AN OVINE HEPATOTOXICOSIS CAUSED BY THE PLANT HERTIA PALLENS (DC.) KUNTZE (ASTERACEAE)

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ABSTRACT


A field outbreak of Hertia pallens poisoning in sheep is described. The hepatotoxicity of the plant was experimentally demonstrated in 7 sheep which developed lesions that ranged from a diffuse degeneration to centrilobular necrosis. These lesions occasionally extended to the midzonal area of the lobules. In addition to a lung oedema, a diffuse mononuclear interstitial pneumonia was present in 3 of the sheep. Botanical, clinical and pathological data are given.

INTRODUCTION

Information on the toxicity of Hertia pallens is limited. The only report on this intoxication is that by Steyn (1934), who described the clinical signs and gross lesions in a sheep dosed with the plant.

The purpose of this report is to describe in more detail the clinical signs, pathology and chemical pathology of sheep that died during a field outbreak of H. pallens poisoning and of sheep experimentally poisoned with the plant.

FIELD OUTBREAK

A farmer in the Douglas district in the northern Cape Province, lost 92 Persian sheep out of a flock of 258 from December to February 1980/81. The veld was in poor condition and the farmer had been advised to reduce the size of the camps to prevent selective grazing.

Clinically affected animals were anorectic and apathetic and although a few survived for up to 5 d others died within 12 h. Gross lesions in four 3-month-old lambs necropsied on the farm included a lung oedema, ascites and widespread ecchymotic and petechial haemorrhages in the mediastinum, coronary grooves and connective tissue surrounding the trachea and oesophagus.

The livers were slightly enlarged and pale, and most of the animals were icteric. Microscopically, the hepatic lesions in 2 animals ranged from bridging centrilobular necrosis, which occasionally extended to the midzonal area of the lobules, and congestion, to diffuse hepatocellular degeneration (cloudy swelling, hydropic degeneration and fatty metamorphosis), in the others. Single necrotic hepatocytes were scattered throughout the parenchyma and the Kupffer cells were activated (proliferation and hypertrophy). These changes were accompanied by a mild bile ductular proliferation. Apart from a lung oedema in 2 animals, no other noteworthy lesions were present.

Blood samples were randomly collected in sterile vacuum tubes from 9 animals in the flock. The level of gamma glutamyl transpeptidase (y-GT) activity in 3 samples was mildly elevated.

Large numbers H. pallens plants were identified on the farm.

DESCRIPTION, DISTRIBUTION AND ECOCLOGY OF THE PLANT

Family: Asteraceae (Compositae)
Name: Hertia pallens (DC.) Kuntze
Common Names: Malkopharpuis, Springbokbossie (Smith, 1966)

Description: (Fig. 1 & 2) Glabrous, rigid, bushy shrub up to 1 m high; branches terete, numerous, erect, with many short branchlets, bark pale, papery. Leaves alternate, sessile, with a decurrent line from each side of the base, linear-oblong, up to 25 mm long and 4 mm broad, slightly concave, coriaceous, glaucous, smooth, subobtuse, nerveless, almost entire except for a few minute

FIG. 1-2 H. pallens
AN OVINE HEPATOTOXICOSIS CAUSED BY THE PLANT HERTIA PALLENS

Cape Province: Gordonia, Postmasburg, Taung, Kimberley, Herbert, Prieska, Phillipstown, De Aar, Hanover, Colesberg, Aliwal North, Victoria West, Middelburg, Cradock, Tarka, Queenstown, Glen Grey

Ecology: *H. pallen* occurs on calcareous soil, sand, loam, shale and white quartzite. It grows on hillsides, rocky ridges, in dry river beds, on flats, plains, in low lying places and along roadsides. It is common in grassveld and especially in degraded and mismanaged secondary grassveld; it can become a spreading weed and is often found as a pioneer on denuded soils and in eroded, open veld. It is recorded from 300–1 500 m above sea-level. The plants are only browsed by stock in times of drought and according to Acocks (1975), *H. pallen* is an undesirable plant which should be reduced in number by appropriate veld management. He lists *H. pallen* in 2 of his veld types, namely, No. 16, Kalahari Thornveld, where it is of general occurrence in the grassveld constituent of the Central form; and No. 17, Kalahari Thornveld invaded by Karoo, where *H. pallen* becomes common on sandy, calcareous tufa.

