external wrap.

REFERENCES

Diffuse Alveolar Hemorrhage Secondary to Superwarfarin Ingestion*

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A 27-year-old woman with severe vitamin K deficiency presented with hemoptysis and diffuse pulmonary infiltrates. She rapidly developed respiratory failure requiring ventilatory support. Sustained ingestion of brodifacoum, a long-acting warfarin derivative, was ultimately found to be the cause of her coagulopathy and DAH.

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APPT = activated partial thromboplastin time; DAH = diffuse alveolar hemorrhage; JVD = jugular venous distention

A new class of rodenticides with powerful anticoagulant effects has been developed. These derivatives of warfarin have been called "superwarfarins" and exhibit enhanced potency and prolonged duration of action. Hemorrhagic complications of superwarfarins recently have been presented; however, no cases of pulmonary hemorrhage have been reported.1,2 We present a case of brodifacoum (D-Con) ingestion leading to prolonged, severe coagulation abnormalities and DAH. This case also illustrates the difficulty that can arise in diagnosing surreptitious superwarfarin ingestion.

CASE REPORT

A 27-year-old Hispanic woman, a ½ pack-per-day cigarette smoker, presented with fever, dyspnea and cough productive of ½ cup of blood. She denied chest pain, prior hemoptysis, or ingestion of aspirin, anticoagulants or illegal drugs.

Seven months previously she developed profuse vaginal bleeding and a coagulopathy was discovered. Factor analysis showed levels of factor II, 3 percent; factor VII, 5 percent; factor IX, 2 percent; and factor X, 1 percent. Severe vitamin K deficiency was found to be the cause; however, the etiology of her vitamin K deficiency could not be established. She had a rapid but short-lived response to vitamin K1 and required 40 mg intravenously three times a day for control of her coagulopathy. In subsequent months she suffered a thrombotic stroke, epistaxis and soft tissue hematomas. She did not take her vitamin K, in the five days prior to the current admission.

On examination she was an ill-appearing woman with a respiratory rate of 50 breaths per minute, blood pressure of 140/90 mm Hg, a heart rate of 150 beats per minute and a temperature of 40°C. There were no skin lesions, JVD, third heart sound or edema. Lung examination showed bilateral basilar crackles.

Laboratory values revealed a hemoglobin value of 10.1 mg/dL, WBC, 16.4/cu mm with 89 percent neutrophils and 8 percent band cells. The platelet count was 389,000/cu mm. Her PT was 84 s (control of 12 s), and her APTT was 65 s (control of 28 s). Arterial blood gas values, while breathing 50 percent O2 by face mask, showed a pH of 7.43; PaO2 of 27 mm Hg; PaCO2 of 59 mm Hg. A chest x-ray showed diffuse alveolar infiltrates (Fig 1).

She continued to have hemoptysis and rapidly developed respiratory failure requiring intubation. Large amounts of blood were suctioned from the endotracheal tube. Postintubation ABG values while breathing 60 percent O2 were pH, 7.42; PaCO2, 36 mm Hg; and PaO2, 60 mm Hg. She was treated with fresh frozen plasma and 60 mg of vitamin K1 intravenously. Her PT returned to 15.8 s (control of 12.9 s) within 30 min.

She required ventilatory support with 15 cm H2O of PEEP and an FiO2 of 0.6 for ten days. Her coagulopathy was controlled with 40 mg of vitamin K1, given intravenously three times a day. She had no further episodes of bleeding. She was extubated on her 13th hospital day and discharged nine days later while receiving 40 mg of vitamin K1, SQ three times a day. Her room air ABG prior to discharge showed a pH of 7.48; PaO2, 31 mm Hg; and PaCO2, 67 mm Hg.

Subsequently, she admitted ingesting D-Con rat poison, containing brodifacoum, because of depression. Blood samples showed high levels of brodifacoum. She is currently undergoing psychiatric treatment.

DISCUSSION

Diffuse alveolar hemorrhage is characterized by wide-

![Figure 1. Admission chest x-ray showing bilateral alveolar infiltrates.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21656/ on 03/31/2017)
spread hemorrhage from the microvasculature of the lung and often presents with the clinical triad of hemoptysis, anemia and diffuse alveolar consolidation. It is caused by diseases that damage the alveolar-capillary barrier or by disorders of coagulation. Both heparin and warfarin anticoagulation have been reported as etiologies of DAH; however, the incidence of DAH in anticoagulated patients is low and represents a small proportion of bleeding complications. 

The superwarfarins are a recently developed group of rodenticides. Their method of action is identical to warfarin, with blockage of vitamin K-dependent gamma-carboxylation of precursor factor proteins. Superwarfarins have an extremely long duration of action with anticoagulation persisting for weeks to months. Vitamin K (phyloquinone) in very large doses often is required to control coagulation.

The Association of Poison Control Centers reported 4,382 cases of superwarfarin ingestion in 1987. Most of these cases represent accidental ingestion by children with no serious effects. Ingestion by adults, however, often is intentional and of large amounts with an increase in adverse effects. In addition, ingestion in adults can be surreptitious and repeated over an extended period of time. It is likely that the patient in this case ingested brodifacoum on several occasions.

The long duration of action of these compounds combined with their easy accessibility make it probable that pulmonary complications will be increasingly encountered. As this case demonstrates, the diagnosis can be difficult. Superwarfarin ingestion should be suspected in any patient not on coumadin with bleeding and abnormalities in the vitamin K-dependent coagulation factors. An assay for brodifacoum in blood is now available and may facilitate diagnosis in difficult cases.

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