Studies on some paraclinical indices on intoxication in horses from freshly cut Jimson weed (*Datura stramonium*)-contaminated maize intended for ensiling

R Binev[a*], I. Valchev[a] and J. Nikolov[a]

ABSTRACT

Monitoring of changes in some blood laboratory parameters in 34 horses after ingesting freshly harvested maize that was to be used for ensiling, heavily contaminated with young *Datura stramonium* plants, is described. For a 7-day period the following parameters were monitored: haemoglobin content (HGB), red blood cell counts (RBC), white blood cell counts (WBC), haematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC), differential white cell counts (DWC), erythrocyte sedimentation rate (ESR), protein fractions, aspartate aminotransferase (AST), lactate dehydrogenase (LDH), total bilirubin (TB), direct bilirubin (DB), blood glucose (Glu), total protein (TP), globulin (Glob) and albumin (Alb). The intoxication was accompanied by erythrocytosis, leukocytosis, regenerative left shift neutrophilia, lymphopaenia, eosinopaenia, increased haematocrit values, low erythrocyte sedimentation rate, hyperglycaemia, bilirubinaemia, hypoproteinaemia and increased activity of AST and LDH. No changes occurred in the mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC), albumin, globulin and globulin fractions (α_1, α_2, β_1, β_2 and γ). The blood parameters returned to normal between post-intoxication days 2 and 5. The observed changes in clinical chemistry indices could be used in the diagnosis, differential diagnosis and prognosis of Jimson weed intoxication.

Key words: atropine, *Datura stramonium*, horses, intoxication, Jimson weed, scopolamine, tropane alkaloids.

Binev R, Valchev I, Nikolov J **Studies on some paraclinical indices on intoxication in horses from freshly cut Jimson weed (*Datura stramonium*)-contaminated maize intended for ensiling**. *Journal of the South African Veterinary Association* (2006) 77(3): 145–149 (En.). Department of Internal Medicine, Faculty of Veterinary Medicine, Trakia University, Stara Zagora, Bulgaria.

INTRODUCTION

Jimson weed (*Datura stramonium*) is an annual of the family Solanaceae. The toxic principles are tropane belladonna alkaloids which possess strong anticholinergic properties[5,31,32,36]. They include hyoscyamine (stems, leaves, roots, seeds)[27], hyoscine (roots); atropine (*d,l*-hyoscyamine) and scopolamine (*l*-hyoscine)[11,16,20,23,27,31,32,39]. All parts of the plant are toxic, but the greatest amount of alkaloids are contained in ripe seeds[7,13,16,36], young, dried leaves[28] and stems and leaves of young plants[27]. They act as competitive antagonists to acetylcholine at peripheral and central muscarinic receptors at a common binding site[7,20,36]. Poisoning results in widespread inhibition of parasympathetic innervated organs[7,15,20,35,38].

The wide distribution, high toxicity and the potential for occurrence in foodstuffs are responsible for the numerous incidents in humans[1–3,6,7,9–12,14–16,18,19,21,25,30,32–35,39,40].

Cases of Jimson weed intoxication are considerably less frequent in animals such as cattle[29], swine[23,24,26,43], dogs[22,41], sheep and goats[17] and poultry[13,44]. In horses, poisoning has occurred after ingestion of Jimson weed seeds[4,37,42] and dried tef hay contaminated with young *Datura* plants (*D. stramonium* and *D. ferox*)[28].

There are no published reports of cases of intoxication in horses by freshly cut Jimson weed (*D. stramonium*)-contaminated maize intended for ensiling.

The objective of this study was to evaluate the usefulness of clinical chemistry parameters of horses after Jimson weed intoxication for rapid and correct diagnosis, prognosis and effective treatment of this intoxication.

MATERIALS AND METHODS

In October 1999, 3 stallions were referred to the Clinic of Internal Diseases and Clinical Toxicology of the Faculty of Veterinary Medicine, Stara Zagora, Bulgaria. The history revealed that 18 hours previously, 34 horses, owned by the Horse Station of the Faculty of Agriculture of the Trakia University, were fed *ad libitum* with freshly harvested and chopped maize that was to be used for ensiling, heavily contaminated with young *D. stramonium* plants. All animals that ingested the forage manifested signs of intoxication to varying degrees. The animals with the most evident clinical signs were referred to the clinic.

