Acta Scientiae Veterinariae, 2026. 54(Suppl 1): 1085.

CASE REPORT
Pub. 1085

ISSN 1679-9216

Uremic Encephalopathy in Horses

Filipe Simeão Fröhlich Klug, Raquel Yvonne Arantes Baccarin, André Luis do Valle De Zoppa & Carla Bargi Belli

ABSTRACT

***Background*:** Uremic encephalopathy refers to neurological manifestations resulting from renal failure. There are reports of this condition in several animal species. Regardless of the species, it may occur in association with acute or chronic cases of renal failure. There are almost no reports or studies in the literature on horses. Specific publications are limited to individual reports and a survey of 5 cases over a 20-year period, in addition to citations in books and reviews on renal alterations in this species. The objective of this study is to report 6 cases of uremic encephalopathy in horses, relating the manifestations to the underlying condition, laboratory results and outcome of the case.

***Cases*:** Six cases of uremic encephalopathy were identified, representing 0.1% of the total number of equine cases treated between 2008 and 2023 (6,200) at the Veterinary Hospital of FMVZ-USP, of different breeds and ages, related to both acute and chronic cases of renal failure, with a predominance of acute cases. The clinical manifestations, primary conditions, laboratory values of urea and creatinine close to neurological manifestations, and case outcome are described. Regarding the underlying causes, in our cases, we found an association with acute renal failure during the course of colic syndrome in 3 animals (1 due to excess non-steroidal anti-inflammatory drug for 14 days), 1 related to a case of rhabdomyolysis and 1 with chronic renal failure. In the others and also in the previous ones, several factors contributed to the renal injury, such as excessive use of non-steroidal anti-inflammatory drugs and/or in combination with dehydration. The 6 cases presented the same set of manifestations reported for humans and animals, with a predominance of mental state alterations, which appeared in 5 of the 6 horses. Treatment was based mainly on fluid therapy, correction of electrolyte imbalances and use of anticonvulsants when necessary. Five cases died and 1 was discharged from hospital, unrelated to the degree of azotemia, but with milder manifestations. Excluding the animal that was discharged from hospital, histopathology of the brain was performed in only 2 of the 5 animals that died, with findings compatible with those of other species, but without the findings in astrocytes reported for other cases in horses.

***Discussion*:** The cases presented a pattern similar to that described in the literature for horses and other species, both in relation to the findings and the historical information. Our cases presented variation in urea and creatinine values, but all were quite high and without significant improvement with fluid therapy, indicating the presence of intrinsic renal injury. It is important to remember that in addition to the initial condition and the possibility of previous renal disease without manifestations, the abusive use of medications that are harmful to the kidney and failure to deal with dehydration may contribute to the occurrence of renal injury or worsening of preexisting conditions, and may favor the occurrence of neurological manifestations associated with renal failure. It is concluded that uremic encephalopathy in horses is rare, with no predisposition to breed, sex or age, and may be associated with acute and chronic cases of renal failure of various origins, with no direct relation to the severity of azotemia, with a poor prognosis. Therefore, it is necessary for veterinarians to be aware of the possibility of this condition for prompt diagnosis and differentiation with other situations that can cause similar manifestations and concomitant with renal failure.

Keywords: azotemia, fluid therapy, kidney disease, renal, uremia.

DOI: 10.22456/1679-9216.147519

Received: 4 October 2025 Accepted: 12 February 2026 Published: 28 March 2026

Programa de Pós-Graduação em Clínica Veterinária (PCVet), Faculdade de Medicina Veterinária e Zootecnia (FMVZ), Universidade de São Paulo (USP), São Paulo, SP, Brazil. CORRESPONDENCE: F.S.F. Klug [filipe.medico.veterinario@gmail.com]. Departamento de Clínica Médica, FMVZ - USP. Avenida Professor Doutor Orlando Marques de Paiva n. 87. Cidade Universitária. CEP 05508-270 São Paulo, SP, Brazil.

INTRODUCTION

Uremic encephalopathy refers to neurological manifestations resulting from renal failure [13]. It is reported in several animal species, including humans, where it is most studied and known.

There are reports, albeit isolated, in cats [6], dogs [2], goats [12], cattle [7], monkeys [9], horses and others. Regardless of the species, it may occur in association with acute or chronic cases of renal failure.

