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Original Contribution

INTOXICATION WITH POISON HEMLOCK (*CONIUM MACULATUM L.*) IN CALVES

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ABSTRACT

The case of spontaneous poisoning of 38 calves with Poison Hemlock (*Conium maculatum*) was described. The following indices were measured, among others: 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), etc. The resulting intoxication was accompanied by altered clinical status, some of which were: hyperthermia, tachycardia, polypnoea with dyspnoea, etc. There were also changes in clinical chemistry indices. The observed clinical signs, the changes in haematological and biochemical indices in blood, morphological alterations and the applied therapy could be used in the diagnosis, differential diagnosis, prognosis and treatment of Poison Hemlock (*Conium maculatum*) intoxication.

Key words*:* calves, intoxication, Poison Hemlock (*Conium maculatum*)

INTRODUCTION

The Poison Hemlock (*Conium maculatum*) is a common annual or biennial herb from *Apiaceae* family The plant is found in Europe, North and South America, West Asia, Australia, New Zealand and Oceania (1-28).

Spontaneous intoxication with Poison Hemlock have been reported, for example in cattle: (8-11, 14, 20, 26, 27, 29-39), pigs (5, 8, 10, 11, 14, 20, 27, 33, 34, 37, 38, 40-43); in sheep (8, 10, 11, 19, 20, 26, 27, 32, 33, 34, 36-39, 44-46); in goats (8, 10-12, 15, 27, 33, 34, 37, 38, 47, 48); in horses (8, 10, 11, 20, 27, 32, 33, 34, 37, 38); in donkeys (8), elks (49), rats (21, 50), rabbits (21), slugs (28), birds (1, 3, 50, 51) and humans (7, 11, 14, 52-57).

Animal intoxication with Poison Hemlock presents two clinical forms – severe and chronic (10, 16, 33, 47, 48).

The level of severity depends on the animal species: for the severe form– cattle, sheep, goats and pigs, and for the chronic form – cattle, sheep and pigs (9). Our data (8) from other investigations on the distribution, season, reasons and clinical symptoms of Poison Hemlock intoxication showed that most sensitive are cattle, followed by horses, pigs, sheep and finally goats. The lethal dose of coniine for cattle following injection is 16mg/kg weight. For the sheep this dose is 240mg/kg weight. Severe toxic signs are observed in 3.3mg/kg oral coniine in cattle, and 15.5 mg/kg in horses. The dose of 44 mg/kg has a medium effect in sheep. The lethal dose for pigs in contact with Poison Hemlock semen is 1 g/kg weight, and 2g/kg weight in humans (10, 11, 18, 19, 20, 27, 39, 44, 45).

The data from other authors concerning the common clinical symptoms of severe form in animals are: hypo- or hyperthermia, tachycardia, polipnoe, dispnoe, arexia, tremor, ataxia and etc. (8, 20, 21, 29, 39). Species specific symptoms are: high level timpani and full atone for cattle (8, 11, 14, 20, 26, 29, 35, 39), relapsed timpani and urine incontinence in goats (8, 10, 11, 12, 15, 27, 47, 48), green diarrhoea and polakysuria in sheep (8, 10, 11, 19, 20, 26, 27, 39, 44 - 46), third eyelid dropping, pupil extension and temporary blindness in pigs (5, 8, 10, 11, 14, 20, 27, 34, 41), medium strong, short, interrupted by short painless pauses of colic attack in horses (Dilatatio ventriculi acuta) (8, 10, 11, 20, 27), weak, short, interrupted by long painless pauses of colic attack in donkeys

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(Meteorismus intestinorum) (8).

More authors reported the clinical appearance of chronic intoxication after experimental reproduction (30, 31, 32) and very rare for spontaneous intoxications. The latter comprised teratogenic effects (scoliosis, open palate, arthrogriposis, hydrocephaly, torticolis and congenital flexi of joints) in calves, pigs and lambs (27, 30 – 34, 36–38, 40 – 45, 47, 48).

The information in the literature, about clinical, paraclinical and morphological investigations and effective treatment of spontaneous Poison Hemlock (*Conium maculatum*) intoxication in calves are contradictory and exiguous. This motivated our clinical, para-clinical and morphological investigations in calves with Poison Hemlock intoxication with regard to the rapid and correct diagnosis, differential diagnosis and prognosis and consequently, effective treatment of this intoxication.