The examination *in situ* showed that the horses were 3–14 years old, with a live body weight of 400-600 kg, of various breeds (Trakehner, Hanoverian, Danube, East-Bulgarian, Arabian etc.) and type. The animals were of both sexes: 18 mares, 12 stallions and 4 geldings.

Depending on the severity of clinical signs, the animals were divided into 3 groups:

Group 1 (n = 18) – horses with typical clinical signs: 9 stallions, 1 gelding, 8 mares, 3 of them pregnant.

Group 2 (n = 16) – horses with less obvious signs of intoxication: 3 stallions, 3 geldings, 2 pregnant mares, 8 lactating dams (the suckling foals were not included in the group).

Group 3 (n = 18) – horses owned by the Mounted Police, housed on the same premises under similar conditions, but fed another forage, served as controls.

On days 1, 2, 3, 4, 5, 6 and 7 after the incident, blood was collected from the jugular vein for determination of the following laboratory parameters: haemoglobin content (HGB; g/dℓ), red blood cell counts (RBC; 10^{12}/ℓ), white blood cell counts (WBC; 10^{9}/ℓ), haematocrit (HCT; ℓ/ℓ), mean corpuscular volume (MCV; fl), mean corpuscular haemoglobin (MCH; g/ℓ), mean corpuscular haemoglobin concentration (MCHC; g/ℓ) and differential white cell counts (DWC; %) on an automated analyser (Cell dyn 4500, USA); the erythrocyte sedimentation rate (ESR; mm/h) on an automated analyser (Greiner bio one, Austria); protein fractions (Dade Behring Nephelometer II, Germany); aspartate aminotransferase (AST; U/ℓ); lactate dehydrogenase (LDH; U/ℓ), total bilirubin (TB; mg/dℓ), direct bilirubin

[a]Department of Internal Medicine, Faculty of Veterinary Medicine, Trakia University, Stara Zagora, Bulgaria

*Author for correspondence. E-mail: binew@abv.bg

Received: November 2005. Accepted: July 2006.

Table 1: **Mean haematological indices in horses after intoxication with Jimson weed** (*Datura stramonium*) **(groups I and II) and in the control group (group III).**

Parameter	Gr	Day after intoxication						
		1	2	3	4	5	6	7
HGB, g/dℓ	I	17.8 ± 1.25[c]	16.2 ± 1.34[a]	16.6 ± 1.21[b]	14.8 ± 1.28[a]	15.5 ± 1.18[a]	12.8 ± 0.92	12.0 ± 1.04
	II	15.4 ± 1.03[b]	14.3 ± 1.26	13.8 ± 1.12	12.4 ± 1.08	11.9 ± 0.89	11.1 ± 0.99	11.7 ± 0.86
	III	11.2 ± 0.92	11.8 ± 0.88	11.1 ± 0.82	10.8 ± 0.77	12.1 ± 1.03	11.7 ± 0.78	10.9 ± 0.82
RBC, 10^{12}/ℓ	I	13.12 ± 1.12[b]	11.05 ± 0.93	9.14 ± 1.02	8.74 ± 0.93	8.52 ± 0.78	9.14 ± 0.77	8.89 ± 0.84
	II	11.68 ± 0.84[a]	9.63 ± 1.04	8.49 ± 0.96	9.13 ± 1.07	8.78 ± 0.76	8.12 ± 0.85	8.43 ± 0.79
	III	8.45 ± 0.62	8.74 ± 0.73	8.82 ± 0.81	8.34 ± 0.75	9.01 ± 0.88	8.73 ± 0.94	8.18 ± 0.69
WBC, 10^{9}/ℓ	I	16.81 ± 0.88[c]	18.11 ± 1.21[c]	12.77 ± 1.58[a]	10.34 ± 1.04	9.88 ± 1.03	8.89 ± 0.93	9.29 ± 1.13
	II	14.62 ± 1.54[b]	10.32 ± 1.78	9.31 ± 0.82	8.86 ± 0.96	9.39 ± 1.27	8.47 ± 0.91	8.55 ± 0.96
	III	8.55 ± 0.92	9.16 ± 0.88	8.84 ± 0.98	8.33 ± 0.78	8.14 ± 0.93	8.93 ± 0.76	8.78 ± 0.91
ESR, mm/h	I	30.3 ± 5.2[c]	58.3 ± 7.2	75.4 ± 4.3	66.6 ± 5.8	74.6 ± 6.3	72.5 ± 6.5	72.3 ± 8.4
	II	52.4 ± 5.8[b]	63.4 ± 5.2	78.8 ± 5.4	69.7 ± 4.9	70.1 ± 8.4	71.4 ± 7.6	77.3 ± 7.8
	III	76.2 ± 6.6	75.1 ± 8.9	78.8 ± 6.2	78.4 ± 6.1	71.7 ± 6.8	74.4 ± 5.9	71.9 ± 6.5
HCT, ℓ/ℓ	I	0.58 ± 0.06[c]	0.56 ± 0.06[c]	0.44 ± 0.06	0.38 ± 0.04	0.33 ± 0.04	0.35 ± 0.03	0.34 ± 0.04
	II	0.46 ± 0.05[a]	0.42 ± 0.05	0.32 ± 0.04	0.36 ± 0.2	0.31 ± 0.04	0.37 ± 0.05	0.36 ± 0.06
	III	0.31 ± 0.03	0.33 ± 0.04	0.36 ± 0.05	0.34 ± 0.03	0.35 ± 0.06	0.38 ± 0.05	0.32 ± 0.04