There are almost no reports or studies in the literature on horses. Specific publications are limited to individual reports [1,3] and a survey of 5 cases over a 20-year period [4], in addition to citations of the possibility of the condition in books and reviews on renal alterations in this species [7,10].

The low occurrence of these conditions and the lack of knowledge by clinicians can lead to delays in recognition, with the manifestations often being initially related to other causes, especially when there is an underlying disease outside the urinary system.

The objective of this study is to report 6 cases of uremic encephalopathy in horses, relating the manifestations to the underlying condition, laboratory results and outcome of the case, contributing to the knowledge of this condition for more prompt recognition and treatment in the species.

CASES

Among the total number of cases treated between 2008 and 2023 (6,200) at the Veterinary Hospital (HOVET) of the Faculty of Veterinary Medicine and Animal Science (FMVZ) of the University of São Paulo, 6 cases of uremic encephalopathy were identified (0.1% of the total).

The animals came from different farms in the state of São Paulo, Brazil. Information regarding the identification of the animals, initial clinical presentation, clinical manifestations, urea and creatinine levels close to the neurological presentation, and case outcome were collected, with necropsy evaluation, when performed.

The information on breed, sex, age, and primary complaints and conditions of the animals are presented in Table 1. The results of urea and creatinine levels close to the neurological manifestations are presented in Table 2. The neurological clinical manifestations, treatment instituted, and case outcome are shown in Table 3.

Table 1. Information on identification and condition / primary complaints of animals.

Case	Breed	Sex	Age (years)	Primary condition/complaints
1	Mixed race	male	23	Colic syndrome (enterolith) and apathy
2	Andalusian	male	15	Chronic renal failure, pneumonia/chronic hyporexia, weight loss, decubitus
3	American Trotter	female	13	Acute renal failure, myositis / Hyporexia, weight loss
4	Andalusian	female	1.5	Colic syndrome, Renal failure
5	American Trotter	female	3	Colic syndrome
6	Mangalarga	male	7	Acute renal failure / apathy, hyporexia, difficulty walking

Table 2. Presentation of serum biochemistry variables (mg/dL) and the Urea:Creatinine ratio close to neurological manifestations.

Variables	C1	C2	C3	C4	C5	C6	Reference
Urea	122.9	639.0	260.5	380.0	183.8	239.1	30 - 40
Creatinine	3.0	10.9	8.0	13.29	3.1	8.2	1 - 2
Urea/Creat	40.97	58.63	32.56	28.59	27.0	29.1	

Urea/Creat: Urea/Creatinine.

Table 3. Neurological manifestations, treatment instituted and cases outcome.

Case	Manifestations	Treatment	Outcome
1	Incoordination, decreased proprioception, apathy, dementia, with loss of threat response, seizure	Fluid therapy, diazepam	Death
2	Proprioception deficiency, ataxia and reduced panniculus reflex, dementia	Fluid therapy	Death
3	Nystagmus when walking, kyphosis, difficulty flexing the neck	Fluid therapy	discharge
4	Apathy, depression	Fluid therapy	Death
5	Delirium, compulsive walking, followed by recumbency	Fluid therapy	Death
6	Apathy, dementia, followed by agitation	Fluid therapy, sedation	Death

In addition to what is stated in the tables, the following information is worth highlighting:

Case 1 **(C1):** Horse was treated with intermittent colic syndrome for 14 days. Before being treated at HOVET, in addition to other treatments (fluid therapy, magnesium sulfate, vitamin complex and calcium), was medicated with flunixin meglumine[1], at the correct dose [Banamine® 50 mg/mL - 1.1 mg/kg but BID instead of SID, for all 14 days]. Two days before being referred, presented decreased appetite. At HOVET, presented a heart rate (HR) of 80 BPM, respiratory rate (RR) of 36 MPM, rectal temperature (RT) of 39.7ºC, congested oral mucosa with halo, capillary filling time (CFT) of 3-4, brownish urine, in addition to neurological manifestations. Initial treatment for colic and fluid therapy were provided. The neurological manifestations worsened and within a few hours he began to have seizures (Figure 1) and died. The necropsy revealed an enterolith in the left dorsal colon without major alterations in the intestinal wall, kidneys with a brownish coloration and whitish areas and soft consistency. The histopathological examination diagnosed moderate chronic interstitial nephritis, mild diffuse global membranous glomerulonephritis and severe tubular nephrosis. The central nervous system was not evaluated.