**MATERIALS AND METHODS**

**Dosing trials**

Nine Merino sheep were dosed per stomach tube with milled, fresh green and dry plant material at the dosage levels and time intervals as outlined in Table 1.

The sheep were examined daily, and the following routine chemical pathological determinations were done on their sera collected daily in sterile vacuum tubes: y-GT, aspartate aminotransferase (AST), total bilirubin, urea nitrogen, red blood cell volume and haemoglobin.

Specimens of various organs were collected in 10% buffered formalin at necropsy, routinely processed and stained with haematoxylin and eosin. Additional staining techniques applied to various liver sections included Hall's bile stain, Oil red O for lipids and Masson's trichrome stain for collagen (Anon, 1968).

**RESULTS**

The findings are summarized in Table 1.

**Clinical signs:** Seven of the 9 experimental sheep became intoxicated. The clinical signs included apathy, anorexia, icterus, reduction of ruminal motility or atony, tymphany, dyspnoea, cyanosis and sudden death.

Some animals were affected intermittently over a period of weeks (Sheep 3, 6 & 9) others were sick for only a day or 2 (Sheep 1 & 2) or had to be destroyed within hours of the first evidence of clinical signs (Sheep 5). In many cases death appeared to result from asphyxia.

**Chemical pathology**

The activities of y-GT (79–120 μg/l) and/or AST (156–380 μg/l) were elevated in the sera of some of the sheep (Sheep 5, 6, 7 & 9), and 1 sheep (Sheep 5) had a bilirubinaemia (3.1 mg/100 ml).

**Pathology**

The livers were slightly enlarged and pale (Fig. 4) in all the animals, and 2 sheep were slightly icteric. Lung oedema was evident in 4 sheep, and a mild nephrosis was present in most animals (Table 1).

The most constant microscopical lesion in all the sheep was diffuse hepatocellular degeneration (cloudy swelling, hydropic degeneration and mild to severe fatty...
metamorphosis), interspersed with single necrotic hepatocytes, some of which were infiltrated by neutrophils (Fig. 5). Less common lesions included mild bile ductular proliferation, oedema of the portal triads and centrilobular coagulative necrosis which occasionally extended to the midzonal area of the lobules (Fig. 6).

Apart from the lung oedema, a mild to moderate diffuse mononuclear interstitial pneumonia with epithelial hyperplasia especially of the smaller bronchi and bronchioli, was present in 3 animals (Fig. 7 & 8). In 5 animals the kidneys were affected by cloudy swelling and hydropic degeneration of the epithelial cells in the proximal and distal convoluted tubules. Protein casts were visible in lumens of these tubules.

DISCUSSION

Overgrazing often forces animals to graze non-selectively and thus to consume poisonous plants which are usually unpalatable. The reduction of the camp sizes to induce non-selective grazing was most probably the reason why H. pallescens poisoning occurred in that particular camp and not on neighbouring farms where non-selective grazing was not enforced.

H. pallescens is a hepatotoxic plant in South Africa which hitherto has received little attention. Steyn (1934) reported severe dyspnoea and cyanosis in a sheep that received 600 g of dried plant material. The animal died 16 h later. Gross lesions included severe hydrothorax, lung oedema and hepatic fatty metamorphosis.

In both the experimental and field cases a spectrum of hepatic lesions was present. While diffuse degeneration occurred in the majority of the animals, centrilobular necrosis, which occasionally extended to the midzonal area of the lobules, was seen in 2 experimental and 2 field cases. Both the degenerative and necrotic changes were often accompanied by a mild bile ductular proliferation.

Similar findings were reported in sheep poisoned with Athanasia trifurcata and Asaemia axillaris (Coetzer & Bergh, 1983; Kellerman, Coetzer, Schneider & Welman, 1983). In these reports it was emphasized that the diagnostic value of zonal necrosis should be viewed with caution.

According to Steyn (1949), H. pallescens poisoning in sheep is often misdiagnosed as ketosis. In the light of the fatty changes induced in the experimental animals this is not surprising.