[a]$P < 0.05$; [b]$P < 0.01$; [c]$P < 0.001$.
Key: Gr, group; HGB, haemoglobin; RBC, erythrocyte; WBC, leukocyte; ESR, erythrocyte sedimentation rates; HCT, haematocrit.

(DB; mg/dℓ), blood glucose (Glu; mg/dℓ), total protein (TP; g/dℓ) and albumin (Alb; g/dℓ) on an automated biochemical analyser (Olympus AU 600, Japan). The globulin concentrations were calculated as albumin subtracted from total protein.

All results were statistically processed using ANOVA (Statistica software). The significance of differences was evaluated *vs* the control group for each time interval. The results were determined as statistically significant when $P < 0.05$.

RESULTS

Haematological studies

The haemoglobin content (Table 1) in group I was significantly higher between post-intoxication days 1 to 5. Peak values in groups I and II were measured on the 1st day: 17.8 ± 1.25 g/dℓ ($P < 0.001$) and 15.4 ± 1.03 g/dℓ ($P < 0.01$), respectively, compared with the control group (11.2 ± 0.92 g/dℓ).

The average red blood cell count (Table 1) in horses from groups I and II was elevated only during the 1st day: 13.12 ± 1.12 10^{12}/ℓ ($P < 0.01$) and 11.68 ± 0.84 10^{12}/ℓ ($P < 0.05$), respectively, compared with the control group (8.45 ± 0.62 10^{12}/ℓ).

The mean white blood cell count (Table 1) in group I was statistically significantly higher on post-intoxication days 1 to 3. The maximum count was recorded on day 2: 18.11 ± 1.21 10^{9}/ℓ, compared to 9.16 ± 0.88 10^{9}/ℓ in the control group ($P < 0.001$). In group II, a significant difference was only detected on day 1 (14.62 ± 1.54 10^{9}/ℓ, $P < 0.01$).

The mean differential white blood cell count (Table 2) revealed no eosinophils on certain days, in group I for 3 days, whereas in group II on post-intoxication days 1 and 2.

Neutrophils in groups I and II were elevated between days 1 and 6. The highest count for banded neutrophils was observed on the 2nd day: 16.1 ± 1.3 % ($P < 0.001$) and 7.2 ± 0.5 % ($P < 0.001$), respectively, compared with control percentage of 0.8 ± 0.1 %.

Segmented neutrophils were significantly higher only in group I on post-intoxication days 1 to 4. The highest levels occurred on day 1: 78.3 ± 6.7 % ($P < 0.01$)

Table 2: **Differential white counts (%) (mean, SD) in horses after intoxication with Jimson weed** (*Datura stramonium*) **(groups I and II) and in the control group (group III).**