Case 2 **(C2):** The animal was treated due to decreased appetite for 4 months, which worsened 2 weeks ago, respiratory infection for 20 days, prolonged penile exposure for 1 week and, for 1 day, anorexia, difficulty drinking water, moving and standing up. Upon examination at HOVET, the animal presented a low Body Condition Score (BCS), HR: 76 BPM, RR: 40 MPM, hyperemic mucous membranes, other parameters within normal limits, in addition to neurological alterations. It was treated with fluid therapy lactate[2] [Ringer Lactato® - 3 mg/mL i.v. SD] and glucose 5 %] and glucose[2] [Solução Glicosada® - 50 mg/mL i.v. SD]. Upon admission, the neurological condition worsened and it died within a few hours. The necropsy identified pneumonia, kidneys with an irregular surface, marked decrease in the corticomedullary relationship, opaque meninges adhered to the brain. Histopathology revealed proliferative glomerulonephritis, associated with foci of glomerulosclerosis, moderately degenerated proximal tubules, moderate to severe multifocal

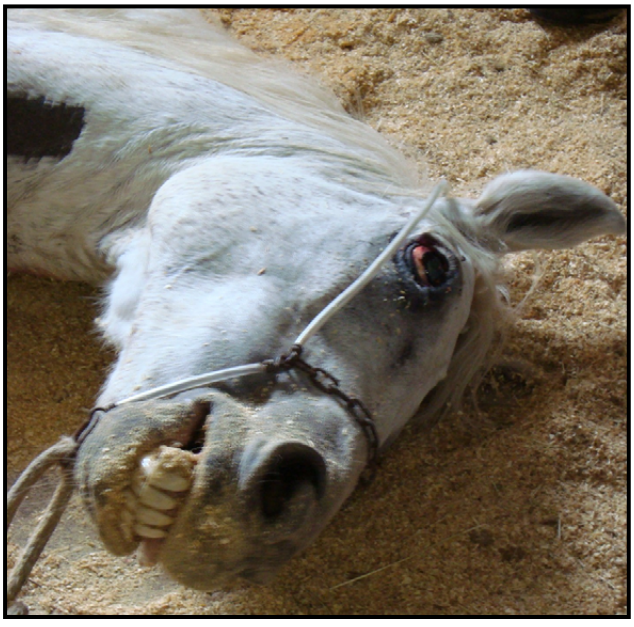

Figure 1. *Case 1*. Moment of convulsion prior to death.

chronic interstitial nephritis; and brain and cerebellum with mild edema and congestion.

***Case 3* (C3):** The history shows that the animal participated in a long-distance walk 30 days ago, with hyporexia for 10 days, during which it received enrofloxacin[3] [Enrofloxacin MND SD p.o.] and a single dose of flunixin meglumine[3] [Flunixin MND - 50 mg/mL, SD]. Upon admission, the following were observed: HR 48 BPM, RR 42 MPM, hyperemic ocular and pink oral mucosa, low ECC and other parameters without alterations. It presented manifestations of myositis/rhabdomyolysis (generalized muscle sensitivity), kyphosis, difficulty in flexing the neck, and nystagmus after walking in a circle. The urinalysis also showed alterations (low density, proteinuria, myoglobinuria). CSF was collected, but it showed no alterations in the laboratory evaluation. During admission, it was treated mainly with fluid therapy and glucose. The neurological manifestations and appetite gradually improved, as did the laboratory evaluations. Upon hospital discharge, 16 days later, the Urea concentration was 34 mg/dL and the Creatinine concentration was 2.4 mg/dL.

***Case 4* (C4):** Referral was due to abdominal discomfort for 3 days, with intermittent symptoms. During this period, in addition to other treatments, the animal received applications of dipyrone[4] [D-500® 500 mg/mL - 24.10 mg/kg, single dose] and flunixin meglumine[3] [Flunixin MND 50 mg/mL - 0.37 mg/kg, BID for 3 days]. Upon admission, HR was 60 BPM and RR was 12 MPM, with no changes in other parameters. Oral treatment for colon compaction was started at HOVET, in addition to IV fluid therapy, both for colic and azotemia. Motility returned slowly at first, but with subsequent worsening, with no return of appetite and worsening of apathy and depression, accompanying the worsening of the azotemia despite treatment. Due to the worsening of the renal condition, the owner opted for euthanasia. At this time, the urea value was 448 mg/dL and creatinine was 14.8 mg/dL. Necroscopy showed an enlarged kidney with a brownish coloration. Histopathology showed acute tubular necrosis with preservation of the basement membrane, brain with diffuse and discreet edema, cerebellum with moderate edema and vacuolization of white matter and necrosis of Purkinje cells.

