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

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ABSTRACT

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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 (γ-GT) activity in 3 samples was mildly elevated.

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

DESCRIPTION, DISTRIBUTION AND ECOLOGY 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*

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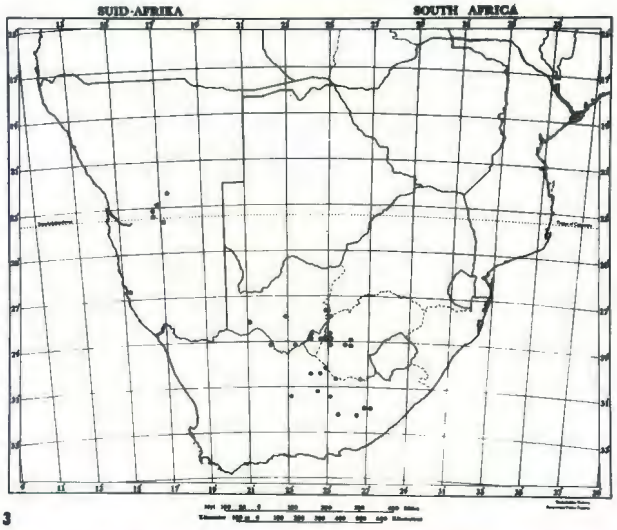

FIG. 3 Distribution of *H. pallens*

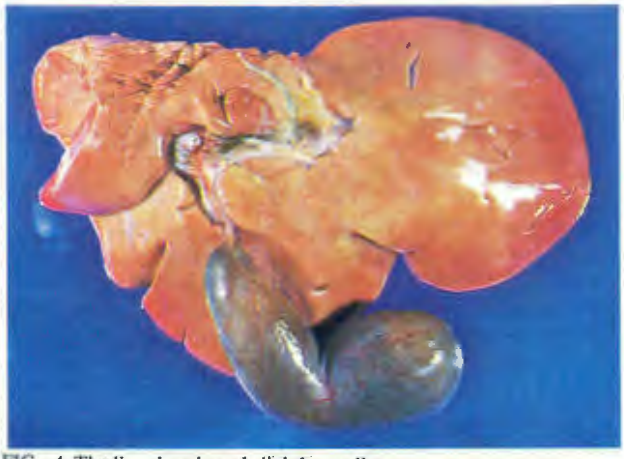

FIG. 4 The liver is pale and slightly swollen

teeth, axils woolly. *Capitula* up to 15 mm long, solitary, terminating the branchlets, shortly pedunculate, heterogamous, radiate, many-flowered, florets golden-yellow to orange. *Involucre* campanulate, 5-lobed, uniseriate, connate below, lobes acute, membrane-edged, up to 10 mm long. *Receptacle* flat, nude. *Radiate florets* 5, fertile, female, corolla shorter than ovary, ligule about 10 mm long, shortly exserted; ovary biconvex, obovoid, ribbed, villous; style terete, longer than corolla-tube, with linear, obtuse branches. *Achenes* silky-villous, pappus of many bristles. Disc-florets sterile, rarely fertile, corolla-tube widened above, ribbed, glabrous, with 5 ovate lobes; anthers faintly sagittate at base, with ovate apical appendage; ovary ribbed, glabrous; style terete, branched. *Achenes* almost glabrous, pappus of few bristles. *Flowering time* from August to November, but mostly in August and September.

The distinctive characteristics of *H. pallens* are the pale, papery stems, the flat, linear-oblong, alternate leaves and the solitary, radiate, flowerheads terminating the branchlets (Harvey, 1865; Dyer, 1975).

Distribution: (Fig. 3) The plant occurs in South West Africa, the South-western Transvaal, the Western Orange Free State, the Northern Cape and the upper and eastern Karoo. It has been recorded in the following districts:—

South West Africa: Windhoek, Rehoboth, Lüderitz

Transvaal: Christiana

Orange Free State: Boshof, Brandfort, Bloemfontein, Fauresmith

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. pallens 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 500–1 500 m above sea-level. The plants are only browsed by stock in times of drought and according to Acocks (1975), *H. pallens* is an undesirable plant which should be reduced in number by appropriate veld management. He lists *H. pallens* 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. pallens* 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 Tabel 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, tympany, 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 $\mu g/\ell$) and/or AST (156–380 $\mu g/\ell$) 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

