

BRIEF COMMUNICATIONS

Cantharidin poisoning of emu chicks by ingestion of *Pyrota insulata*

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In April and May 1992, the Texas Veterinary Medical Diagnostic Laboratory (TVMDL) received for necropsy several frozen 1-3-month-old emu chicks from 1 ranching operation. Insects had swarmed to blooms on mesquite trees (*Prosopis glandulosa*) near the chick barn, and when a cool front arrived, lights were left on inside to encourage the chicks to seek shelter. As dusk fell, the insects were attracted to the lighted area, and they could be seen covering the floor of the barn and other surfaces accessible to the chicks. The history accompanying the birds described the chicks' avid consumption of the insects. Some chicks remained clinically normal while others became ataxic, vomited beetles, became prostrate, and died. Some survived by vigorous treatment with oral fluids. Submissions to TVMDL consisted of proventricular contents, the frozen carcasses of 4 emu chicks, and a sample of the suspect insects.

At necropsy, the esophagus of each bird was congested and edematous, and the proventricular serosa was hemorrhagic. The gizzard linings were sloughed, the intestinal contents were pink and the livers were pale. Tissue samples for histopathologic evaluation were fixed in 10% neutral buffered formalin, processed, embedded, sectioned and stained with hematoxylin and eosin (HE) prior to microscopic examination. Ingesta were examined under a dissecting microscope. Only 1 of the birds had a large volume of intact ingesta, which included fibrous plant material, small pebbles and 1 beetle about 1 cm long. Insect fragments were found in the proventricular and gizzard contents of 2 of the other chicks.

Microscopically, the lesions varied in severity within each animal (Fig. 1). In some chicks, areas of the esophageal mucosa were overlaid with a thick coat of mucus (Fig. 1b). In others, there was multifocal epithelial ulceration with diffuse submucosal necrosis and thrombosis of small submucosal blood vessels (Fig. 1c-e). Catarrhal esophagitis and multifocal esophageal ulceration with underlying inflammation and necrosis were suggestive of exposure to a chemical irritant in a solid form, which was consistent with ingestion of a blister beetle, as opposed to a liquid irritant, which would have produced a more uniform lesion.

Ingesta and insects were extracted and analyzed for can-

tharidin by previously developed methods⁸ except that the insect sample was oven dried and extracted into double its mass instead of an equal mass of 1 N NaOH. Because of the scant sample volume and because the insect bore only slight resemblance to blister beetles previously seen in this laboratory, the samples were combined for cantharidin extraction. The ingesta pooled from 1 pair of birds contained 37.1 ppm cantharidin, and that from the second pair contained 0.793 ppm (wet weight basis). Extraction of 6 beetles of various wing cover coloration patterns gave 34,000 ppm cantharidin on a dry weight basis for the combined sample.

The captured sample of the suspect insects was forwarded to the Texas Agricultural Extension Service Entomology Group for identification. This sample included insects with a wide variation in wing cover patterns, all in black and gold (Fig. 2). The beetles were identified as *Pyrota insulata*, a blister beetle commonly found throughout Texas and Oklahoma, whose main food source is the blooms of mesquite trees.⁵

Cantharidin is a naturally produced vesicant found in the hemolymph of some members of the insect family Meloidae, commonly known as blister beetles. Cantharidin is colorless, odorless, and only slightly soluble in water.¹⁰ Cantharidin has a natural receptor, the membrane protein phosphatase 2A, that is expressed on the surfaces of a wide variety of cell types; binding of cantharidin to this protein is the first step in the acantholytic series of events.²⁻⁴ In many blister beetle species, the male carries all of the cantharidin, and the female receives the protective toxicant only as mating occurs.¹

Horses with blister beetle poisoning may exhibit anorexia, depression, shock, oral ulcerations, colic, diarrhea, hypocalcemia, cardiac arrhythmias, stranguria and hematuria, or sudden death.^{9,10} At necropsy, exposed horses have blistered and hemorrhagic gastrointestinal epithelium, pale and inflamed kidneys, and hemorrhagic urogenital tract epithelia. Microscopically, the mucosae are blistered and necrotic, with submucosal edema and hemorrhage.¹⁰ Human cantharidin intoxications (Spanish fly) result in profuse vomiting of bloody fluid, tachycardia, fever, and dehydration. Autopsy reveals the most extreme lesions in the lower esophagus, with secondary hematuria and renal damage.⁶ Cantharidin intoxications have also occurred in sheep, cattle, goats, dogs, cats, rabbits, and rats.⁹ To date, the only report of cantharidiasis in birds has been in chickens.⁷

The clinical history received with the emu chicks was consistent with clinical signs observed in other cantharidin-poisoned species.^{6,7,9} The cantharidin content of 34,000 ppm on a dry weight basis is within the range (<100 to >50,000 ppm)⁹ of that of other blister beetle species tested. Regur-