***Case 5* (C5):** Horse was received with colic syndrome, and diagnosed with colon displacement. At HOVET, the animal underwent exploratory laparotomy to correct the displacement, which occurred without complications. The initial laboratory examination showed no azotemia. In the postoperative period, the animal received standard medication: gentamicin[3] [Gentamicin MND - 6.6 mg/kg, IV, SID], enrofloxacin[3] [Enrofloxacin MND - 5 mg/kg, IV, SID], flunixin meglumine[3] [Flunixin MND - 1.1 mg/kg single dose and then 0.25 mg/kg TID for 3 days], in addition to fluid therapy when necessary. The animal presented azotemia in the laboratory examination on the 5th postoperative day [urea 128.9 mg/dL and creatinine 3.7 mg/dL] and reduced appetite. At that time, the urinalysis showed proteinuria and the presence of a large quantity (3 crosses) of renal epithelial cells. In the following days, the hyporexia, diarrhea and polakiuria worsened, without significant improvement in the azotemia. Two days later, the patient presented with delirium, with compulsive walking (Figure 2), followed by recumbency and death. No *post mortem* examination was performed.

***Case 6* (C6):** Horse treated at HOVET with a history of participating in a long horse ride 4 days ago and feeling tired at the end of it. Afterwards, it presented apathy, appetite loss and pain when walking with increased temperature in the hooves. During this period, it was medicated with phenylbutazone, flunixin meglumine, meloxicam (doses not specified and MND[3]) and ice on the hooves. At HOVET, it presented tachycardia, tachypnea, intestinal hypomotility, reluctance to walk (but without increased temperature in the hooves and light digital pulse) and was oblivious to the environment (dementia), in addition to severe azotemia. It was treated with fluid therapy, but the following day its neurological condition worsened, with great agitation and death. At necropsy, the kidneys were more reddish in color. No histopathological examination was performed.

DISCUSSION

Although the condition is also uncommon in humans, there are indications that it may occur in 10 to 30% of cases of untreated or inadequately treated acute or chronic kidney disease [11]. In dogs, a survey showed that uremic encephalopathy was responsible for 2.1% of cases of seizures [2]. In the only survey in horses, the condition represented 1.5% of cases of kidney disease [4] and, in our survey, the caseload was also low, 0.1% of the total number of cases treated, showing that it is indeed quite uncommon, although a few additional cases may not have been counted

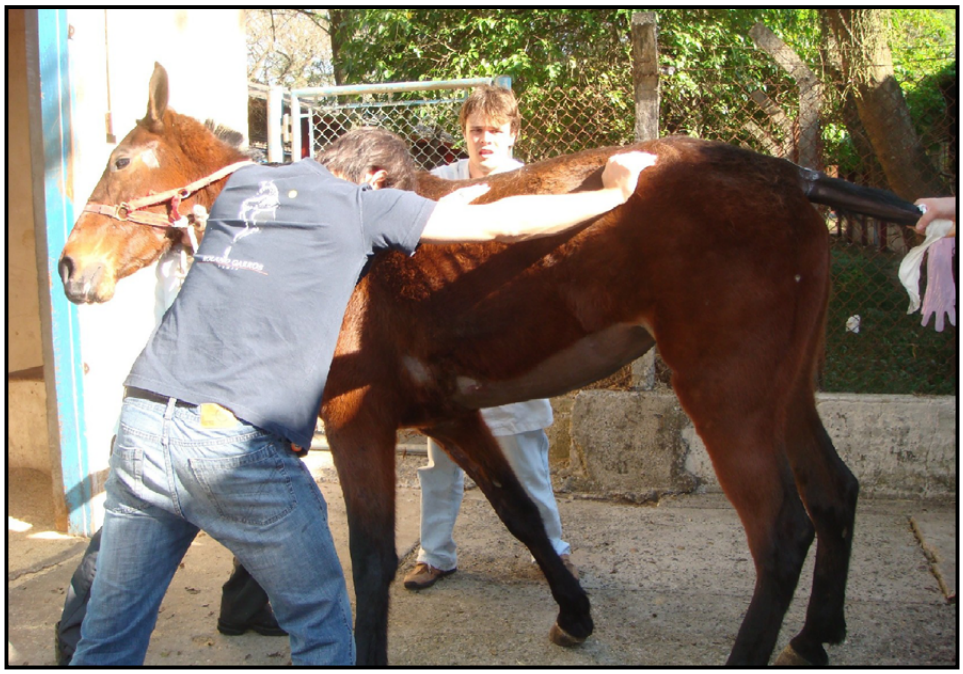

Figure 2. *Case 5*. A change in mental state to delirium is observed. The horse walked compulsively, requiring several people to restrain it.

because they presented very subtle manifestations and were not characterized as uremic encephalopathy in the medical records.