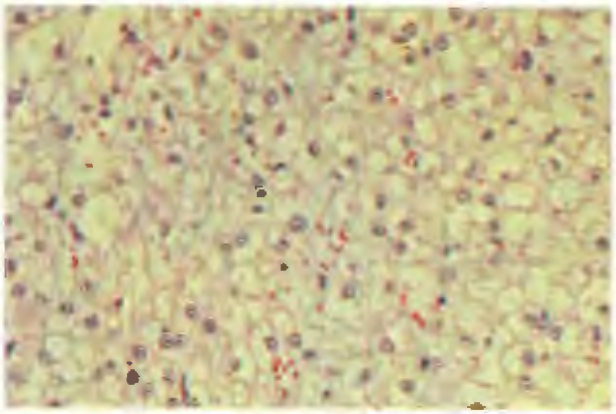

FIG. 5 Diffuse fatty metamorphosis in the liver: HE × 160

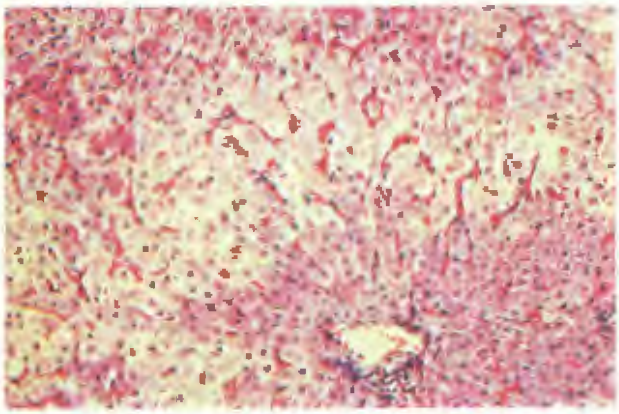

FIG. 6 Centrilobular to midzonal coagulative necrosis: HE × 40

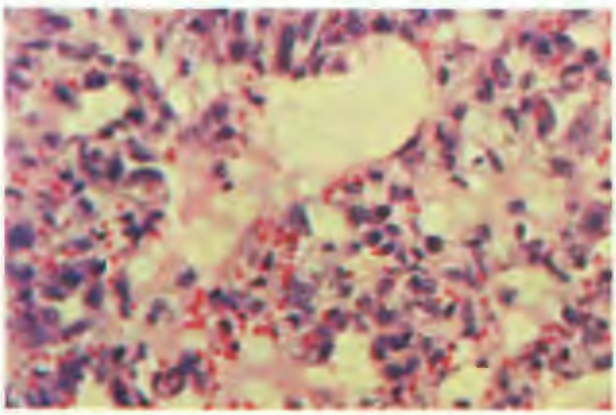

FIG. 7 Alveolar oedema and an interstitial pneumonia: HE × 160

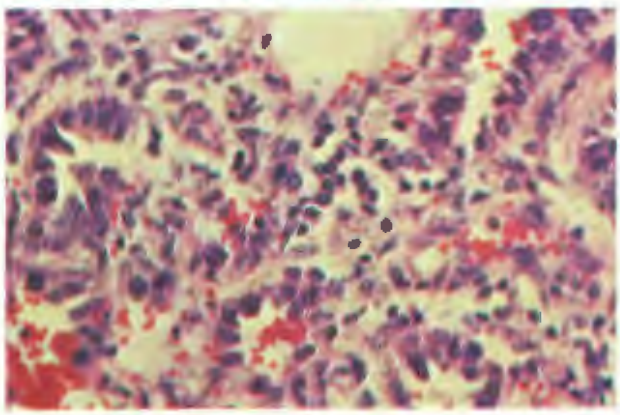

FIG. 8 Epithelial hyperplasia of the smaller bronchi: HE × 160

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. pallens* poisoning occurred in that particular camp and not on neighbouring farms where non-selective grazing was not enforced.

H. pallens 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. pallens* 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. pallens* 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 especiallly 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. pallens*.Sheep, however, appeared to vary greatly in individual susceptibility to intoxication.

