

THE CHANGES OF KIDNEY STATUS IN HORSES WITH ACUTE FUNGOUS POISONING

Golovakha V.I., PhD
Zhyla I.A., assistant
Kutsan O.T., PhD

Bila Tserkva State agrarian university, Bila Tserkva

Institute experimental and clinical veterinary medicine, Kharkiv

The kidney failure in horses with acute fungous poisoning are characterized by hypercreatinemia, increasing GGT activity in urine, changes urine and blood ratios (Cr_u/Cr_s , CTR, U_u/U_s), pathological urine sediment (erythrocytes, renal epithelium, granular and hyaline casts) and histological changes.

Last ten years in Ukraine more and more attention is given to development of horse breeding. But the successful conducting this field mostly depends on qualitative forages basis. Therefore forages production must be directed on qualitative stocking up and correct preparation it for horse feeding. But on many horse farms preparation of forages and its savings do not give due attention that results to its contamination by microscopic fungous – micromycetises. High mycotoxicity of the forages (more than 40 thousands spores in 1 gramme of the forages) promotes intoxication [1] and infringement of functions of many systems and organs, including kidneys, which changes reveal more often postmortally in. The forages poisonings are described in most cases in cattle, pigs and chicken [2–5]. In horses with poisoning the changes of renal system are poorly investigated. Therefore purpose of our work was the studying of a kidney status of in horses with alfalfa hay poisoning and recognizing most informative tests for the diagnosis of kidney failure.

Material and methods. The object of research was the 2-3 years ages race thoroughbred offspring horses, which were fed by alfalfa hay. The hay was contaminated by micromycetises (*Penicillium* spp., *Aspergillus restrictus*, *Alternaria alternata*, *Cladosporium herbarum*, *Myrothecium* spp., *Rhodotorula rubra*, *Rhizopus nigricans*, *Fusarium sporotrichiella*, *Aspergillus amstelodami*, *Aureobosidium pullulans*, *Fusarium avenaceum*, *Fusarium sambucinum*), total count of the fungous spores were 4.800 thousands in 1 gramme.

Functional kidney status was studied on the parameters: urea, creatinine and GGT activity in urine and blood serum. We also counted up indexes of these parameters: the coefficient of the tubular reabsorption (CTR), the ratio of creatinine in urine to blood serum (Cr_u/Cr_s), the ratio of urea in urine to blood serum (U_u/U_s), ratio of the urea nitrogen to creatinine (U_N/Cr).

We also studied urine sediment and postmortem changes in kidney (histological).

Results of research. The clinical signs in thoroughbred horses were: depression, anorexia, diarrhea (sometimes with some blood), colic. In one horse the poisoning proceeded in the very severe form and, except total signs, marked signs of urine colic (often desires to micturition but at the same time noted oliguria and hematuria). The temperature in this animal raised up to 39,4°C. In 42 hours after occurrence of the first clinical signs of the poisoning this horse has died with encephalopathy.

The essential changes were revealed in the renal system, that confirms high creatinine level in blood serum. It was $413,3 \pm 106,3$ mkmol/l, that was in 2,7 times more than healthy horses (tab. 1). At the same time creatinin levels in urine had the tendency to decreasing. The average creatinine excretion was $17345,0 \pm 3249,0$ mkmol/l that on 6,8 % less than in healthy animals. The ratio of creatinine in urine to blood serum in sick horses was in 3,9 times lower than in healthy animals. In 40 % of the sick horses this ratio pointed on the kidney damages, in others - on prerenal failure. In one horse (with severe poisoning) the creatinine level in blood serum achieved 837,4 mkmol/l at simultaneously low its excretion with urine. Accordingly Cr_u/Cr_s in this horse was lowest. That is in horse with severe poisoning at the first day after occurrence of clinical signs was marked attributes of the damages of the renal glomerule and tubules.

The estimation of a status of the renal tubules is impossible without calculation of the coefficient of tubular reabsorption [6]. In horses with poisoning it was low, but reliability difference with healthy horses was not. Lowest CTR was in horse with severe form of a poisoning. On our thought,

Table 1 – The creatinine levels and its ratios in horses with poisoning (n=42, M±m)

The groups of horses	Creatinine, mkmol/l		Cr _u /Cr _s	CTR
	blood serum	urine		
Healthy	125,7 – 173,7	11283,0 – 26269,0	77,6–209,0	98,7–99,5
	151,5±11,1	18611,0±3740,0	125,7±26,7	99,1±0,2
Sick	265 – 837,4	10759,0 – 24062,0	13,1–81,1	92,5–98,8
	413,3±106,3	17345,0±3249,0	51,8±12,7	97,1±1,2
p<	0,05	0,5	0,05	0,5

the level of the tubular reabsorption is enough stable value and it decreasing even on 0,2-0,3 % below minimum norm (98,8 %) is disadvantageous sign.