Although icterus was seen in a few animals that died during the field outbreak, and in 2 experimental cases, cholestasis was not histologically detectable.

The presence of a lung oedema both in experimental animals and sheep that died in the field outbreak suggests that in H. pallescens poisoning the insult is not confined to the hepatocytes but also includes the lungs. In our experiments, the pulmonary lesions ranged from an alveolar oedema to a diffuse mononuclear interstitial pneumonia with epithelial hyperplasia of especially the smaller bronchi and bronchioli. Clinically, the animals experienced respiratory distress and were cyanotic, and hence it is reasonable to assume that in this intoxication the lung lesions can be a primary cause of death.

Few conclusions could be drawn about the effect of length and temperature of storage on toxicity of H. pallescens. Sheep, however, appeared to vary greatly in individual susceptibility to intoxication.
**TABLE 1 Dosing regimen, clinical signs and pathology of sheep dosed with *H. pallens***

<table>
<thead>
<tr>
<th>No.</th>
<th>Mass (kg)</th>
<th>Sex</th>
<th>Age</th>
<th>Source</th>
<th>State</th>
<th>Approximate temperature of storage °C</th>
<th>Approximate length of storage (days)</th>
<th>Dose (g/kg×n)</th>
<th>Period dosed</th>
<th>Duration of experiment (days)</th>
<th>Fate</th>
<th>Clinical signs</th>
<th>Clinical pathology</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>24</td>
<td>M</td>
<td>MT</td>
<td>Douglas 1</td>
<td>Green</td>
<td>-10</td>
<td>14</td>
<td>5×2</td>
<td>0–1</td>
<td>2</td>
<td>Died</td>
<td>Tympany, depression, dyspnoea, stands with front legs apart and head down, foam at mouth, cyanosis. Died within 18 h of first clinical signs</td>
<td>Not available</td>
<td>Hydrothorax, hydropericardium and cyanosis. <strong>Liver:</strong> Centrilobular to midzonal necrosis, bile ductular proliferation and Kupffer cell activation. <strong>Kidneys:</strong> Nephrosis. <strong>GIT:</strong> Stasis</td>
</tr>
<tr>
<td>2</td>
<td>44</td>
<td>M</td>
<td>2T</td>
<td>Douglas 1</td>
<td>Green</td>
<td>-10</td>
<td>27</td>
<td>2,5×2</td>
<td>0–2</td>
<td>4</td>
<td>Killed</td>
<td>Mild tympany, dyspnoea, reduced ruminal movements, anorexia. Destroyed 48 h after beginning of clinical signs</td>
<td>No notable changes</td>
<td>Liver: Diffuse degeneration, mild fatty metamorphosis and Kupffer cell activation. <strong>Lungs:</strong> Oedema, mononuclear interstitial pneumonia with bronchi and bronchiolar epithelial hyperplasia. <strong>Kidneys:</strong> Nephrosis</td>
</tr>
<tr>
<td>3</td>
<td>41</td>
<td>F</td>
<td>FM</td>
<td>Douglas 1</td>
<td>Green</td>
<td>-10</td>
<td>38</td>
<td>2,5×11</td>
<td>0–29</td>
<td>48</td>
<td>Died</td>
<td>Green material elicited intermittent slight reduction in ruminal movement and inappetence (0–29). Dried material induced inappetence depression, reduction in ruminal movements, dyspnoea and sudden death</td>
<td>No notable changes</td>
<td>Liver: Diffuse fatty metamorphosis and degenerative changes. <strong>Lungs:</strong> Congestion, haemorrhages and oedema. <strong>GIT:</strong> Catarrhal enteritis</td>
</tr>
<tr>
<td>4</td>
<td>27</td>
<td>F</td>
<td>MT</td>
<td>Douglas 1</td>
<td>Dry</td>
<td>10–32</td>
<td>377</td>
<td>5×2</td>
<td>0–1</td>
<td>4</td>
<td>Discharged</td>
<td>No clinical signs</td>