Parameter	Gr	Day after intoxication						
		1	2	3	4	5	6	7
Eosinophils	I	0[c]	0[c]	0[c]	0.8 ± 0.1[c]	3.2 ± 0.4	4.1 ± 0.3	5.2 ± 0.2
	II	0[c]	0[c]	1.1 ± 0.1[c]	3.7 ± 0.4	4.1 ± 0.3	3.7 ± 0.3	4.2 ± 0.5
	III	3.8 ± 0.4	4.4 ± 0.3	5.2 ± 0.3	4.1 ± 0.4	3.9 ± 0.4	3.4 ± 0.4	4.3 ± 0.5
Banded neutrophils	I	13.7 ± 0.9[c]	16.1 ± 1.3[c]	7.7 ± 0.9[c]	5.8 ± 0.7[c]	2.6 ± 0.3[c]	1.5 ± 0.2[a]	1.4 ± 0.3
	II	6.6 ± 0.6[c]	7.2 ± 0.5[c]	2.6 ± 0.5[b]	1.8 ± 0.1[c]	2.2 ± 0.1[c]	1.7 ± 0.2[a]	1.9 ± 0.4
	III	1.2 ± 0.3	0.8 ± 0.1	0.7 ± 0.2	1.1 ± 0.1	1.0 ± 0	0.9 ± 0.3	1.2 ± 0.1
Segmented neutrophils	I	78.3 ± 6.7[b]	74.2 ± 8.6[a]	70.7 ± 4.8[b]	72.1 ± 6.2[b]	62.7 ± 5.5	49.1 ± 6.3	50.7 ± 4.3
	II	61.8 ± 8.8	62.3 ± 6.4	54.7 ± 6.2	59.6 ± 6.8	46.6 ± 6.1	48.8 ± 4.1	47.3 ± 3.9
	III	48.5 ± 3.4	51.1 ± 5.3	46.9 ± 5.8	49.3 ± 3.9	50.5 ± 5.2	47.6 ± 3.8	48.9 ± 3.3
Lymphocytes	I	5.8 ± 0.5[c]	11.1 ± 1.0[c]	24.1 ± 3.1[c]	23.8 ± 3.1[c]	32.1 ± 2.2	44.2 ± 4.1	43.8 ± 4.2
	II	33.8 ± 2.8[a]	32.8 ± 3.3	44.8 ± 4.3	36.8 ± 2.9	45.8 ± 3.6	46.1 ± 3.6	47.1 ± 3.9
	III	46.1 ± 3.7	44.3 ± 4.5	47.6 ± 3.8	45.5 ± 5.2	46.7 ± 3.9	46.2 ± 3.7	45.3 ± 3.1

[a]$P < 0.05$; [b]$P < 0.01$; [c]$P < 0.001$.
Key: Gr, group.

Table 3: **Mean clinical chemistry parameters in horses after intoxication with Jimson weed** (*Datura stramonium*) **(groups I and II) and in the control group (group III).**

Parameter	Gr	Day after intoxication						
		1	2	3	4	5	6	7
Glu (mg/dℓ)	I	172.0 ± 21.9[c]	159.0 ± 16.2[c]	146.2 ± 19.8[c]	110.2 ± 14.4[a]	78.8 ± 12.6	65.3 ± 10.8	68.1 ± 9.0
	II	139.4 ± 14.4[c]	92.2 ± 12.6	71.3 ± 10.8	68.1 ± 9.1	70.9 ± 7.2	71.3 ± 10.8	69.9 ± 16.2
	III	65.0 ± 10.8	67.0 ± 9.0	55.3 ± 9.0	62.3 ± 16.2	67.0 ± 10.8	62.6 ± 9.8	66.4 ± 7.4
TB (mg/dℓ)	I	2.91 ± 0.39[b]	2.92 ± 0.35[b]	3.43 ± 0.33[c]	2.67 ± 0.34[a]	2.70 ± 0.28[b]	1.74 ± 0.25	1.77 ± 0.22
	II	2.84 ± 0.30[b]	2.90 ± 0.28[c]	2.65 ± 0.30[b]	2.49 ± 0.22[a]	1.89 ± 0.25	1.57 ± 0.18	1.29 ± 0.16
	III	1.73 ± 0.22	1.42 ± 0.21	1.33 ± 0.16	1.59 ± 0.19	1.66 ± 0.18	1.43 ± 0.16	1.44 ± 0.15
DB (mg/dℓ)	I	0.72 ± 0.09	0.98 ± 0.11[b]	1.24 ± 0.15[b]	1.10 ± 0.13[b]	0.89 ± 0.09[a]	0.80 ± 0.07	0.74 ± 0.09
	II	0.75 ± 0.09	0.84 ± 0.07[a]	1.16 ± 0.13[b]	1.03 ± 0.12[b]	0.80 ± 0.10	0.72 ± 0.09	0.68 ± 0.08
	III	0.56 ± 0.05	0.60 ± 0.05	0.57 ± 0.07	0.63 ± 0.05	0.59 ± 0.07	0.67 ± 0.05	0.57 ± 0.05
AST (U/ℓ)	I	483 ± 54.2[c]	409 ± 36.5[c]	362 ± 38.6[b]	348 ± 46.8[b]	309 ± 35.3[a]	246 ± 29.5	238 ± 25.4
	II	315 ± 42.8[a]	278 ± 34.2	238 ± 19.4	229 ± 17.9	231 ± 17.4	209 ± 21.6	216 ± 29.8
	III	212 ± 18.6	209 ± 23.9	224 ± 26.2	196 ± 21.1	218 ± 19.8	228 ± 17.9	207 ± 19.5
LDH (U/ℓ)	I	515 ± 84.3[a]	473 ± 62.6[a]	379 ± 48.7	282 ± 36.9	294 ± 37.2	306 ± 28.4	255 ± 35.3
	II	408 ± 52.7	315 ± 34.1	269 ± 37.3	307 ± 44.8	324 ± 26.3	316 ± 31.7	288 ± 32.5
	III	288 ± 46.9	264 ± 32.4	312 ± 28.0	293 ± 37.6	269 ± 42.5	288 ± 36.9	309 ± 30.0
TP (g/dℓ)	I	4.71 ± 0.24[b]	5.24 ± 0.37[b]	6.88 ± 0.45	6.29 ± 0.35	6.38 ± 0.41	6.79 ± 0.36	6.46 ± 0.48
	II	5.82 ± 0.38	6.32 ± 0.44	6.54 ± 0.38	6.78 ± 0.48	6.62 ± 0.36	6.32 ± 0.33	6.11 ± 0.38
	III	6.62 ± 0.41	6.17 ± 0.31	6.34 ± 0.39	6.52 ± 0.31	6.08 ± 0.26	6.42 ± 0.44	6.38 ± 0.41

