bird data and images for this case study was authorized by the pet owner.

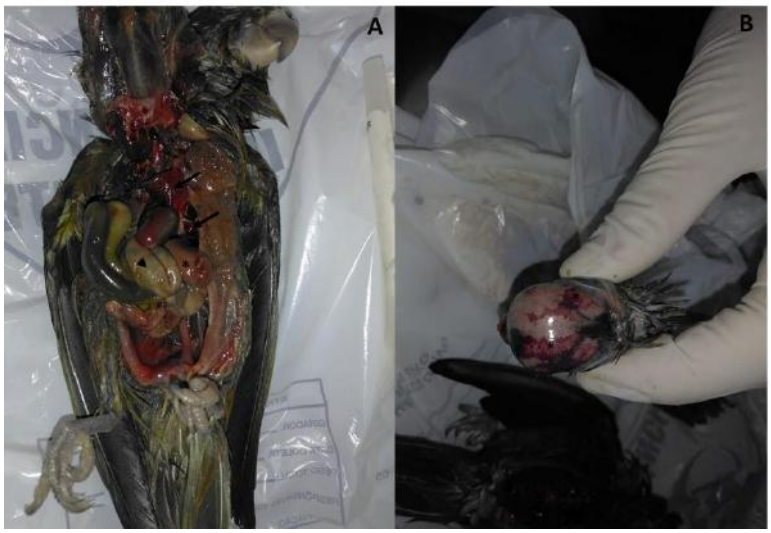
The content analysis of this case study is quantitative, like most case studies, since we seek to interpret the data about the phenomenon under study in a descriptive way concerning studies and authors cited in the literature (Pereira et al., 2018).

## 3. Case Report

Twelve hours after ingesting dry household glue from a handcrafted nest, a plain parakeet (Brotogeris tirica), six months old, unknown sex, suddenly began to experience recurrent vomiting episodes, which rapidly progressed to a critical condition of dehydration, weight loss, dyspnea and severe prostration. During the process, the feces became dark and diarrheic, with partially digested blood. Urate changed its color to mustard yellow, suggesting gastrointestinal and hepatic damage.

Support therapy, with oxygen, fluids, gastric and hepatic protectors, a single dose of dexamethasone sodium phosphate (rapid-acting corticosteroid) and broad-spectrum antibiotics were tried, but, unfortunately, the animal died about 36 hours after the beginning of the symptoms. Gross necropsy was performed immediately after death and revealed pulmonary congestion and hemorrhage; massive discoloration of hepatic parenchyma; gaseous distension of bowel loops and the presence of free serosanguinous fluid inside the celomatic cavity and in the trachea lumen. Intracranial hemorrhage was also found (Figure 1). Histopathological analysis of liver (Figure 2) and lung (Figure 3) fragments showed extensive congestion and hemorrhage. Only a few inflammatory cells were noted. No infectious agents were observed.

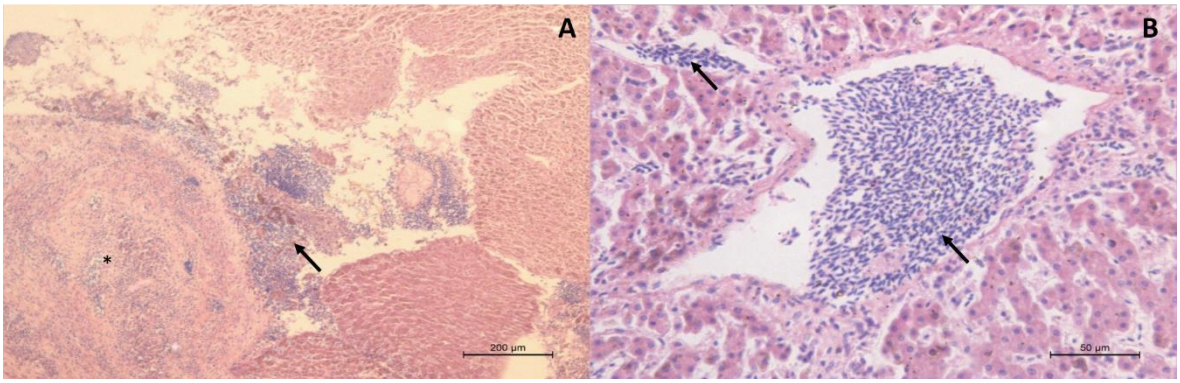
**Figure 1 –** Photos from the avian Brotogeris tirica necropsy. (A) Massive discoloration of hepatic parenchyma, suggesting hepatic necrosis (asterisk); pulmonary hemorrhage (arrows); gaseous distension of bowel loops (arrow head); free serosanguinous fluid inside the celomatic cavity and (B) intracranial hemorrhage (arrows) on gross necropsy.