MATERIALS AND METHODS

In June 2005 we observed the large-scale intoxication of calves in the region of Kotel. The anamnesis determined that the animals were reared whole day on pasture (uncultivated land about 2 500 dka on the riverside of Kamchia). This pasture had plenty of clover, lupines, hedge grass and etc. We observed large-scale distribution of Poison Hemlock (*Conium maculatum*), which, in most cases, was attracted to animals. The owner raised 95 calves (7 to 18 months age). All animals were males, Bulgarian Brown Breed and crossed with Bulgarian Black and White breed. The clinical signs of intoxication were observed in 38 calves. Three of them had lethal outcome.

The animals were divided into 2 groups:

- Group one (n=38) – calves with clinical signs of intoxication.
- Group two (n=57) – calves, reared at the same pasture conditions, but without clinical signs of intoxication.

The complete clinical status of the two groups of calves was performed by taking body (rectal) temperature using an electronic thermometer (*GT 2038 Geratherm Medical, Germany*), then respiratory and heart rates, rumen movements, pH on rumen liquid, colour of visible mucosae, appetite, general behaviour, locomotion, sensory perceptions, urination, defecation etc.

Blood was collected from *V. jugularis* for determination of the following laboratory parameters: haemoglobin content (HGB; g/L), red blood cell counts (RBC; T/L) and white blood cell counts (WBC; G/L), haematocrit (HCT; %), 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 (α_1, α_2, β_1, β_2 and γ) (Dade Behring Nephelometer II, Germany), aspartate aminotransferase (AST; U/L), alanine aminotransferase (ALT; U/L), lactate dehydrogenase (LDH; U/L), creatinine kinase (CK; U/L), alkaline phosphatase (AlP; U/L), total bilirubin (TB; μmol/L), blood glucose (Glu; mmol/L), creatinine (Creat; μmol/L), urea (mmol/L), total protein (TP; g/L) and albumin (Alb; g/L) on an automated biochemical analyser (Olympus AU 600, Japan). The globulin concentrations were calculated as albumin subtracted from total protein.

All calves with clinical symptoms were medically treated.

The 3 calves that died were necropsied.

All results were statistically processed using the ANOVA test (Statistica software). The significance of differences was evaluated against the control group for each time interval. The results were determined as significant at the $p<0.05$ level.

RESULTS

I. Clinical studies

The changes in the clinical parameters (body temperature, heart and respiratory rates, rumen movements and pH on rumen liquid) are presented in **Figure 1**. The rectal temperature was elevated in calves from the first group – 40.7 ± 0.2^0C ($p<0.001$), *vs.* the control measurements – 38.8 ± 0.3^0C.

Heart rates were accelerated in calves with intoxication – 114.8 ± 8.8 min^{-1} ($p<0.001$), compared to the control values (66.3 ± 6.8 min^{-1}). A weak, soft and arrhythmic pulse and visible cardiac shocks in cardiac region were registered.

The respiratory movements were also accelerated in group I – 54.5 ± 6.1 min^{-1} ($p<0.001$), *vs.* the controls group – 19.2 ± 3.3 min^{-1}. The respiratory movements were hardly embarrassed, accompanied by stridors. With half of the cases we determined breathing with open mouth and presence of bilateral, serous nasal effluents, sometimes mixed with air.