Neurological manifestations in cases of renal failure in humans occur due to a combination of several factors: retention of uremic solutes, changes in hormonal metabolism, alterations in electrolyte in electrolyte and acid-base homeostasis, as well as changes in vascular reactivity, transport across the blood-brain barrier, and inflammation [13]. In animals, including horses, it is not known whether all of these factors have the same degree of importance as in humans [8], as there is still a lack of research on the subject.

The cases of horses reported in the literature involve various breeds, aged between 2 and 20 years, both males and females, information similar to what we observed in our survey.

In humans, there are several manifestations related to uremic encephalopathy: spasms, nystagmus, lack of concentration, fatigue, memory loss, changes in alertness, convulsions, etc., which can lead to coma [12,13], the main ones being related to changes in mental state. It is also reported that the manifestations can be more severe in acute cases of glomerular filtration failure and more insidious in cases of slow progression. The low occurrence in chronic cases in humans may be due to the initiation of treatment and dialysis before neurological manifestations occur [13].

In animals, including the various species in which it is reported, the following are described: convulsions, behavioral changes, changes in mental state, tremors, ataxia, apathy, coma and death [2,3,6,9,12], associated or not with other manifestations of uremic syndrome, such as gingivitis with ulceration, weight loss, polyuria, polydipsia, anorexia, thick rough hair and lethargy [16] and possible associated diseases.

The 6 cases reported here presented the same set of manifestations as in humans and animals, with a predominance of mental state changes, which appeared in 5 of the 6 animals, but with a distribution between depression, dementia and delirium.

Although in humans the manifestations are generally generalized encephalic, there are rare reports of involvement of only the brainstem, with manifestations, for example, of palpebral ptosis and ocular myasthenia [14] and unilateral changes in the white matter [15]. Interestingly, in 1 of the cases described here, the main manifestation was nystagmus when walking, which may represent a predominant change in the brainstem, although not exclusive.

As in humans [13] and other species, the cases described here also occurred associated with both acute and chronic renal failure, but with a predominance of acute cases in relation to horses, unlike reports and surveys in humans, where a majority of chronic cases of renal failure are observed.

Regarding the underlying causes, we found an association with acute renal failure while colic syndrome in 3 animals (in C1, clearly due to excessive use of non-steroidal anti-inflammatory drugs and in 1, C5, which developed the condition in the postoperative period), 1 related to a case of rhabdomyolysis and 1 with chronic renal failure. In the others and also in the previous ones, several factors contributed to the renal injury, such as excessive use of non-steroidal anti-inflammatory drugs and/or in combination with dehydration. Furthermore, as also suggested in other cases [4], the animals could have had previous renal injuries, from various causes, but without failure until the time of the new injuries and worsening of the manifestations. It is important to remember that in addition to the initial condition, the abusive use of medications that are harmful to the kidney and failure to deal with dehydration may contribute to the occurrence of renal injury or worsening of pre-existing conditions.

In humans, as in animals, there are several conditions that can be confused with cases of uremic encephalopathy and may even occur concomitantly and contribute to the manifestations, such as hemolytic uremic syndrome, malaria, leptospirosis, sepsis, etc. [13]. Among several differential diagnoses are hypertensive encephalopathy and posterior reversible encephalopathy, both linked to a very large increase in blood pressure [13].

In the work with case surveys [4], cases associated with colic and endotoxemia were excluded. We chose not to exclude this, otherwise we would not be able to identify acute cases. However, we took care to verify whether the cases were really associated with kidney damage and not with severe electrolyte and acid-base alterations, hypovolemic or toxemic shock.