Source: Authors.

In Figure 1, it is possible to verify images of the necropsy performed on the bird of the reported case, evidencing the organic alterations identified in the bird. Such necroscopic findings together with the histopathological analysis, clinical signs and history of the animal, help to suggest a possible process of intoxication by the ingested white glue.

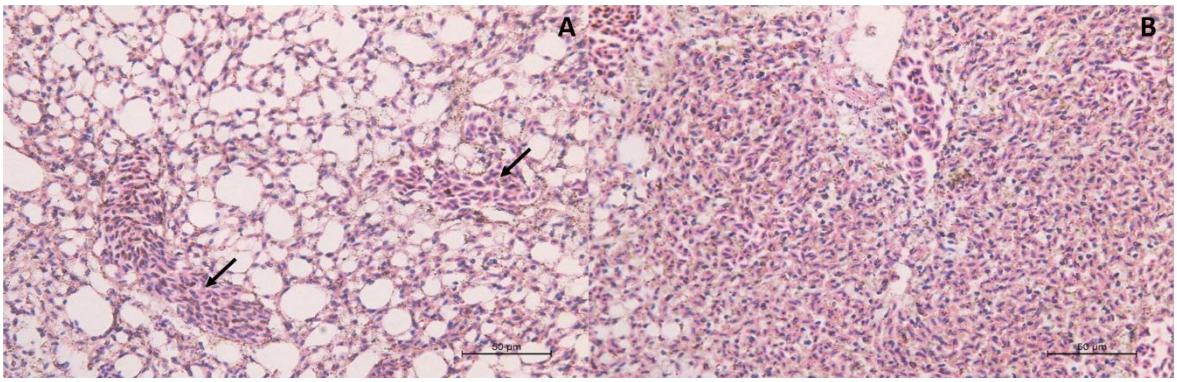
**Figure 2 –** (A) Photomicrographs of the liver, showing areas of necrosis (asterisk) and congestion (arrow). Bar = 200 µm. (B) Congested vessels are also seen in a larger image of the organ (arrows). Bar = 50 µm. Hematoxylin and Eosin stain.




Source: Authors.

Figure 2 shows two photomicrographs obtained from the histopathological examination of the bird's liver, revealing hepatic alterations, such as congestion and areas of necrosis, which may be present in processes that cause damage to the organ, such as intoxications.

**Figure 3 –** Photomicrographs of the lung, in which (A) congested vessels (arrows) and (B) diffuse hemorrhage can be observed. Bar = 50 µm. Hematoxylin and Eosin stain.




Source: Authors.

Figure 3 exhibits two photomicrographs obtained from the histopathological examination of the bird's lung, in which it is possible to observe the presence of congestion and hemorrhage, indicating pulmonary involvement.

## 4. Discussion

Avian anatomy naturally predisposes birds to be more sensitive than other animals to toxic substances. The efficiency of the avian respiratory system, for example, makes birds more susceptible to inhaled toxins, such as gases and fumes, which often result in acute death. In birds, inhaled air stays longer in the respiratory tract and, because of their high metabolic rate, inhaled toxins are quickly absorbed and distributed to the entire organism, causing damage (Heatley & Conejo, 2012). Another factor that contributes to high sensitivity is that birds can absorb excreted toxins from the cloaca. Moreover, the small size of the animals, as well as the small size of their livers (compared to the same sized mammals), also predispose them to accidental overdose of toxic substances due to hepatic overload, resulting in the accumulation of toxins in the organism. Besides that, to maintain their body temperatures at about 41oC to 42oC, birds require a high rate of food intake, and thus tend to be more susceptible to ingesting toxins (Walker, 1983).

Little is known about predisposing factors involved in avian sensitivity to toxic agents, however several products that are relatively harmless to humans may have profound effects on birds. The most common substances reported to cause toxicosis are ingested heavy metals, such as lead and zinc; inhalant toxins, for instance smoke from cooking or burning material and fumes from aerosols or cleaning agents; pesticides, toxic plants and foods (Bauck & Labounde, 1997; Guthrie et al., 2020; Vetere et al., 2020).