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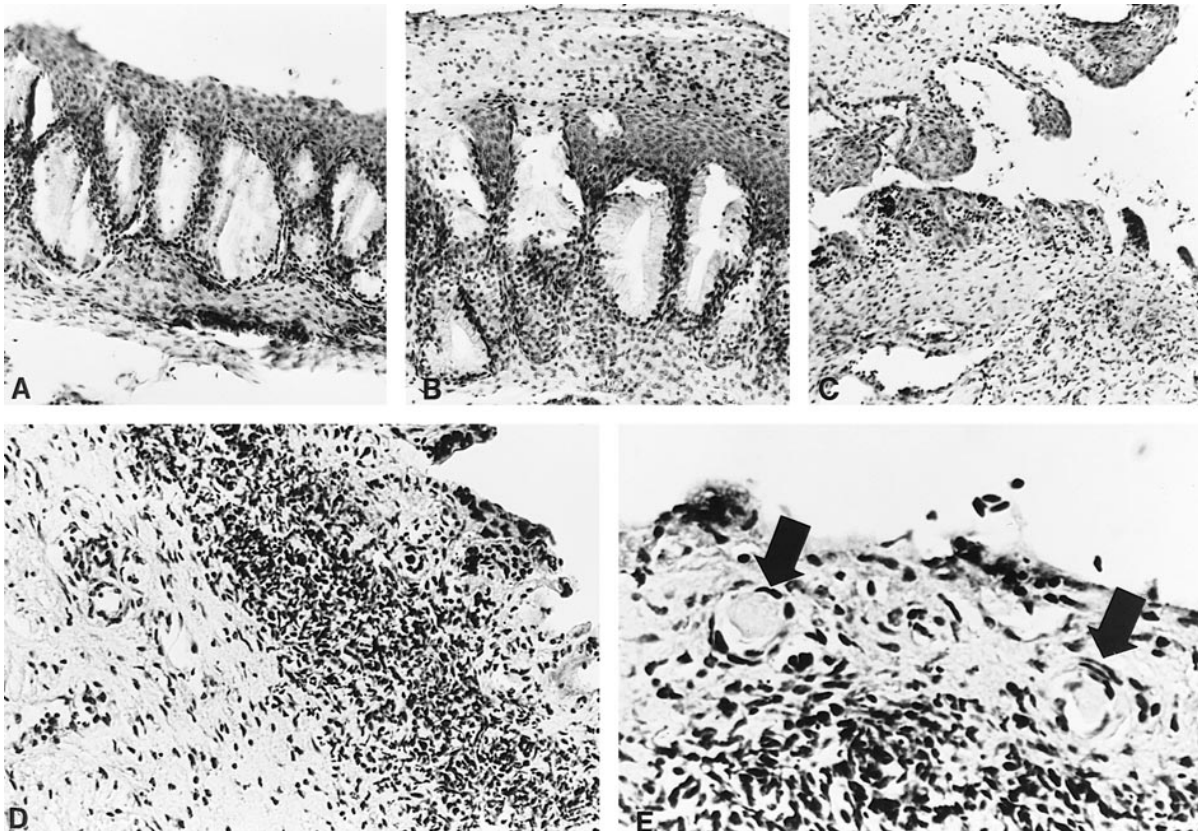


Figure 1. Esophageal lesions from emu chicks poisoned by ingestion of the blister beetle *Pyrota insulata*. HE. **a.** Normal frozen esophagus. Note the torn submucosa, probably a freezing artifact. **b.** Catarrhal esophagitis. Note the thick layer of mucus and cellular debris coating the mucosal surface. **c.** One mucosal surface still carries some stratified squamous epithelium, although the mucous glands are exposed and the opposite face is ulcerated. **d.** Coagulative necrosis, hemorrhage, and inflammation of the submucosa. **e.** Thrombi (arrows) of the submucosal microvasculature. Classic acantholytic lesions were indistinguishable from freezing artifact.

gitation of the beetles may have contributed to the formation of the severe lesions in the esophagus but probably prevented renal and urogenital tract lesions. The variability of reactions in the chicks seen following consumption of the beetles may be attributed to the life cycle of the beetles; the female beetles contain little or no cantharidin prior to mating. Because of the small number of beetles submitted, correlations between cantharidin content and either wing cover pattern or sex of individual beetles could not be determined.

Pyrota insulata has not been implicated as an agent of cantharidin toxicosis in horses or ruminants. Its affinity for

mesquite blooms⁵ rather than lower vegetation prevents this species from being overconsumed by grazing animals. The critical circumstance to the poisoning of these emu chicks was the open chick barn kept lighted after dark.