[a]$P < 0.05$; [b]$P < 0.01$; [c]$P < 0.001$.
Key: Gr, group; Glu, glucose TB, total bilirubin; DB, direct bilirubin; AST, aspartate aminotransferase; LDH, lactate dehydrogenase; TP, total serum protein.

compared with control percentage of 48.5 ± 3.4 %.

A considerable reduction in lymphocytes was noticed. In group I significant changes were observed from day 1 to day 4 after the poisoning and in group II, only on the 1st day. The lowest lymphocyte count in both groups was determined on day 1: 5.8 ± 0.5 % ($P < 0.001$) and 33.8 ± 2.8 % ($P < 0.05$) in groups I and II, respectively.

A low mean erythrocyte sedimentation rate (Table 1) was only observed on the 1st day in both groups: 30.3 ± 5.2 mm ($P < 0.001$) and 52.4 ± 5.8 mm ($P < 0.01$) respectively, compared with a mean ESR of 76.2 ± 6.6 mm in the control group.

The mean haematocrit (Table 1) in horses from group I was significantly higher on post-intoxication days 1 and 2. The highest packed cell volume was determined on day 1 (0.58 ± 0.06 ℓ/ℓ, $P < 0.001$). In group ll the high mean haematocrit (0.46 ± 0.05 ℓ/ℓ) was observed only on day 1

Changes in the mean corpuscular volume, mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration were not statistically significant ($P > 0.05$).

Biochemical analyses

The mean blood glucose concentration (Table 3) in horses from group I was significantly elevated from post-intoxication days 1 to 4. The highest concentration was determined 1 day after Jimson weed ingestion: 172.0 ± 21.9 mg/dℓ ($P < 0.001$) and 139.4 ± 14.4 mg/dℓ ($P < 0.001$) in groups I and II, respectively.

The mean total bilirubin (Table 3) in intoxicated horses increased. In group I significantly higher concentrations occurred from days 1 to 5, whereas in group II elevated concentrations were detected from days 1 to 4. The highest total bilirubin concentrations in group I occurred on day 3 (3.43 ± 0.33 mg/dℓ, $P < 0.001$). In group ll, the peak total bilirubin concentration was observed on day 2 (2.90 ± 0.28 mg/dℓ , $P < 0.001$).