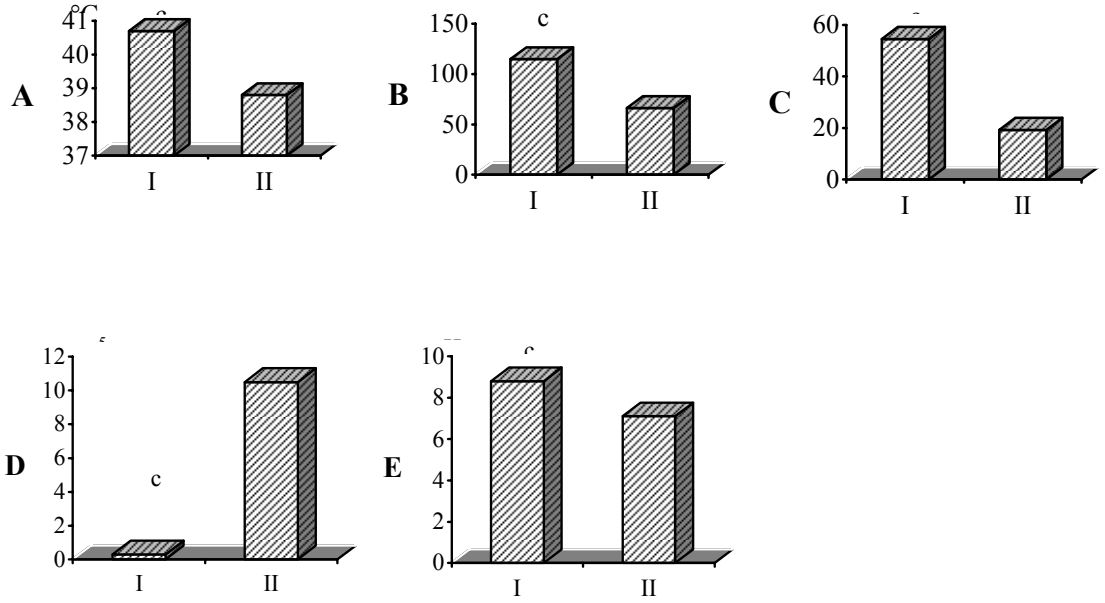

Figure 1. *Change in clinical indices - Body temperature (A), Heart rate (B), Respiratory rate (C), Rumen movements (D) and pH on rumen liquid (E) in calves after intoxication with Poison Hemlock (Conium Maculatum) (Group I) and controls (Group II). Level of significance vc controls:* $^{a}p<0.05$; $p^{b}<0.01$; $p^{c}<0.001$.

In 32 (84 %) of intoxications we observed full absence of rumen movements. In the rest 6 calves (16 %), the rumen movements were from 1 to 2 and almost imperceptible.

The pH on rumen liquid was elevated in calves from the first group – 8.8±0.2 ($p<0.001$), *vs*. the control measurements – 7.1±0.1.

In calves with intoxication we observed also refusal to eat, lack of defecation and urination, hyper salivation, lack of thirst, diffusely reddened conjunctivae and mydriasis, eyelid dropping, groaning, grinding of teeth, disturbances in locomotion, muscle cramps, ataxia, hyporreflexia, dermal anaesthesia, convulsions.

The continuance of observed symptoms of intoxication with Poison Hemlock (*Conium Maculatum*) in calves was from 10 to 12 h.

II. Haematological studies

The changes in haematological parameters (haemoglobin content, red blood cell counts, white blood cell counts and haematocrit) in calves after intoxication with Poison Hemlock (*Conium Maculatum*) are presented in **Figure 2**. The haemoglobin content in calves from group I was significantly higher – 168.3±11.6 g/L ($p<0.001$), compared to the control (111.6±8.3 g/dL).

The average red blood cell count in calves with intoxication (group I) and group II were elevated – 12.82±1.18 T/L ($p<0.01$), compared to the control group (8.65±0.78 T/L).

The mean white blood cell count in group I was significantly higher – 14.3±1.2 G/L, as compared to 9.8±0.97 G/L in the controls ($p<0.01$).

The mean haematocrit in group I was significantly higher – 57.6±4.6 % ($p<0.001$), vs. the control group – 33.4±3.5 %.

The mean eosinophils count in the control group are 4.1±0.3 %.

Neutrophils in groups I were elevated – 9.8±1.3 % ($p<0.001$), compared to control percentages – 0.7±0.1 %.

Segmented neutrophils were significantly higher in group I on post intoxication – 61.5±6.2 % ($p<0.01$ than controls – 39.4±3.3 %). A considerable reduction in lymphocytes was noticed. In group I the mean lymphocytes counts were 28.6±2.5% ($p<0.001$), compared to controls group – 49.2±3.8 %.

Significant changes in the mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC) and erythrocyte sedimentation rate (ESR) were not detected ($p>0.05$).

The mean differential white blood cell count (**Figure 3**) showed no eosinophils in blood on calves after intoxication with Poison Hemlock (*Conium Maculatum*).