In humans, there is no gold standard for diagnosis, and the case can be closed by exclusion and retrospectively with the improvement of the neurological condition after dialysis or transplantation [13]. Even the values of nitrogenous urea compounds are not good indicators of the chance of developing neurological manifestations in humans, and there is no indicative value for the immediate start of dialysis [13]. In horses, some suggest that the finding of hyperammonemia without hepatic or intestinal alterations could help in the suspicion of uremic encephalopathy [4,8]. Although some MRI findings ("fork sign") are quite common for the condition, their absence does not exclude it, and the diagnosis should be made by analyzing the manifestations, laboratory and imaging findings together [17].

The cases in this study presented varying levels of urea and creatinine, but all were quite high and did not improve significantly with fluid therapy, indicating the presence of intrinsic renal injury. In our practice, we do not have the possibility of using magnetic resonance imaging to aid in diagnosis. Furthermore, many animals reported here would not have the conditions or time to undergo this evaluation, even if it were available, due to the rapidity and severity of the progression.

The histopathological findings of the kidneys vary according to the cause and progression, but severe tubular and glomerular lesions appear to be quite common, regardless of the species. Regarding the central nervous system, the findings appear to vary according to the species, although the number of reports in each species is too small to form a pattern. In goats, myelin vacuolization was reported in localized spots [12]. In a cat, there was vacuolization of the white matter of the basal ganglia and cerebellum, and Alzheimer type II astrocytes in the cerebral cortex and hippocampus [6]. In monkeys, the hippocampus showed necrosis and loss of neurons and glial cells, in addition to edema in the cerebral cortex and multifocal hemorrhage. Changes (edema) in astrocytes and the presence of Alzheimer type II cells have been reported in horses [1,4].

In our cases, excluding the 1 that was discharged from hospital, histopathology of the brain was performed in only 2 animals, with findings of edema in the brain and cerebellum in both, and vacuolization of white matter and necrosis of Purkinje cells in the cerebellum of 1 of them, compatible with some of the findings in other species, but without the findings in astrocytes reported for other equine cases.

Treatment in humans is mainly based on dialysis, although there are other strategies that can be adopted and/or associated with it, such as oral activated charcoal, hemoperfusion, nutritional treatment, correction of electrolyte and acid-base alterations, review of medications that may cause neurological manifestations [13], in addition to research into new drug treatments [18].

Blood dialysis is not available in horses. Although peritoneal dialysis has been described in this species [5], it was not used in any of our 6 cases or in those in the literature, but it may represent a valid

treatment attempt for future cases. Treatment was based mainly on fluid therapy, correction of electrolyte imbalances and use of anticonvulsants when necessary. Treatment was successful in only 1 animal, without any relation to the severity of azotemia, but this was a case with milder manifestations. Of the cases in the literature, success is reported in only 1 opportunity [3].

The prognosis of uremic encephalopathy with severe neurological manifestations is guarded to poor [7]. Of the 5 cases reported in a survey of the literature [4], all were euthanized or died, some within a few hours of hospital care, similar to what we saw in the cases that died in our survey.

It is concluded that uremic encephalopathy in horses under our care is also rare, with no predisposition to breed, sex or age. It may be associated with acute and chronic cases of renal failure of various origins, with no direct relation to the severity of azotemia, with a poor prognosis. Therefore, it is necessary for veterinarians to be alert to the possibility of this condition for prompt diagnosis and differentiation with other situations that may cause similar manifestations and concomitant with renal failure.

MANUFACTURERS

[1]Merck & Co., Inc. Rahway, NJ, USA.
[2]Fresenius Kabi Brasil Ltda. Aquiraz, CE, Brazil.
[3]Manufacturer Not Declared. São Paulo, SP, Brazil.
[4]Zoetis. São Paulo, SP, Brazil.

Declaration of interest. The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

REFERENCES

1 **Bouchard P.R., Weldon A.D., Lewis R.M. & Summers B.A. 1994.** Uremic Encephalopathy in a Horse. *Veterinary Pathology*. 31(1): 111-115: DOI: 10.1177/030098589403100116.

2 **Brauer C., Jambroszyk M. & Tipold A. 2011.** Metabolic and toxic causes of canine seizure disorders: A retrospective study of 96 cases. *The Veterinary Journal*. 187(2): 272-275: DOI: 10.1016/j.tvjl.2009.10.023.

3 **Fernandes T. & Robin M. 2025.** Metabolic encephalopathy associated with uraemic syndrome and hyperammonaemia in a horse presenting with renal failure. *Equine Veterinary Education*. 37(5): e85-e91: DOI: 10.1111/eve.14096.