Lead poisoning is one of the most frequently reported and clinically known from the pet clinic. Lead is a highly present element in homes, and can even be found in bird toys, wheel swings, lead-based paints, varnishes, bells with lead clappers, and jewelry used by tutors, among other products. Clinically, lead intoxication can be acute or chronic, and the severity of intoxication is determined by the amount of lead ingested. Clinical signs include lethargy, depression, anorexia, weakness, regurgitation, polyuria, diarrhea, ataxia, and seizures, among other signs, which can lead to death. Some birds may

die without any clinical signs, and others showing only weakness and weight loss (Dumonceaux & Harrison, 1994; Martel, Doss & Mans, 2020). Zinc is another heavy metal that causes poisoning in birds. The galvanized wire used to construct enclosures is a common source of zinc, causing a poisoning often referred to as "new wire disease", as the shinier the wire, the greater the amount of zinc. The most common clinical signs are polyuria, polydipsia, gastrointestinal problems, weight loss, weakness, anemia, cyanosis, and seizures. Histopathological findings include focal mononuclear degeneration in the liver, kidney, and pancreas (Dumonceaux & Harrison, 1994; Savarese et al., 2020).

Toxic plants can also cause poisoning in birds. However, plant poisoning is rare due to the birds' habit of shredding leaves more than ingesting them, in addition to the rapid gastrointestinal transit of plants, factors that may play a role in the low incidence of plant poisoning. Harmful foods offered to birds by owners can also cause poisoning, through single or chronic exposure. Chocolate and foods with a high amount of sodium are the main contraindications for birds. Another common cause of poisoning in birds is poisoning from inhalant gases such as tobacco products and polytetrafluoroethylene (PTFE) gas (Dumonceaux & Harrison, 1994; Wire, 2021). PTFE is released when non-stick surfaces such as Teflon overheat above 530º F (280º C). PTFE is a common respiratory toxin in birds, with the lung being the target organ of intoxication. Clinical signs are usually limited to sudden death, but depending on the degree of exposure may include somnolence, dyspnea, incoordination, weakness, respiratory distress, and seizures. Hemorrhage and congestion of the lungs are the main post-mortem findings (Dumonceaux & Harrison, 1994).

However, in the present case, there was no evidence of the presence of anything that suggested ingestion of heavy metals in the bird's gastrointestinal tract, such as traces of paint, objects with lead or zinc in their composition, toxic plants, or harmful foods. In addition, there is also no history of exposure to Teflon non-stick cookware, a common cause of sudden death in birds. Thus, having ruled out other possibilities of intoxication, it is suggested that the animal has potentially become intoxicated from the ingestion of white glue from the handmade nest. However, it cannot be disregarded that other causes may be linked to white glue intoxication since several potentially toxic products can culminate in the same clinical signs, and it is sometimes difficult to accurately determine the real cause of intoxication. However, considering white glue intoxication as the most likely hypothesis, we sought to understand and discuss the pathophysiological mechanisms by which intoxication may have occurred.

Polyvinyl Acetate (PVAc; CAS no. 9003-20-7), the main component of white glue (also called school glue or Elmer's glue) is a synthetic product manufactured by the polymerization of vinyl acetate monomers, using peroxide catalysts. The polymerization reaction is most widely carried out in aqueous solution, forming an emulsion that is white to off-white in color. In its most common application, PVAc serves as the film-forming ingredient in water-based (latex) paints and as an adhesive (Conner, 2001) for paper, wood, glass, metal, and porcelain. It also has a wide range of industrial, medical and food applications, for example in cosmetics; in the manufacture of paper and paperboard products which would come in contact with foods and as a base for chewing gums. In veterinary medicine, PVAc is used as an adhesive film former in antibiotic aerosol sprays for cattle treatments (Nair, 1992).

Although PVAc is described as a non-hazardous substance for humans and some animal species, there is a lack of information describing its toxicological effects in species other than mammals.

Data on the consequences of PVAc and white glue ingestion are rarely found in the literature and most of the available information is associated with bowel obstruction in men (Tanimu et al., 2019) and in some animal species, such as dogs (Lubich et al., 2004). In humans, PVAc poisoning is mostly described in glue sniffing; however, in these cases, damage is caused by inhaling glue additives, such as toluene and other solvents; not described to be present in the ingested white glue (Jain & Verma, 2016).