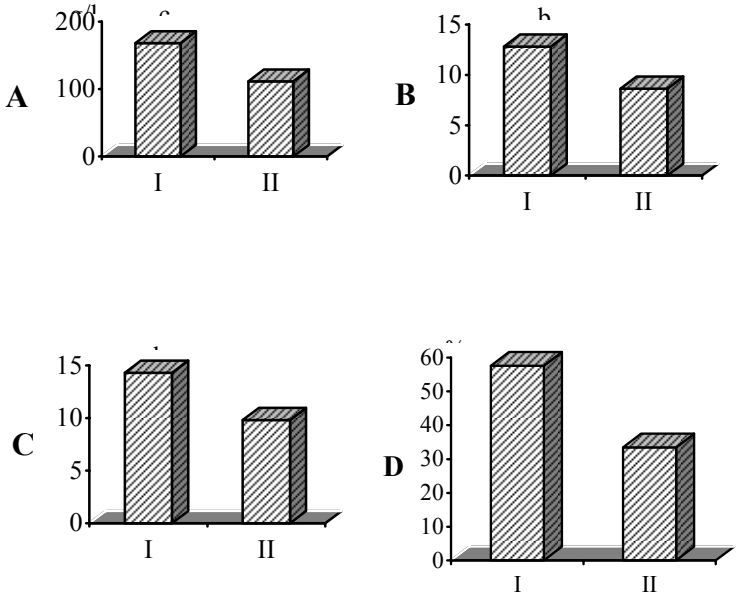

Figure 2. *Change in haematological indices - haemoglobin content (A), red blood cell counts (B), white blood cell counts (C) and haematocrit (D) in calvses after intoxication with Poison Hemlock (Conium Maculatum) (Group I) and controls (Group II). Level of significance vs. controls:* $^{a}p<0.05$; $p^{b}<0.01$; $p^{c}<0.001$.

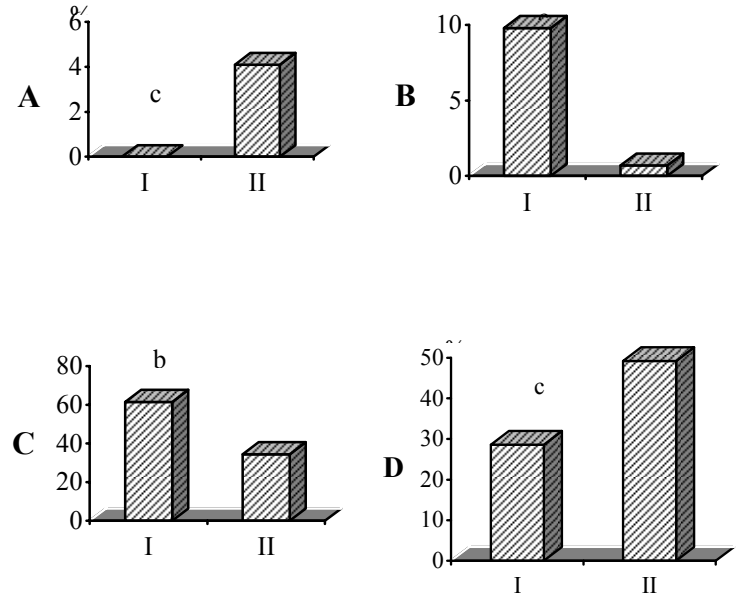

Figure 3. *Change in Differential white counts (DWC) - Eosinophils (A), Banded neutrophils (B), Segmented neutrophils (C) and Lymphocytes (D) in calvses after intoxication with Poison Hemlock (Conium Maculatum) (Group I) and controls (Group II). Level of significance vc controls:* $^{a}p<0.05$; $p^{b}<0.01$; $p^{c}<0.001$.

III. Biochemical analyses

The changes in the biochemical parameters (blood glucose, total bilirubin, creatinine and urea) in calves after intoxication with Poison Hemlock (*Conium Maculatum*) are presented in **Figure 4.**

The mean blood glucose concentration (**Figure 4 A**) in animals from group I was significantly elevated – 5.33±0.41 mmol/L ($p<0.001$), compared to the control values (3.12±0.32 mmol/L).

The mean total bilirubin concentration (**Figure 4 B**) in intoxicated calves increased. In group I the concentrations were 11.36±1.82 μmol/L ($P<0.001$), compared to the control determinations – 4.61±0.37 μmol/L.