4 **Frye M.A., Johnson J.S., Traub-Dargatz J.L., Savage C.J., Fettman M.J. & Gould D.H. 2001.** Putative uremic encephalopathy in horses: five cases (1978–1998). *Journal of the American Veterinary Medical Association*. 218(4): 560-566: DOI: 10.2460/javma.2001.218.560.

5 **Gallatin L.L., Couëtil L.L. & Ash S.R. 2005.** Use of continuous-flow peritoneal dialysis for the treatment of acute renal failure in an adult horse. *Journal of the American Veterinary Medical Association*. 226(5): 756-759: DOI: 10.2460/javma.2005.226.756.

6 **Machado M., Wilson T.M., Sousa D.E.R., Gonçalves A.A.B., Martins C.S. & Castro M.B. 2020.** Uraemic Encephalopathy in a Persian Cat with Chronic Kidney Disease. *Journal of Comparative Pathology*. 180: 100-104: DOI: 10.1016/j.jcpa.2020.09.005.

7 **Mayhew I.G.J.M. & MacKay R.J. 2022.** Metabolic diseases. *Large Animal Neurology*. Hoboken: Wiley-Blackwell, pp.503-520.

8 **Mullen K.R. 2022.** Metabolic Disorders Associated with Renal Disease in Horses. *Veterinary Clinics of North America: Equine Practice*. 38(1): 109-122: DOI: 10.1016/j.cveq.2021.11.008.

9 **Mustonen A., Gonzalez O., Mendoza E., Kumar S. & Dick E.J. 2018.** Uremic encephalopathy in a rhesus macaque (*Macaca mulatta*): A case report and a brief review of the veterinary literature. *Journal of Medical Primatology.* 47(4): 278-282. DOI: 10.1111/jmp.12348.

10 **Olsen E. & van Galen G. 2022.** Chronic Renal Failure-Causes, Clinical Findings, Treatments and Prognosis. *Veterinary Clinics of North America: Equine Practice*. 38(1): 25-46. DOI: 10.1016/j.cveq.2021.11.003.

11 **Pilato F., Norata D., Rossi M.G., Di Lazzaro V. & Calandrelli R. 2025.** Consciousness disturbance in patients with chronic kidney disease: Rare but potentially treatable complication. Clinical and neuroradiological review. *Behavioural Brain Research*. 480: 115393. DOI: 10.1016/j.bbr.2024.115393.

12 **Radi Z.A., Thomsen B. V. & Summers B.A. 2005.** Renal (Uremic) Encephalopathy in a Goat. *Journal of Veterinary Medicine Series A*. 52(8): 397-400: DOI: 10.1111/j.1439-0442.2005.00752.x.

13 **Rosner M.H., Husain-Syed F., Reis T., Ronco C. & Vanholder R. 2022.** Uremic encephalopathy. *Kidney International*. 101(2): 227-241. DOI: 10.1016/j.kint.2021.09.025.

14 Ruwanpathirana P. & Chang T. 2024. Uraemic brainstem encephalopathy mimicking ocular myasthenia: a case report. *BMC Neurology*. 24(1): 121. DOI: 10.1186/s12883-024-03626-y.

15 Sakurai K., Ikenouchi H., Yamamoto N., Furuta K., Ogawa R. & Endo K. 2023. Uremic Encephalopathy Presenting with Unilateral Destructive Leukoencephalopathy Successfully Treated with Hemodialysis. *Intern Med*. 62: 1351-1353. DOI: 10.2169/internalmedicine.9494-22.

16 Schott H.C. 2007. Chronic Renal Failure in Horses. *Veterinary Clinics of North America: Equine Practice*. 23(3): 593-612. DOI: 10.1016/j.cveq.2007.10.002.

17 Sina F., Najafi D., Aziz-Ahari A., Shahraki E., Ahimahalle T.Z., Namjoo Z. & Hassanzadeh S. 2022. Uremic encephalopathy: A definite diagnosis by magnetic resonance imaging? *European Journal of Translational Myology*. 32(3): DOI: 10.4081/ejtm.2022.10613.

18 Xu L. & Zhang R. 2025. Nobiletin alleviates brain injury in uremic mice and inhibits indoxyl sulfate-induced neurotoxicity in HT22 cells through the phosphatidylinositol 3-kinase/protein kinase B signaling pathway. *Cytojournal*. 22(27): DOI: 10.25259/cytojournal_233_2024.