Experimental studies on PVAc ingestion toxicity were reported in a review by Nair (1992). A single dose of PVAc (25g/Kg), administered orally to unspecified strains of rats and mice was reported to induce lymphoid infiltration of the liver, depigmented epithelial cells of the renal tubules and a slight increase in the number of polynucleated cells in the spleen. Moreover, when PVAc (250mg/kg) was administered orally for 12 months to rats and mice, fluctuations in weight, changes in blood composition, changes in liver-to-body weight ratios and changes in cholinesterase and catalase activities were reported.

In the present case, intoxication was probably caused by the direct ingestion of the glue from the handmade nest. However, it is not possible to discard the inhalation of glue particles derived from nest pecking. The ingested glue did not have any other compounds, but PVAc and water in its chemical formulation. However, it is not possible to discard the presence of the non-mentioned impurities. Nair (1992) reports the presence of small amounts of arsenic, free acetic acid, heavy metals, lead and residual vinyl acetate in the PVAc used as a food chemical. Depending on whether the PVAc is supplied as a solid or as an emulsion, it may contain hardeners, plasticizers, emulsifiers, thickeners, biocides, pigments, and other additives used to impart desired characteristics in the final product. Although the quantity of additives and impurities presented have no described hazardous effects in humans, it cannot be affirmed that they will act in the same way in birds.

The most common differential diagnosis for acute onset of dyspnea in birds must include foreign body aspiration; inhaled respiratory toxins and acute infectious diseases (Hillyer et al., 1997). However, the gross necropsy and histopathological findings were very indicative of an acute toxicological event. Extensive parenchymal discoloration of the liver with markedly decreased consistency is frequently seen in acute toxic hepatosis (Maxie, 2015; Krishna, 2017). Moreover, diffuse alveolar hemorrhage can also be caused by the action of toxins (Park, 2013), as well as regurgitation, vomiting and gastric disorders (Hoefer, 1997).

In birds, polytetrafluoroethylene (PTFE), most commonly marketed under the trade name Teflon, is known to cause pulmonary hemorrhage and multifocal centrilobular hepatic necrosis after exposure to vapors from overheated nonstick cookware (Shuster et al., 2012) or ovens in self-cleaning mode (Hillyer et al., 1997). At temperatures above 280°C, PTFE-coated surfaces begin to emit degradation products in the form of particulates and gas. Subsequent inhalation of these products by birds can result in various clinical signs, including open-beak breathing, chirping, incoordination, lateral recumbency, convulsions, and death (Shuster et al., 2012).

The pet bird described in this report, was not exposed to PTFE nor to any other source of toxic substances. There was no evidence of traumatisms nor foreign body aspiration. There was no history of unusual environmental and management issues. Lastly, no similar problems were identified in the four other contacting birds.

Although the Material Safety Data Sheet on PVAc states that the substance contains no components considered to be persistent, bioaccumulative or toxic, it also informs that there are no available data on the PVAc toxicological effects, such as acute toxicity, specific target organ toxicity and aspiration hazard. In our opinion, and accordingly to Nair (1992), a lack of information about an ingredient is not sufficient to justify a determination of safety. Moreover, a review made by the National Research Council of The National Academies (2013) reported that although no data on lethality were found in humans after acute exposure to vinyl acetate, gross necropsy of rats, mice, guinea pigs and rabbits exposed to vinyl acetate vapor revealed pulmonary congestion and hemorrhage in the animals that died. The LC50 (lethal concentration, 50% lethality) was calculated to be 3,680 (2,660–5,100) ppm in rats; 1,460 (925 2,305) ppm in mice; 5,210 (3,500–7,740) ppm in guinea pigs and 2,760 (1,800–4,200) ppm in rabbits. Unfortunately, it was not possible to accurately determine the amount of the substance ingested by the animal.

It is known that PVAc can be degraded by hydrolase enzymes (Albuquerque et al., 2014), which also exist in the animal gastrointestinal tract. By the action of those enzymes, PVAc is broken down into vinyl acetate monomers (Gross &

Kalra, 2020), which, in turn, is metabolized, mainly via hydrolysis, by B-esterase enzymes, such as carboxylesterases and to a minor extent, cholinesterases. Vinyl acetate hydrolysis forms acetic acid and acetaldehyde (Fedtke & Wiegand, 1990), which, in short exposures, do not cause toxic effects in humans nor in some mammal species (Bogdanffy, 2002). B-esterase enzymes are also related to the biotransformation of other substances, such as insecticides (Parker & Goldstein, 2000), representing an important detoxification pathway to vinyl acetate and other xenobiotics, for instance carbamates and organophosphates (Fedtke & Wiegand, 1990; Thompson, 1993).