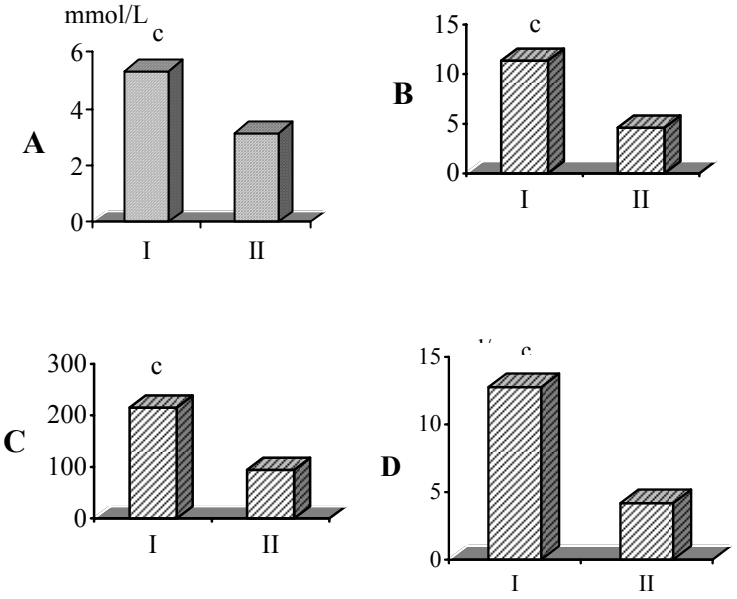

Figure 4. *Change in some biochemical parameters - blood glucose (A), total bilirubin (B), creatinine (C) and urea (D) in calves after intoxication with Poison Hemlock (Conium Maculatum) (Group I) and controls (Group II). Level of significance vc controls:* $^{a}p<0.05$; $p^{b}<0.01$; $p^{c}<0.001$.

The mean creatinine concentration (**Figure 4 C**) in calves with intoxication (group I) was significantly elevated – 214.7±18.9 μmol/L ($p<0.001$), compared to the controls – 94.1±8.2 μmol/L.

The mean urea concentration (**Figure 4 D**) in calves from group I was significantly higher – 12.77±1.98 mmol/L ($p<0.001$), *vs.* the control group – 4.17±0.52 mmol/L.

The changes in activity of aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase, creatinine kinase and alkaline phosphatase in calves after intoxication with Poison Hemlock (*Conium Maculatum*) are presented in **Figure 5**.

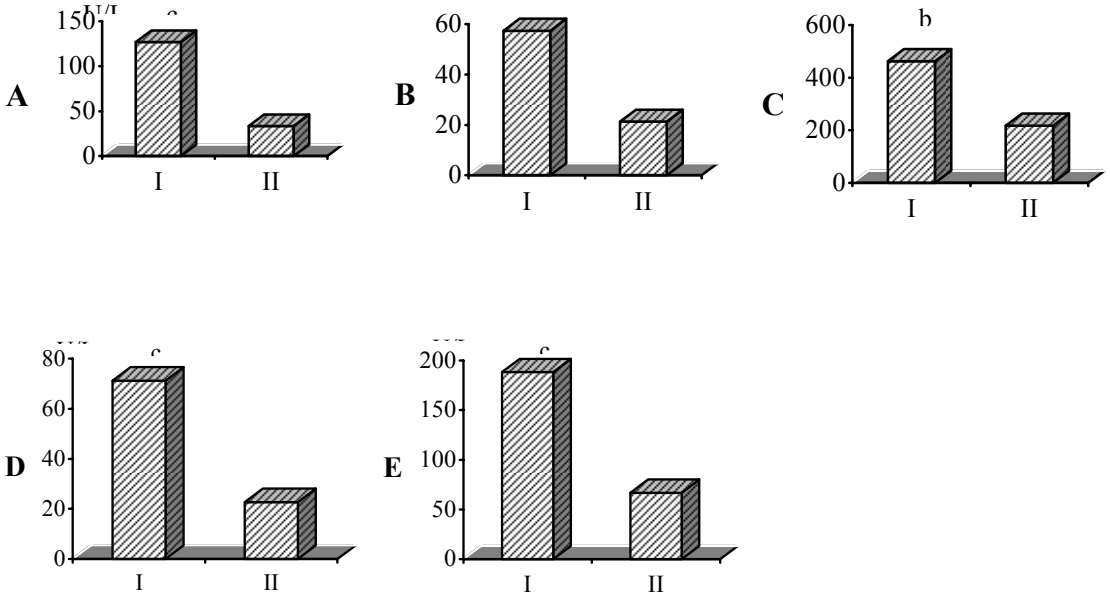

Figure 5. *Change in activity of aspartate aminotransferase (AST) (A), alanine aminotransferase (ALT) (B), lactate dehydrogenase (LDH) (C), creatinine kinase (CK) (D) and alkaline phosphatase (AlP) (E) in calvses after intoxication with Poison Hemlock (Conium Maculatum) (Groups I) and controls (Group II). Level of significance vs. controls:* $^{a}p<0.05$; $p^{b}<0.01$; $p^{c}<0.001$.