Changes in direct bilirubin were similar to those in total bilirubin (Table 3) In group I, statistically significant differences were present between days 2 and 5, with the highest concentrations observed on day 3 (1.24 ± 0.15 mg/dℓ, $P < 0.01$). In group ll direct bilirubin was increased between post-intoxication days 2 and 4, with a similar peak on day 3 (1.16 ± 0.13 mg/dℓ, $P < 0.01$).

Mean aspartate aminotransferase (AST) activity (Table 3) was higher from day 1 to day 5 day in group I, whereas in group ll only on day 1. The highest activities were measured on day 1 in both groups: 483 ± 54.2 U/ℓ ($P < 0.001$) and 315 ± 42.8 U/ℓ ($P < 0.05$), respectively, in groups I and II, compared with the control group (212 ± 18.6 U/ℓ).

The mean activity of lactate dehydrogenase (Table 3) was elevated on days 1 and 2 in group I only. The peak level was measured on day 1: 515 ± 84.3 U/ℓ ($P < 0.05$), compared with 288 ± 46.9 U/ℓ in the control group.

The mean total serum protein concentrations (Table 3) was significantly lower in horses in group I on days 1 and 2. The lowest mean concentration was determined on day 1 (4.71 ± 0.24 g/dℓ, $P < 0.01$).

There were no statistically significant changes in mean albumin, total globulins and globulin fractions (α_1, α_2, β_1, β_2 and γ) between the different groups ($P > 0.05$).

DISCUSSION

The toxic effect of Jimson weed was characterised by changes in the values of haematological parameters studied – erythrocytosis, leukocytosis, regenerative left shift neutrophilia, lymphopaenia, eosinopaenia, increased haematocrit values and a low erythrocyte sedimentation rate.

The increased haematocrit could probably be attributed to the amount of dehydration, resulting from the lack of thirst[2,3,11,17,23,35,43]. High haematocrit levels were responsible for measuring higher RBC counts (relative polycythaemia) and haemoglobin content in intoxicated horses. The study showed delayed ESR only on post-intoxication day 1. The observed changes could be attributed to observed bilirubinaemia and increased haematocrit that impeded the sedimentation of erythrocytes. The changes in total WBC counts and the percentages of leukocyte classes in equine blood after intoxication with Jimson weed included elevated total WBC counts, elevated neutrophils and segmented neutrophils,

reduced lymphocyte percentages and absence of or reduced eosinophils. The leukocytosis, neutrophilia, lymphopaenia and eosinopaenia are all typical for what we call a 'stress leukogram' due to excessive endogenous cortisol release. This is one of the possible causes for the blood glucose elevation. After Jimson weed intoxication, colics are observed[3,4,16–18,29,32,37,38], because of the anticholinergic effect of plant alkaloids[4,11,16,17,20,28,32,35,41]. The colic could be related to increased amounts of catecholamines (epinephrine amd dopamine), resulting from adrenal hyperfunction under stress. The increase in catecholamines activated glycogenolysis and inhibited insulin release and, therefore, blood glucose increased. On the other hand, hyperglycaemia could be due to dystrophic liver parenchyma changes[18], as the glycogen released by hepatocytes is metabolised to glucose. A 3rd possible explanation for higher blood glucose could be the inhibited function of gastrointestinal glands[16,17,20,25,35,36], including the pancreas, correlated with reduced insulin production.

The blood biochemical analysis revealed increased total and direct bilirubin concentrations as well as increased AST and LDH activities and reduced total protein levels. These changes corresponded to the observed gross changes in liver parenchyma[18] following Jimson weed intoxication. The lower total protein concentrations could also be explained by the loss of appetite[17,23,26,28,29,35,37,41–44] during the intoxication.

These results are similar to those reported by other authors[18] and support the hepatotoxic effect of Jimson weed alkaloids.

CONCLUSIONS

Intoxication with Jimson weed in horses was characterised with changes in some haematological indices – erythrocytosis, leukocytosis, regenerative left shift neutrophilia, lymphocytopenia, eosinopaenia, increased haematocrit values, and low erythrocyte sedimentation rate.

Blood biochemical changes consisted of hyperglycaemia, bilirubinaemia, hypoproteinaemia and increased activity of aspartate aminotransferase (AST) and lactate dehydrogenase (LDH).