This report details the first diagnosis of cantharidin intoxication in emu chicks and the first known poisoning by *Pyrota insulata*. No other cases of cantharidin toxicosis in raptives have been received at this laboratory.

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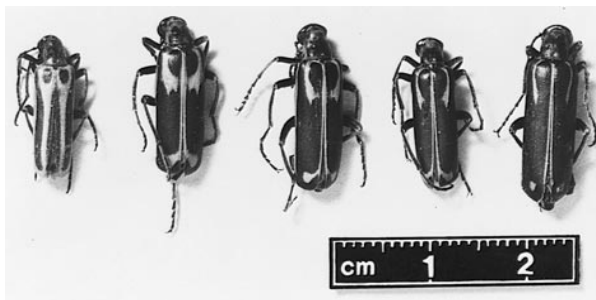


Figure 2. *Pyrota insulata*. Several variations occur in the wing cover patterns of this insect, all in black and gold.

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Pulmonary metastasis of a feline vaccination-site fibrosarcoma

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Recently, chronic inflammatory lesions and sarcomas have been described at sites of previous vaccination in cats. In lesions of both types, macrophages that contain a gray-brown, granular to crystalline foreign material have been found.^{1,3,4,6,9} Aluminum was identified in macrophages from the sarcomas by electron probe x-ray microanalysis.⁶ Epidemiologic studies have confirmed the association between sarcoma development and sites commonly used for vaccination of cats.^{6,8} It has been hypothesized that chronic inflammatory and immunologic reactions to vaccine components predispose some cats to a derangement of their fibrous connective tissue repair response that eventually results in neoplasia.⁶ Vaccination-site sarcomas are locally aggressive and have a high rate of local recurrence,^{1,2,6,7,10} but histopathologically confirmed metastasis has been reported only twice.^{1,10} In this report, we describe pulmonary metastasis of a subcutaneous vaccination-site fibrosarcoma.

A 5.5-year-old neutered male domestic longhair cat owned by 1 of the authors (LM) developed a 5- × 2- × 1-cm freely movable subcutaneous mass in the interscapular space 25 months after a killed rabies vaccine was injected in the same site. No other vaccines had been administered in this location. The mass was surgically excised, fixed in neutral buffered formalin, and processed routinely for histopathologic examination.

Histologically, the mass was composed of spindle-shaped cells arranged in long interlacing streams and bundles (Fig. 1). Neoplastic cells had indistinct cytoplasmic borders and moderate amounts of fibrillar, lightly eosinophilic cytoplasm. Nuclei differed slightly in size, were oval to fusiform, and had fine chromatin and 1–3 magenta nucleoli. Mitotic figures were common. The neoplastic cells were often surrounded by an eosinophilic fibrillar matrix that stained blue with Masson's trichrome stain and was interpreted to be collagen. Small arteries and veins at the periphery of the neoplasm were surrounded by small to moderate numbers of lymphocytes and fewer plasma cells. Small numbers of macro-

phages that contained a gray, granular to crystalline material were also scattered at the margins of the neoplasm (Fig. 2). The histopathologic diagnosis was vaccination-site fibrosarcoma.

One month after the surgery, multiple smaller masses were noted in the dermis and subcutis at the surgical site. Three radiation treatments (2 orthoradiation and 1 topical probe application of Strontium-90) and 3 additional surgical excisions were performed over the next 2 years. Local recurrence followed each treatment. Twenty-seven months after the initial surgery, euthanasia was performed.

At necropsy, several masses were present. The largest mass was 10 × 6 × 3 cm and involved the skin and subcutis just cranial to the interscapular space and to the right of the midline. This mass was extensively ulcerated and necrotic. Smaller subcutaneous masses were present within the interscapular space and firmly adhered to the right scapula. Other masses craniolateral and caudal to the scapula were firmly attached to deep connective tissue and muscle. A single 2- × 1- × 1-cm firm, cream-colored mass was present in the center of the right middle lung lobe. Tissues were placed in formalin and processed routinely for histopathologic examination.

Histologically, masses in the interscapular space and around the right scapula were highly invasive. Extensive areas of ulceration and necrosis were present. Cellular morphology was similar to that of the initial biopsy sample. Peripheral lymphoid aggregates and scattered macrophages were present, but the macrophages did not contain gray material.

The pulmonary mass (Fig. 3) was circumscribed and expansile. Its histologic features were generally similar to those of the primary neoplasm and the other tumors from the scapular area except that many of the neoplastic cells had larger nuclei and nucleoli; several abnormal mitotic figures were also present. Neoplastic cells filled the lumina of some pulmonary arteries. Lymphoid aggregates were seen peripherally around small arteries and veins. The pulmonary mass was interpreted as metastatic fibrosarcoma.

The location and histopathologic findings in the initial biopsy specimen are consistent with those previously de-

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