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Oak leaf (*Quercus pyrenaica*) poisoning in cattle

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ABSTRACT

Three experiments were conducted to study the clinical and pathological findings associated with poisoning in cattle due to ingestion of young oak leaves (OL) and the main factors responsible for toxicosis. In Experiment 1, six 1.4 year-old bulls were fed up to 5 kg of young OL per animal per day and showed no signs of toxicity, apart from a slight proteinuria. In Experiment 2, another six 1.4 year-old bulls were first subjected to severe feed restriction for eight days and then fed a higher amount of OL (approx. 10 kg) daily. A marked increase of serum creatinine and blood urea (BUN) was detected in urine as well as clinical signs consistent with renal failure. At necropsy, animals showed gastrointestinal ulcers and kidney tubular necrosis. Since these results suggested a crucial role of the feed restricting period, a third experiment was conducted administering the same amount of young OL as in Experiment 1, but adding the severe feed restricting period as in Experiment 2. There was a wide variation in clinical signs, with one bull showing clinical signs and lesions, another recovering after showing mild clinical signs and high levels of creatinine and BUN, and the third appearing clinically normal. The relevance of restriction access to food in the development of OL toxicosis appears to be critical because the intoxication was only elicited when the OL administration was preceded by a severe feed restricting period.

1. Introduction

Poisoning of ruminants, mainly cattle, by the consumption of oak (*Quercus* spp.) leaves (OL) and shrubs occurs worldwide (Cedervall et al., 1973; Pixon et al., 1979; Spier et al., 1987; Pizarro et al., 1992; Yeruham et al., 1998; Radostits et al., 2007). The toxicity of oak leaves is due to the presence of hydrolysable tannins, although the toxic principles have not been fully established (Newman et al., 2007; Radostits et al., 2007).

There are many different species and varieties of oaks and their leaves are used as animal feed in several countries such as India, especially when other green fodder is not available (Kumar and Vaithiyanathan, 1990; Makkar, 2003). However, they are not commonly used in Europe or USA, since intoxications commonly occur due to the consumption of young OL, considered more toxic than mature ones (Jones et al., 1997). The consumption of OL as a small part of the diet is not associated with illness (Radostits et al., 2007). It is assumed that animals can fed OL when the availability of other feed resources is scarce (Spier et al., 1987; Yeruham et al., 1998) but the precise conditions that predispose to the development of oak toxicosis are not clear.

In Spain, oaks are present in wide areas of the country where cattle graze and several cases of oak poisoning have been reported resulting in the deaths of many animals (Pizarro et al., 1992; Mazzuchelli et al., 2000; Frutos et al., 2005). Affected cattle show diarrhoea or constipation with bloody or mucoid faeces together with clinical signs related to uremia, as severe acute renal failure with tubular necrosis is the main consequence of oak consumption (Radostits et al., 2007; Grant Maxie and Newman, 2007; Guitart et al., 2010). Most of the reports describe advanced clinical cases in which animals have been eating OL for several days prior to the onset of clinical signs and the collection of samples for diagnostic investigation (Cedervall et al., 1973; Neser et al., 1982; Holliman, 1985; Spier et al., 1987; Yeruham et al., 1998; Mazzuchelli et al., 2000). As such, there is a lack of information of the initial clinical signs or the clinical pathology findings that occur at the early stages of exposure to OL in the diet. This is related to the difficulty of reproducing experimentally oak toxicosis in cattle, as shown in a previous attempt where oral administration of commercial tannic acid resulted in a methaemoglobinemia rather than renal disease in calves (Plumlee et al., 1998).

Three experiments were conducted with two aims: to experimentally reproduce OL toxicosis and to study the clinical signs, clinical pathology and pathological findings associated with young OL poisoning under controlled conditions. In the Experiment 1, young bulls received a low (2.5 kg on a fresh matter (FM) basis, per animal per day) or high amount of OL (up to approx. 5 kg

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FM) following an adaptation period in which a limited amount of feed was offered. Since none of the bulls showed signs of toxicity, Experiment 2 was planned to take into account that the animals may need to have suffered feed restriction, or fed larger amounts of young OL. Animals in Experiment 2 showed intoxication, so a third experiment was undertaken to see if toxicity could be induced by feeding the same amount of young OL as in Experiment 1, but after feed restriction.

2. Materials and methods

Three experiments were performed in accordance with Spanish Royal Decree 1201/2005 for the protection of animals used for experimental and other scientific purposes. The general schedule of the experiments is showed in Fig. 1.

2.1. Oak leaves

Very young oak (*Quercus pyrenaica*) leaves (less than 2 week-old) were manually collected from saplings during the spring on a country estate in northwest Spain (Sahechores, León) at a mean altitude of about 900 m and immediately stored at −30 °C until the experiments were performed. As described by Doce et al. (2009), the total tannin content of young OL (determined using the Folin–Ciocalteu method in combination with polyvinyl–polypyrrolidone) was 230 g of tannic acid equivalents/kg dry matter (DM), and the content of hydrolysable tannins (analyzed by ion-pair high-performance liquid chromatography) was 11 and 19 g/kg DM as gallic acid and ellagic acid, respectively.