Mean aspartate aminotransferase (AST) activity (**Fig. 5 A**) was higher in calves from group I – 126.9±9.3 U/L ($p<0.001$), compared to controls (33.5±6.6 U/L).

Mean alanine aminotransferase (ALT)

activity (**Fig. 5 B**) in calves with intoxication (group I) was increased – 57.4±6.7 U/L ($p<0.001$), vs. the controls group (21.3±2.1 U/L).

The mean activity of lactate dehydrogenase (LDH) (Fig. 5 C) was significantly elevated in group I – 463±76.6 U/L ($p<0.01$), compared to controls calves – 217±41.2 U/L.

Mean creatinine kinase (CK) activity (**Fig. 5 D**) in calvses after intoxication with Poison Hemlock (*Conium Maculatum*) was increased – 71.2±8.1 U/L ($p<0.001$), compared to controls group – 22.7±4.4 U/L.
The mean activity of alkaline phosphatase (AlP) (**Fig. 5 E**) was significantly elevated in calves with intoxication (group I) – 188.3±31.5 U/L ($p<0.001$), compared to controls – 66.8±7.2 U/L.

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

IV. Morphological studies

Despite the therapy (see below), three calves died after 10-12 hours post intoxication.

Several hours (1-2 h) prior to the lethal issue in calves, we observed the strong tympani, nasal effluents, cyanosis of conjunctiva, hypothermia (35 - 35.5^{0}C) and asphyxia convulsions.

The patho-anatomical study showed dark and thick blood in abdominal and pectoral cavity. The mesenterial blood vessels were highly hyperaemic, and on the mesenterium and intestinal serosa, petechial haemorrhages were present. Mesenterial lymphonodes were oedematous, with a moist rose-reddish cross section and single haemorrhages. The liver was congestive with a brown colour and, in some areas the enlarged and blood-filled central veins of the lobules were clearly distinguished. The mucosae of the spleen, kidneys, epicardium and the endocardium of both atria exhibited petechial and more extensive haemorrhages. The lungs were strongly reduced in size, hyperaemic, with increased density, congestive and dark; the imprints where the ribs were in contact were clearly visible. The accretion of lungs with pulmonary walls was observed in many sections. The heart was with soft consistency and the right ventricle and auricle were filled with blood. The left half of heart does not contain blood. The meninges were strongly hyperaemic.

V. Treatment

In the calves with intoxication, a single gastric lavage with suspension of 0.5% of activated charcoal was performed via a naso-oesophageal intubation. Half an hour later, a 10% solution of $MgSO_4$ (Biovet, Pestera, Bulgaria) - 1.5 g/kg and 1% solution of activated charcoal - 1.5 g/kg was administered via a naso-oesophageal intubation.

To change the foamy timpani in gas we applied once the product Sicaden (Biovet, Pestera, Bulgaria) in dose 50 ml with 3L water for each calf with intoxication symptoms. The preparation was administered via a naso-oesophageal intubation. After 1 hr drilling was made to evacuate the collected gases. Against the alkaline rumen content we used 6 to 10 g lactic acid, depending on the weight of animals. Simultaneously we used oral Rumenotonicum (Biovet, Pestera, Bulgaria) 80 to 100ml to restore rumen movement. The two products were diluted in 3 L water, applied only once for each animal with intoxication symptoms and were administered via a naso-oesophageal intubation. For restoration of normal rumen microflora we applied oral 100 ml Natrium propionicum (Ursopron, Biovet, Pestera, Bulgaria), diluted in 2 L water.

A single bloodletting was done in all calves with intoxication. The amount of blood was calculated according to the body weight – on the average - 10 ml/kg. Intravenous administration of 2litre Ringer solution (Balkanpharma, Troian, Bulgaria), 5 g ascorbic acid (Vitamin C, Biovet, Pestera, Bulgaria), 0.5 g thiamine (Vitamin B_1, Biovet, Pestera, Bulgaria), 50 ml sodium N-acethyl methionine (5 % Metionin, Sinaps, Veliko Tarnovo, Bulgaria) and 10 ml 20% caffeine sodium benzoate (Biovet, Pestera, Bulgaria) was applied. For restoration and reinforcement of diuresis, the medication therapy was extended by intramuscular administration of Furosemide (in 2 ml injectable solution) – 0.5 mg/kg body weight.