Birds are described to be more sensitive to pesticides than mammals. This characteristic is mainly associated with less quantity of detoxifying enzymes, such as carboxylesterases and cholinesterases (Parker & Goldstein, 2000). The expression of the carboxylesterase enzyme is ubiquitous, with high levels in the liver, small intestine, kidneys and lungs (Shibamoto & Bjeldanes, 2014). According to Thompson (1993), B-esterase enzymes, especially carboxylesterases, show variable expressions in different avian species. Moreover, birds with lower levels of these enzymes or with a lesser range of B-esterase forms are more sensitive to insecticide poisoning. The variability of carboxylesterase enzymes among birds is mainly related to species, eating habits and size. Carnivorous birds tend to have lower levels of B-esterase activity than omnivorous and herbivorous. Moreover, small birds have higher levels of plasmatic (Fossi et al., 1996) and lower levels of hepatic (Bush et al., 1973) B-esterases than larger birds. Lastly, some avian species, such as pheasants (Phasianus colchicus), have higher activity levels of B-esterases than other studied avian species, and are more resistant to pesticide poisoning (Thompson, 1993).

Considering that vinyl acetate, in its monomeric form, can cross cellular plasma membranes (Gross & Kalra, 2020) and be hydrolyzed very efficiently by rat and human liver (Fedtke & Wiegand, 1990), pesticides, such as carbamates and organophosphates, are metabolized by the same pathway as vinyl acetate. Moreover, as this enzymatic pathway is less efficient in birds, compared to mammals, it can be suggested that the amount of PVAc that was ingested by the parrot resulted in its intoxication, probably due to the failure of B-esterases to convert vinyl acetate into its lesser toxic metabolites. The gastrointestinal absorption of molecules and their systemic distribution to entire organisms should have caused the signs and symptoms showed.

No data about the characteristics of B-esterases in B. tirica, and other parrot species, were found in the literature. However, based on all the information above, it is suggested that B-esterases activity could be insufficient to protect this avian species from the action of potentially toxic products that are metabolized by the B-estarases pathway. Taken together with the probable high dose of PVAc exposition, this fact could have contributed to the saturation of the metabolic pathway of vinyl acetate as this condition is described in some pathological conditions (Simon et al., 1985), causing bird intoxication.

From what was presented, it is suggested that PVAc is a potentially toxic product for birds. Further studies are needed to better assess the influence of B-esterases on PVAc poisoning. However, this report serves as an alert to veterinarians and bird owners.

## 5. Conclusion

Although characteristics of avian anatomy predispose birds to be more sensitive to toxic substances, little is known about predisposing factors involved in avian sensitivity to toxic agents. Although Polyvinyl Acetate (PVAc) is not described as a toxic substance, there is no data available on its toxicological effects, and the lack of information about an ingredient is not enough to justify its safety, therefore, it is not possible to discard it as a possible cause of bird poisoning in the present case report.

PVAc monomers are degraded by B-esterase enzymes, such as carboxylesterases and cholinesterases, which are also related to the biotransformation of insecticides. These enzymes show variable expression in different avian species. Birds that

have lower levels of these enzymes or with a lesser range of B-esterase forms are more sensitive to insecticide poisoning, and could also be more sensitive to PVAc poisoning, degraded by the same enzymatic pathway.

No data were found on the characteristics of B-esterases in B. tirica and other parrot species. Therefore, further studies are needed on these enzymes in these species and their influence on possible intoxication by PVAc, the main component of white glue. In addition, further studies on the toxicological effects of PVAc are also needed.

Although it is difficult to accurately determine the real cause of the bird's intoxication, due to the absence of evidence of the presence of other possible toxicants, it is suggested that the bird was possibly intoxicated from the ingestion of white glue from the handcrafted nest. Thus, the present case report serves as a warning to veterinarians and bird tutors to be careful with the exposure of these animals to this product.

More studies are needed related to the toxic potential of PVAc for birds, considering, still, understanding the role of B-esterase enzymes in this possible intoxication, to reinforce the hypothesis established here and evidence a possible susceptibility of birds to this product.

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