<td>Not available</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>39</td>
<td>M</td>
<td>MT</td>
<td>Douglas 2</td>
<td>Dry</td>
<td>0</td>
<td>54</td>
<td>5×3</td>
<td>0–2</td>
<td>3</td>
<td>Killed</td>
<td>Apathy, anorexia, icterus, ruminal stasis, bloody nasal discharge, dyspnoea, double respiratory effort, grinding of teeth. Destroyed within 3 h of first clinical signs</td>
<td>y-GT 95 µg/l&lt;br&gt;AST 156 µg/l&lt;br&gt;Total bilirubin 3.1 mg /100 ml</td>
<td>Mild icterus <strong>Liver:</strong> Diffuse degenerative changes and mild fatty metamorphosis and Kupffer cell activation. <strong>Lungs:</strong> Mononuclear interstitial pneumonia with bronchi and bronchiolar epithelial hyperplasia. <strong>Kidneys:</strong> Nephrosis</td>
</tr>
<tr>
<td>6</td>
<td>34</td>
<td>M</td>
<td>MT</td>
<td>Douglas 2</td>
<td>Dry</td>
<td>0</td>
<td>75</td>
<td>2,5×7</td>
<td>0–10</td>
<td>31</td>
<td>Died</td>
<td>Intermittent apathy, anorexia reduction in ruminal movements or stasis, sudden death</td>
<td>AST 356 µg/l</td>
<td>To decompased for diagnosis</td>
</tr>
<tr>
<td>No.</td>
<td>Mass (kg)</td>
<td>Sex</td>
<td>Age</td>
<td>Source</td>
<td>State</td>
<td>Approximate temperature of storage °C</td>
<td>Approximate length of storage (days)</td>
<td>Dose (g/kg×n)</td>
<td>Period dosed (days)</td>
<td>Duration of experiment (days)</td>
<td>Fate</td>
<td>Clinical signs</td>
<td>Clinical pathology</td>
<td>Pathology</td>
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<tr>
<td>7</td>
<td>32</td>
<td>F</td>
<td>MT</td>
<td>Douglas 2</td>
<td>Dry</td>
<td>0</td>
<td>103</td>
<td>5×3</td>
<td>0–2</td>
<td>3</td>
<td>Died</td>
<td>Apathy, died overnight without signs being observed</td>
<td>y-GT 100 µg/l</td>
<td>AST 381 µg/l</td>
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<td></td>
<td></td>
<td>Mild ascites, haemorrhages in connective tissue around trachea and aorta. Liver: Centrilobular necrosis and bile ductular proliferation.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>21</td>
<td>F</td>
<td>MT</td>
<td>Douglas 2</td>
<td>Dry</td>
<td>0</td>
<td>166</td>
<td>2,5×19</td>
<td>0–24</td>
<td>24</td>
<td>Discharge</td>
<td>No clinical signs</td>
<td>No notable change</td>
<td>—</td>
</tr>
<tr>
<td>9</td>
<td>22</td>
<td>W</td>
<td>MT</td>
<td>Douglas 2</td>
<td>Dry</td>
<td>0</td>
<td>49</td>
<td>5×1</td>
<td>29</td>
<td>2</td>
<td>Killed</td>
<td>Intermittent reduction in ruminal movements, apathy, Anorexia, ruminal stasis, apathy, reluctance to stand</td>
<td>y-GT 120 µg/l</td>
<td>AST 272 µg/l</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Fauresmith</td>
<td>Dry</td>
<td>0</td>
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<td></td>
<td></td>
<td></td>
<td>Liver: Diffuse degeneration, severe fatty metamorphosis, and mild bile ductular proliferation. Lungs: Mononuclear interstitial pneumonia with bronchi and bronchiolar epithelial hyperplasia. Kidneys: Nephrosis</td>
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</tbody>
</table>

W = Wether  
M = Male  
F = Female  
g/kg × n = number of daily administrations  
MT = Milk tooth  
2T = Two tooth  
FM = Full mouth  
GIT = Gastrointestinal tract
AN OVINE HEPATOTOXICOSIS CAUSED BY THE PLANT \textit{HERTIA PALLENS}

\section*{ACKNOWLEDGEMENTS}

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\section*{REFERENCES}