TABLE 1 Dosing regimen, clincal signs and pathology of sheep dosed with *H. pallens*

Sheep				*H. pallens*				Dosing regimen			Fate	Clinical signs	Clinical pathology	Pathology
No.	Mass (kg)	Sex	Age	Source	State	Approximate temperature of storage °C	Approximate length of storage (days)	Dose (g/kg×n)	Period dosed	Duration of experiment (days)				
1	24	M	MT	Douglas 1	Green	−10	14	5×2	0–1	2	Died	Tympany, depression, dyspnoea, stands with front legs apart and head down, foam at mouth, cyanosis. Died within 18 h of first clinical signs	Not available	Hydrothorax, hydropericardium and cyanosis. *Liver:* Centrilobular to midzonal necrosis, bile ductular proliferation and Kupffer cell activation. *Kidneys:* Nephrosis. *GIT:* Stasis
2	44	M	2T	Douglas 1	Green	−10	27	2,5×2	0–2	4	Killed	Mild tympany, dyspnoea, reduced ruminal movements, anorexia. Destroyed 48 h after beginning of clinical signs	No notable changes	*Liver:* Diffuse degeneration, mild fatty metamorphosis and Kupffer cell activation. *Lungs:* Oedema, mononuclear interstitial pneumonia with bronchi and bronchiolar epithelial hyperplasia. *Kidneys:* Nephrosis
3	41	F	FM	Douglas 1	Green Dry	−10 15	38 77	2,5×11 5×1 2,5×3 5×3	0–29 32 39–41 46–48	48	Died	Green material elicited intermittent slight reduction in ruminal movements and inappetence (0–29). Dried material induced inappetence depression, reduction in ruminal movements, dyspnoea and sudden death	No notable changes	*Liver:* Diffuse fatty metamorphosis and degenerative changes. *Lungs:* Congestion, haemorrhages and oedema. *GIT:* Catarrhal enteritis
4	27	F	MT	Douglas 1	Dry	10–32	377	5×2 10×3	0–1 2–4	4	Discharged	No clinical signs	Not available	—
5	39	M	MT	Douglas 2	Dry	0	54	5×3	0–2	3	Killed	Apathy, anorexia, icterus, ruminal stasis, bloody nasal discharge, dyspnoea, double respiratory effort, grinding of teeth. Destroyed within 3 h of first clinical signs	γ-GT 95 μg/ℓ AST 156 μg/ℓ Total bilirubin 3,1 mg /100 mℓ	Mild icterus *Liver:* Diffuse degenerative changes and mild fatty metamorphosis and Kupffer cell activation. *Lungs:* Mononuclear interstitial pneumonia with bronchi and bronchiolar epithelial hyperplasia. *Kidneys:* Nephrosis
6	34	M	MT	Douglas 2	Dry	0	75	2,5×7 5×5 10×5	0–10 14–18 21–30	31	Died	Intermittent apathy, anorexia reduction in ruminal movements or stasis, sudden death	AST 356 μg/ℓ	To decoposed for diagnosis

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No.	Mass (kg)	Sex	Age	Source	State	Approximate temperature of storage °C	Approximate length of storage (days)	Dose (g/kg×n)	Period dosed	Duration of experiment (days)				
7	32	F	MT	Douglas 2	Dry	0	103	5×3	0–2	3	Died	Apathy, died overnight without signs being observed	γ-GT 100 μg/ℓ AST 381 μg/ℓ	Mild ascites. Haemorrhages in connective tissue around trachea and aorta. *Liver:* Centrilobular necrosis and bile ductular proliferation. *Lungs:* Oedema. *Kidneys:* Nephrosis. *GIT:* Stasis
8	21	F	MT	Douglas 2	Dry	0	166	2,5×19	0–24	24	Discharge	No clinical signs	No notable change	—
9	22	W	MT	Douglas 2	Dry	0	166	5×15	0–26	26	Killed	Intermittent reduction in ruminal movements, apathy.	γ-GT 120 μg/ℓ AST 272 μg/ℓ	*Liver:* Diffuse degeneration, severe fatty metamorphosis, and mild bile ductular proliferation. *Lungs:* Mononuclear interstitial pneumonia with bronchi and bronchiolar epithelial hyperplasia. *Kidneys:* Nephrosis
				Fauresmith	Dry	0	49	5×1	29	2		Anorexia, ruminal stasis, apathy, reluctance to stand		

W = Wether
M = Male
F = Female
g/kg × n = number of daily administrations
MT = Milk tooth
2T/ = Two tooth
FM = Full mouth
GIT = Gastro intestinal tract

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