REFERENCES

1. Al-Shaikh A M, Sablay Z M 2005 Hallucinogenic plant poisoning in children *Saudi Medical Journal* 26: 118–121
2. Amlo H, Haugeng K L, Wickstrøm E, Koss A, Husebye T, Jacobsen D 1997. Poisoning with Jimson weed. Five cases treated with physostigmine. *Tidsskrift for den Norske Laegeforening* 117: 2610–2612
3. Arouko H, Matray M D, Braganca C, Mpaka J P, Chinello L, Castaing F, Bartou C, Poisot D 2003 Voluntary poisoning by ingestion of *Datura stramonium*. Another cause of hospitalization in youth seeking strong sensations. *Annales de Medecine Interne* 154: 46–50
4. Barney G H, Wilson B J 1963 A rare toxicity syndrome in ponies. *Veterinary Medicine* 58: 419–421
5. Berger E, Ashkenazi I 2003 Jimson weed poisoning. *Harefuah* 142: 364–367 + 397
6. Birmes P, Chounet V, Mazerolles M, Cathala B, Schmitt L, Lauque D 2002 Self-poisoning with *Datura stramonium*: 3 case reports. *Presse Medicale* 31: 69–72
7. Boumba V A, Mitselou A, Vougiouklakis T 2004 Fatal poisoning from ingestion of *Datura stramonium* seeds. *Veterinary and Human Toxicology* 46: 81–82
8. Brown J H, Taylor P 2001 Muscarinic receptor agonists and antagonists. In Hardman J G, Limbird L E, Gilman A G. (eds) *Goodman & Gilman's the pharmacological basis of therapeutics* (10th edn). McGraw-Hill, New York
8. Calbo Mayo J M, Barba Romero M A, Broseta Viana L, Medrano Gonzalez F 2004 Accidental familiar poisoning by *Datura stramonium. Anales de Medicina Interna* 21: 415
10. Castanón López L, Martinez Badás J P, Lapena López de Armentia S, Gomez Mora J, Garcia Arias M L 2000 *Datura stramonium* poisoning. *Anales Espanoles de Pediatria* 53: 53–55
11. Chang S S, Wu M L, Deng J F, Lee C C, Chin T F, Liao S J 1999 Poisoning by *Datura* leaves used as edible wild vegetables. *Veterinary and Human Toxicology* 41: 242–245
12. Crouch B I, Caravati E M, Summers E H 2001 Jimsonweed toxicity and treatment. *Journal of Pharmaceutical Care in Pain and Symptom Control* 9: 25–30
13. Day E J, Dilworth B C 1984 Toxicity of jimson weed seed and cocoa shell meal to broilers. *Poultry Science* 63: 466–468
14. Dewitt M S, Swain R, Gibson L B Jr 1997 The dangers of Jimson weed and its abuse by teenagers in the Kanawha Valley of West Virginia. *West Virginia Medical Journal* 93: 182–185
15. Djibo A, Brah Bouzou S 2000 Acute intoxication to the 'sobi-lobi' (*Datura*). Apropos of 4 cases in Niger. *Bulletin de la Société de Pathologie Exotique* 93: 294–297
16. Dugan G M, Gumbmann M R, Friedman M 1989 Toxicological evaluation of jimson weed (*Datura stramonium*) seed. *Food and Chemical Toxicology* 27: 501–510
17. El Dirdiri N I, Wasfi I A, Adam S E, Edds G T 1981 Toxicity of *Datura stramonium* to sheep and goats. *Veterinary and Human Toxicology* 23: 241–246
18. Ertekin V, Selimoglu M A, Altinkaynak S 2005 A combination of unusual presentations of *Datura stramonium* intoxication in a child: rhabdomyolysis and fulminant hepatitis. *Journal of Emergency Medicine* 28: 227–228
19. Forno F J Jr, Terry R A 1998 Accidental ingestion of jimsonweed by an adolescent. *Journal of the American Osteopathological Association* 98: 502–504
20. Friedman M, Levin C E 1989 Composition of jimson weed (*Datura stramonium*) seeds. *Journal of Agricultural Food Chemistry* 37: 998–1005
21. Haas L F 1995 *Datura stramonium* (Jimsonweed). *Journal of Neurology Neurosurgery and Psychiatry* 58: 654
22. Hanse P, Clerc B 2002 Anisocoria in the dog provoked by a toxic contact with an ornamental plant: *Datura stramonium*. *Veterinary Ophthalmology* 5: 277–279
23. Janssens G, DeWilde R 1989 Toxicity of thornapple (*Datura stramonium* and/or *D. ferox*) seed present in pig feed. *Vlaams Diergeneeskundig Tijdschrift* 58: 84–86