VI. Prophylaxis

To avoid future cases of intoxication with Poison Hemlock (*Conium Maculatum*), we advised the owner of calves to avoid the pasture with plenty of this toxic plant. It is better in the morning to give calves the concentrate feed to obviate the need for animals to eat Poison Hemlock (*Conium Maculatum)* due to scarcity of non-poisonous fodder. The morning pasture is better to be implemented at later hours, because the

presence of dew favours the reinforcement of toxic influence of Poison Hemlock (*Conium Maculatum)* on the calves.

DISCUSSION

The toxic effect of Poison Hemlock (*Conium Maculatum)* is due to the action of its alkaloid contents (2, 3, 5, 7, 10 - 12, 14 - 16, 18, 19, 22 - 24, 26, 27, 38, 39, 50, 57).

The alkaloids of toxic plants affect the spin-brain reflexes, depress the autonomous activity and provoke the nerve-muscular blockade; as a result the activity of the spinal cord is blocked and the reflexes through it are stopped (3, 10 - 12, 19, 22 - 24, 38, 39, 50, 57). Apart from the central effect the alkaloids possess also peripheral brain influence, expressed in high selective paralysis of the motor ends of skeletal musculature and peripheral blockade of parasympathetic and sympathetic ganglia (2, 5, 7, 14 - 16, 18, 24, 26, 27, 50, 57). As a result of this effect we observed symptoms of groaning, moaning, teeth grinding, disturbances in locomotion, muscle cramps, ataxia, hyporreflexia, dermal anaesthesia and convulsions (1, 14, 16, 21, 26, 29, 41, 49, 51, 52, 55).

The established hyperthermia in calves with intoxication is probably the result of depression of the thermoregulatory centre, consequences of hypoxia provoked by alkaloids on the brain centres (10 - 14, 21, 22, 26, 29, 39, 44, 49, 50, 52, 54). The results for presence of hypothermia correlated with the results from our previous investigations (8), as well as the results of other authors (10, 12, 14, 39). The observed hyperthermia is very interesting. On the other hand it could be a result of the fact that alkaloids provoke peripheral vasoconstriction (1, 4, 7, 10 – 16, 19, 21, 26 - 29, 35, 37, 39, 49, 50). The lack of pathological data for inflammatory processes in calves with lethal issue confirms that hyperthermia resulted from impaired thermoregulation, correlating with strongly hyperaemic meninges.

The increased heart rate we observed and confirmed in our previous observations, as well as by other authors (1, 10 - 12, 14, 16, 19 - 21, 26, 29, 39, 49) is due to the initial stimulation of sympathetic ganglia and later – depression and paralysis of parasympathetic ganglia (10 - 14, 21, 22, 44, 50, 52, 54). In addition to possible reasons for tachycardia it is shown that presence of hypoxia correlated with cyanosis (54), and animal organism reacted with compensatory acceleration of the heart rate. The observed changes correlated with observed pathological alterations in hearth.

The observed polypnoea with dyspnoea in accordance with data from literature (54) is probably a consequence of depression of the respiratory centre in brain and blocking of N-choline receptors of the diaphragm nerves (10 - 12, 14, 17, 19, 21, 54) and correspond to pathological changes in lung parenchyma – the strongly congestive hyperaemia and reduced size with increased density.

The alkaloids of Poison Hemlock (*Conium Maculatum)* have high selective ability to paralyse motor ends and as result – the contractions of rumen are blocked (3, 16, 19). This is clinically expressed with high-level atony and lack of defecation. After that the rumen content becomes putrefactive. This is due to a combination of established alkaline pH of the content and quick gas generation leading to appearance of foamy substances.

The paralysed activity of alkaloids appears with constriction of urinary bladder sphincter resulting in accumulation of urine, correlating to observed anuria (3, 8, 14, 16, 53). The observed functional changes in urinary process are explained by some authors as morphological changes in kidneys (3, 14, 46, 52, 53), correlated with myoglobinuria (3, 52) rabdomyolysis and myoglobinuria (3, 52, 53).

The toxic effect of Poison Hemlock (*Conium maculatum)* is supplemented by changes in the values of studied haematological parameters – erythrocytosis, leukocytosis, regenerative left shift neutrophilia, lymphopaenia, eosinopaenia, increased haematocrit values.