2.2. Experiment 1

2.2.1. Animals and diets

Six Brown Swiss bulls (approx. 1.4 years old, 534 ± 29.6 kg live weight (LW) at the beginning of the experiment), each equipped with a ruminal cannula of 10-cm internal diameter (Ankom Technology Corp., Macedon, NY, USA), put in place 2 months earlier, were individually penned and divided into three groups of two animals, balanced for weight: control, L-OL and H-OL. All animals were fed a limited amount of grass hay (average 5 kg per animal per day) for a 14-day adaptation period. Then, the control animals continued to receive the grass hay, while those on treatment L-OL received daily 14 g DM of grass hay plus 7 g DM of OL/kg metabolic weight ($LW^{0.75}$) (on average 1.7 and 2.5 kg FM of hay and OL, respectively) and those on treatment H-OL received 14 g DM of grass hay plus 14 g DM of OL/kg $LW^{0.75}$ (on average 1.8 and 5.2 kg FM of hay and OL). The OL, defrosted at about 4–10 °C and slightly chopped, were administered twice per day (at 08.30 and 20.00 h approx.) through the rumen cannula to ensure that all animals received the established amount. Treatments lasted for 14 days and afterwards all animals received again the same amount of hay offered during the adaptation period for 14 more days (see Fig. 1).

2.2.2. Clinical assessment

Animals were monitored daily for clinical signs of illness (e.g., demenour, appetite, rectal temperature, cardiac and respiratory rate, ruminal movements, and faecal and urine appearance).

2.2.3. Haematology, and serum biochemistry and urine analysis

Blood samples were collected from the caudal tail vein into 10 mL vacutainer tubes (Venoject®, Terumo Europe N.V., Leuven, Belgium) with EDTA for haematological studies and without anticoagulant for serum biochemistry determinations, allowed to clot and the serum stored at −20 °C. Samples were taken immediately prior to the morning feed on days 0 (just before the commencement of the OL administration), 3, 6, 9, 12, 15, 18, 21 and 28 of the experiment.

Complete blood counts were determined with an automatic cell counter (Abacus Junior Vet, Diatron, Viena, Austria). Serum samples were used to determine (using the Autoanalyser 704, Hitachi, Tokyo, Japan) total proteins (g/dL), albumin (g/dL), total and direct bilirubin (mg/dL), glucose (mg/dL), alanine aminotransferase (ALT; U/L), aspartate aminotransferase (AST; U/L), glutamyltransferase (GGT; U/L), alkaline phosphatase (ALP; U/L), blood urea nitrogen (BUN; mg/dL), creatinine (mg/dL), phosphorus (mg/dL), calcium

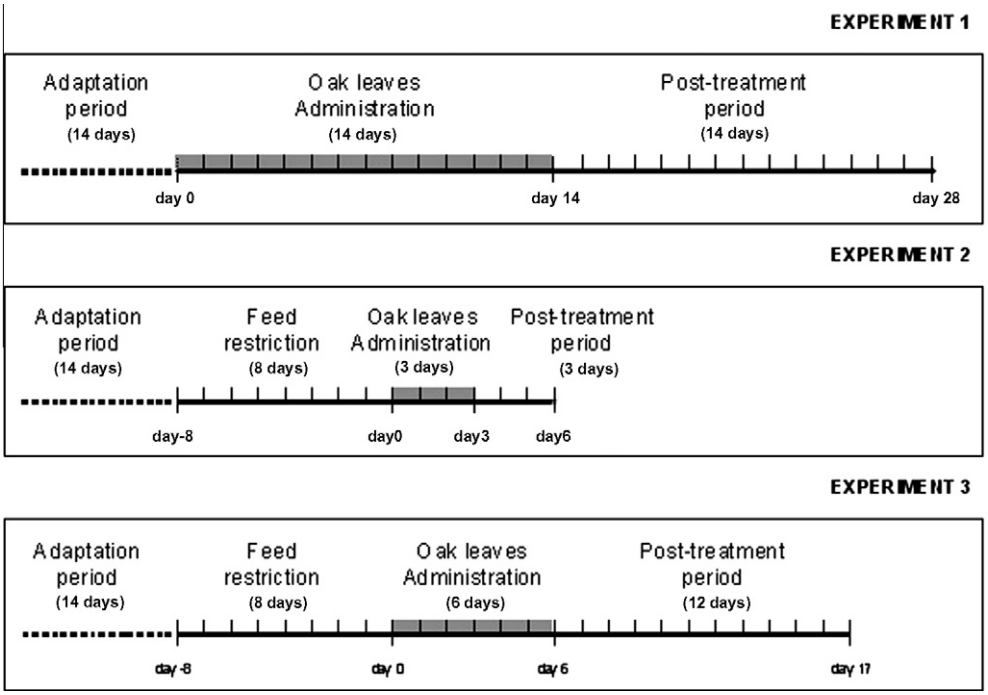

Fig. 1. Diagram showing the experimental schedules.