24. Keeler R F 1981 Absence of arthrogryposis in newborn Hampshire pigs from sows ingesting toxic levels of jimsonweed during gestation. *Veterinary and Human Toxicology* 23: 413–415
25. Kurzbaum A, Simsolo C, Kvasha L, Blum A 2001 Toxic delirium due to *Datura stramonium*. *Israel Medical Association Journal* 3: 538–539
26. Leipold H W, Oehme F W, Cook J E 1973 Congenital arthrogryposis associated with ingestion of jimsonweed by pregnant sows. *Journal of the American Veterinary Medicine Association* 162: 1059–1060
27. Miraldi E, Masti A, Ferri S, Barni Comparini I 2001 Distribution of hyoscyamine and scopolamine in *Datura stramonium*. *Fitoterapia* 72: 644–648
28. Naude T W, Gerber R, Smith R J, Botha C J 2005 *Datura* contamination of hay as the suspected cause of an extensive outbreak of impaction colic in horses. *Journal of the South African Veterinary Association* 76: 107–112
29. Nelson P D, Mercer H D, Essig H W, Minyard J P 1982 Jimson weed seed toxicity in cattle. *Veterinary and Human Toxicology* 24: 321–325
30. Nogue S, Pujol L, Sanz P, De la Torre R 1995 *Datura stramonium* poisoning. Identification of tropane alkaloids in urine by gas chromatography – mass spectrometry. *Journal of International Medical Research* 23: 132–137
31. Nuhu H, Ghani A 2002 Alkaloid content of the leaves of three Nigerian *Datura* species. *Nigerian Journal of Natural Products and Medicine* 6: 15–18
32. Oberndorfer S, Grisold W, Hinterholzer G, Rosner M 2002 Coma with focal neurological signs caused by *Datura stramonium* intoxication in a young man. *Journal of Neurology Neurosurgery and Psychiatry* 73: 458–459
33. Parissis D, Mellidis C, Boutis A, Apostolidis K, Ignatiadis M, Kiosses V, Milonas I 2003 Neurological findings in a case of coma secondary to *Datura stramonium* poisoning. *European Journal of Neurology* 10: 745–746
34. Pinilla Llorente B, Portillo A, Muino Miguez A, Garcia Castano J 1992 *Datura stramonium* poisoning. *Annales de Medecine Interne* 9: 208
35. Roblot F, Montaz L, Delcoustal M, Gaboriau E, Chavagnat J J, Morichaud G, Pourrat O, Scepi M, Patte D 1995 *Datura stramonium* poisoning: the diagnosis is clinical, treatment is symptomatic. *Revue de Médécine Interne* 16: 187–190
36. Ruhwald M 2005 Jimsonweed poisoning and the anticholinergic syndrome. *Ugeskrift for Laeger* 167: 2041–2043
37. Schulman M L, Bolton L A 1998 *Datura* seed intoxication in 2 horses. *Journal of the South African Veterinary Association* 69: 27–29
38. Sopchak C A, Stork C M, Cantor R M, Ohara P E 1998 Central anticholinergic syndrome due to Jimson weed: physostigmine therapy revisited. *Journal of Toxicology and Clinical Toxicology* 36: 43–45
39. Steenkamp P A, Harding N M, Van

Heerden F R, Van Wyk B E 2004 Fatal *Datura* poisoning: identification of atropine and scopolamine by high performance liquid chromatography/photodiode array/mass spectrometry. *Forensic Science International* 145: 31–39
40. Thabet H, Brahml N, Amamou M, BenSalah N, Hedhill A, Yacoub M 1999 *Datura stramonium* poisonings in humans. *Veterinary and Human Toxicology* 41: 320–321
41. Tostes R A 2002 Accidental *Datura stramonium* poisoning in a dog. *Veterinary and Human Toxicology* 44: 33–34
42. Williams S, Scott P 1984 The toxicity of *Datura stramonium* (thorn apple) to horses. *New Zealand Veterinary Journal* 32: 47
43. Worthington T R, Nelson E P, Bryant M J 1981 Toxicity of thornapple (Datura stramonium L) seeds to the pig. *Veterinary Record* 108: 208–211
44. Yaman I, Eroksuz H, Ozer H, Ceribasi A O 2005 Toxicity of dietary *Datura stramonium* seed to broilers. *Indian Veterinary Journal* 82: 379–383