If the creatinine level had essential changes, the values of other component of residual nitrogen – urea, at the majority of animals were without changes. In 56 % of the sick horses its contents both in blood, and in urine remained in norm. Only in one animal the urea level in blood serum increased to 26,7 mmol/l (tab. 2). In this horse U_u/U_s was low (about 22,0) that specified on the renal azotemia. In other animals U_u/U_s was in norm (60,2–86,6). Obviously, the urea level in blood serum does not display the renal failure on the early stages due to perfect compensative system of kidneys. It is confirmed by our investigations, as a blood selection was made at the first hours after a poisoning (when we observed clinical signs in animals) and the urea level had not enough time to change in the majority of animals. Its increasing had revealed only in one horse with severe poisoning (in 12 hours after blood collection this horse has died).

Table 2 – The urea levels its ratios in horses with poisoning (n=42, M±m)

The groups of horses	Creatinine, mkmol/l		U _u /U _s	UN/Cr
	blood serum	urine		
Healthy	6,4–6,8	400,8 – 629,1	58,9–93,9	8,9–13,4
	6,7±0,1	516,1±45,9	77,5±7,7	11,1±0,9
Sick	4,6–26,7	348,9–597,7	22,0–86,6	3,0–7,7
	9,8±4,2	468,3±62,1	66,7±12,8	5,3±0,9

In that cases, when it is impossible to get urine from the horses it is possible to count up the ratio of urea nitrogen to creatinine in blood serum [7]. The average value of this ratio in sick horses decreased and was 5,3±0,9 that is confirmation of acute renal failure. But this index not always informative for the diagnosis of renal failure, as in the animal with severe poisoning it was in norm.

Thus, our investigations show that the urea level in blood serum is not early test for the kidney damages, as well as creatinine levels, which can increase at massive defeats of muscles; including heart (the pathologic-morphological studies are revealed multiple diapedesis hemorrhages). Therefore for early diagnosis of the renal failure should be used indexes of urine and blood serum (Cr_u/Cr_s, CTR, U_u/U_s, and UN/Cr).

The renal failures and changes of the urine parameters, especially urea, change kidney concentrating property. It can be confirmed by low urine density which was observed in 44 % sick horses.

The presence of the pathological elements in urine sediment pointed on renal failure as well as histological studies. We observed changes in cortical and medullar kidney layers. Turbid swelling and protein granular dystrophy of the epithelial tubular cells were revealed in cortical kidney layer. These changes promoted to formation of hyaline casts and its appearance in urine sediment.

The tubular gleam was filled by amorphous protein substance, in the structure which was revealed single shedding epithelial cells. Among convolute tubules were observed singular and sometimes multiple diapedesis hemorrhages.

The signs of acute passive congestive and the blood presence in straight tubules of the medullar kidney layer were observed. Clinically it was displayed by macrohematuria. The epithelial cells of the straight tubules were with protein granular dystrophy. There were amorphous protein substances with shedding epithelium in its lumen which we could find in urine sediment.

The damages of kidney structure were specified by high GGT activity in urine. Its average was $2,44 \pm 1,60$ mkkat/l that is in 13 times higher than healthy horses (tab. 3). The enzyme increasing was observed in 80 % of sick horses. At the same time GGT activities in blood serum was increased only in 20 % sick animals. That shows its poor significance in blood for the diagnosis of kidney failure.

Table 3 – The GGT activities in horses with poisoning (n=42, M±m)

The groups of hotses	GGT, mkkat/l	
	blood serum	urine
Healthy	$0,31 \pm 0,03$	$0,19 \pm 0,08$
	0,33–1,25	0,67–8,74
Sick	$0,66 \pm 0,22$	$2,44 \pm 1,6$
p<	0,5	0,05

Conclusions.

1. The earliest tests for diagnosis of kidney failure in horses with acute poisoning are ratios: Cr_u/Cr_s , CTR, U_u/U_s , hypercreatinemia, high GGT activity in urine and pathological urine sediments (erythrocytes, renal epithelium, granular and hyaline casts).

2. Less informative tests for the early stage of kidney pathology are the urea level and UN/Cr.

3. Histological studies of the kidneys in horses with acute fungous poisoning are characterized by protein granular dystrophy in cortical and medullar renal layers, acute passive congestive and diapedesis hemorrhage.

The literature list

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ИЗМЕНЕНИЯ ФУНКЦИОНАЛЬНОГО СОСТОЯНИЯ РЕНАЛЬНОЙ СИСТЕМЫ У ЛОШАДЕЙ ПРИ ОСТРОМ ОТРАВЛЕНИИ ГРИБАМИ

Головаха В.И., канд. вет. наук, доцент
Жила И.А., асс.
Куцан А.Т., канд. вет. наук

Белоцерковский государственный аграрный университет, г. Белая Церковь
Институт экспериментальной и клинической ветеринарной медицины, г. Харьков

Резюме

Изменения почек при остром кормовом отравлении лошадей характеризуются гиперкреатинемией, изменением индексов мочи и крови (отношение креатинина мочи и крови, отношение мочевины мочи и крови, коэффициент канальцевой реабсорбции), повышением активности ГГТ в моче, появлением элементов патологического осадка мочи (эритроциты, почечный эпителий, гиалиновые и зернистые цилиндры) и гистологические изменения.