The data regarding the increased haematocrit could be probably attributed to the amount of dehydration, resulting from the less level of liquids (8, 14). High haematocrit levels were responsible for producing higher RBC counts (relative polycythaemia) and haemoglobin content in intoxicated horses. The data about the changes in total WBC counts and the percentages of leukocyte classes in equine blood after intoxication with Poison Hemlock (*Conium Maculatum)* showed elevated total WBC counts, elevated neutrophils and segmented neutrophils, reduced lymphocyte percentages and absence of eosinophils. The leukocytosis, neutrophilia, lymphopaenia and aneosinophilia are all typical for what we call a “stress leukogram” due to excessive endogenous cortisol release. This is one of the possible causes for blood glucose elevation. The alkaloids of this toxic plant provoke peripheral vasoconstriction (1, 7, 10 – 16, 19, 21, 26 - 29, 35, 37, 39, 49, 50),

and the blood redistribution occurs, expressed by erythrocytosis, leukocytosis, regenerative left shift neutrophilia and increased catecholamines.

After Poison Hemlock intoxication, tympania are observed (3, 16, 19), because of the paralytic effect of plant alkaloids (2 - 5, 7, 9, 10, 12, 14, 19, 21, 22, 25, 26, 35, 50, 52).

The signs of CNS (10, 55) could be related to increased catecholamines (epinephrine and dopamine) amounts, 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 (14), as the glycogen released by hepatocytes is metabolised to glucose. A third possible explanation for higher blood glucose could be the inhibited function of the pancreas, correlating to reduced insulin production (14).

The blood biochemical analysis revealed increased total bilirubin concentrations as well as increased aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), creatinine kinase (CK) and alkaline phosphatase (AlP) activities. These changes corresponded to the observed changes in liver parenchyma, following Poison Hemlock intoxication. These results are similar to those reported by other authors (10, 14, 55) and support the hepatotoxic effect of Jimson weed alkaloids.

The established increased levels of creatinine and urea in our studies and others established by other authors, e.g., rabdomyolisis (52, 53), correlated with myoglobinuria (52) and functional changes in urinary process (anuria).

The lethal end of intoxication with Poison Hemlock (*Conium Maculatum)* in calves is a result of simultaneous central and peripheral activity of alkaloids, which cause the hypo and anoxaemia in the respiratory centre of the brain; and the respiratory activity is depressed or stopped (2, 3, 5, 7, 14 - 16, 18, 22 - 24, 26, 38, 50). On the other hand the alkaloids paralyse the diaphragm nerves and paralysis of respiratory muscles is observed (10 - 12, 18, 19, 22, 23, 26, 27, 38, 39, 57).

CONCLUSIONS

The clinical signs manifested after intoxication with Poison Hemlock (*Conium Maculatum)* in calves were: hyperthermia, tachycardia, polypnoea with dyspnoea rumenal alkalosis, complete atony with strong tympany, mydriasis, complete refusal of feed, lack of defecation and urination, hyper salivation, lack of thirst, diffusely reddened conjunctivae, eyelid dropping, groaning, grinding with teeth, disturbances in locomotion, muscle cramps, ataxia, hyporreflexia, dermal anaesthesia and convulsions.

The Poison Hemlock intoxication in calves was characterized, complete with changes in some haematological indices – hyperchromaemia, erythrocytosis, leukocytosis, regenerative left shift neutrophilia, lymphocytopenia, aneosinophilia and increased haematocrit values.

Blood biochemical changes consisted in hyperglycaemia, bilirubinaemia, increased urea and creatinine values and increased activities of aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), creatinine kinase (CK) and alkaline phosphatase (AlP).

The pathomorphological studies showed an extensive hyperaemic and haemorrhages in the lungs, kidneys, liver and myocardium.

In therapeutic scheme we recommend the application of electrolytic solutions (dehydration), diuretics for forced diuresis (anuria), rumen tonics (atony), antifermentation (strong tympany) medicines and products for correction of rumen content pH (rumenal alkalosis). Because of the presence of hyperglycaemia we do not recommend the use of glucose in animals with Poison Hemlock intoxication.

The applied schedule of treatment is effective in the initial stage of the intoxication.

The prognosis is more favourable, if during the recovery from intoxication urination and frequent defecation are observed.

The clinical signs, haematological and biochemical indices in blood, morphological alterations and the effect of the treatment in Poison Hemlock intoxication are directly related to the stage of intoxication.

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