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Case Report

CASE REPORT OF A PSEUDORABIES (AUJESZKY'S DISEASE) IN A BITCH

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Summary: Bitch named Neca, mixed breed, spayed, 2,5 years old, was admitted to the Small animal clinic of Veterinary faculty of Ljubljana on 3rd January 2006 at 10.45 a.m. It had no chronic illnesses and was vaccinated against rabies. The day before the clinical signs appeared, it was apparently healthy. During the night it was kept in a garage. From the morning on it has been crying, curling up and foaming at the mouth. It was hospitalised. In spite of intensive care the bitch died 10 hours after admittance to the hospital. Directed questioning of the owners revealed the information that she was fed raw pork 7 days ago. Based on the history and clinical signs, suspected diagnosis of pseudorabies was established. Pathomorphological examination revealed lesions characteristic for pseudorabies, while etiological diagnosis was confirmed by bioassay with rabbit and virus isolation.

Key words: dog diseases; pseudorabies – etiology – diagnosis – pathology; herpesvirus 1, suid – isolation and purification; biological assay; dogs

Introduction

Pseudorabies (Aujeszky's disease, mad itch or infectious bulbar paralysis; AD) is by definition caused by DNA virus belonging to the alpha-herpesvirus family. Infection occurs in most countries of the world with the exception of Australia. Although many mammalian species are susceptible to infection with pseudorabies virus, it represents a predominant problem in pigs which are the main reservoir of the virus. In pigs, the infection is usually subclinical because they have become well adapted to the virus. The disease is usually spreading by commercial movement of infected pigs or contaminated pork products, showing that pseudorabies infection in dogs and cats usually occurs in areas where the disease is enzootic in pigs. Pets infect themselves by consuming contaminated raw pork meat, direct spread from dog to dog has not been described (1). Man and tailless apes seem to be resistant against infection (2).

After ingestion the virus enters nerve endings in the mucosa and spreads to the brain along nerve axons. Inflammation and functional abnormalities in brain cells result in neurological signs after an incubation period of around 3 to 6 days.

The infection of non-adapted species (like dogs are) results with death within hours after showing of the first symptoms (3).

Clinical case

The dog that has been examined at the Small animal clinic of the Veterinary faculty of Ljubljana was a 2,5 year old, mixed breed bitch, weighting 47,5 kg, vaccinated against rabies and spayed. The owners did not know what could have happened to the dog because it was kept in a garage during the night and that particular morning it could not get up, making strange sounds, the saliva was drooling down her mouth. Although the dog moved freely around the

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owner's estate, they thought it was very unlikely that it would eat something strange. Physical exam revealed dog's inability of standing upright. It was laying on it's side, the temperature was normal (38,0 °C), pulse was strong and regular (130 beats per minute), her CRT was less than 1 second and the color of the mouth mucosa was intensely red. Eyes were bright and the dog seemed to be alert at the time of the first examination. The skin around the neck was scratched and the bitch continued to scratch herself also during the physical exam. The abdomen was soft at palpation and there was no pain revealed.

CBC (complete blood count), biochemical profile, urinary profile and X-ray examination were performed. X-ray examination revealed enlarged urinary bladder and gastritis; thorax was without any pathological signs. Urinary profile was normal except high pH of 8 (normal < 7), slight proteinuria of 1,0 g/L and traces of urobilinogen (3,2 μmol/L). CBC showed $25{,}28\times10^9$ /L WBC (normal range 6,0-$18{,}0\times10^9$ /L), high HCT of 0,60 L/L (normal range 0,35-0,55 L/L), high HGB of 200 g/L (normal range 115-180 g/L). Neutrophilia of 91,1 % (normal range 60-80 %) and lymphopenia of 5,2 % (normal range 12,0-35,0 %) were found in differential. Biochemical parameters have shown hyperglicaemia of 14,8 mmol/L (normal range 3,9-5,0) and low potassium level of 2,95 mmol/L (normal range 3,6-5,0 mmol/L).

Initial therapy consisted of urinary catheter, i/v infusion with Ringer-lactate, antibiotics i/v (amoxycilin+ clavulonic acid in a dosage of 20 mg / kg BID) and i/m (gentamicinum in a dosage of 4 mg / kg SID). Within one hour the dog's body temperature started to rise from 38,0 C to 40,8 C and it was scratching itself intensively on the neck and face with it's hind legs. It became very restless and disorientated. It's ears became red and swollen and it swallowed with difficulty, so the sedation with midazolame i/v in a dosage 0,2 mg / kg was applied but without success. Body temperature continued to rise to 41,0 °C. The edema of the face and neck intensified and dexamethasone i/v in a dosage 1mg / kg was administered. Intensive cooling with ice cubs rectally and wet towels covering was performed. When dog's body temperature had raised to 41,4 °C carprofen 2 mg / kg i/v was added. Despite all the medicine that the dog had received the swelling of the head, neck and it's left eye were becoming life threatening. Its mental state was poor, it was not responding to anything, just tried to scratch vigorously. Ten hours after administering to the clinic, the dog died. Necropsy and viral tests were performed with suspicion of pseudorabies.

Results of necropsy and viral tests

Necropsy supported our suspicion of AD by finding diffuse non-suppurative meningoencephalitis and myelitis of the thoracal spinal cord.

The immunofluorescence test (IF), using specific conjugate against rabies virus was negative (data not shown) and therefore excluded rabies as differential diagnosis.

Experimental inoculation of rabbit with material from bitch brain and its death four days post inoculation confirmed the presence of AD-virus. Rabbit itching at the site of contagious material inoculation had been first noted on a second day after inoculation (a.i.). Third day a.i. traumatised skin of about 10 cm^2 was noticed. Excoriation of the skin at latero-ventral site of abdomen was well circumscribed and deep - it extended to subcutis. Apathy was noted on second day and nervous symptoms and restlessness were noted on a third day a.i. Rabbit have died on the 4th day a.i.

Isolation of the virus on porcine kidney cell line (PK-15) was successful. Neutralisation of the isolated virus JAN 05/06 with specific positive serum have proved the Aujeszky's disease virus.

Discussion

Leucocytosis with neutrophilia and lymphopenia were present in the bitch's blood. Since CBC (complete blood count) and biochemistry profile should not show specific changes (3) we presume that leucocytosis, neutrophilia and lymphopenia were not directly connected to the AD virus infection. These blood changes are usually present in acute bacterial infection or severe stress and due to the effects of steroids (6). In our case, they could be explained with severe stress, but also by several disease specific and non-specific pathological lesions found at necropsy like myocarditis, gastritis, enteritis and nephritis.

Higher hematocrite and haemoglobin revealed slight dehydration, which could be the consequence of inability to swallow and loss of liquid through saliva.

Hyperglicemia in our case could appeared due to the seizures and consecutive adrenaline (epinephrine) release, while low potassium levels seems most likely to be a consequence of respiratory alka-

losis (6), which could only be predicted since it was not proved by gas analysis. Urinary bladder has been probably enlarged due to inability to stand upright and urinate, but could be at least partly connected to proliferative and metaplastic lesions found in the bladder mucosa. Mild proteinuria was most probably a consequence of elevated body temperature (6) although multifocal granulomatous nephritis has been found at necropsy. Alkaline urine can support suspicion of respiratory alkalosis and also caused increased urinary excretion of urobilinogen. Swelling of the head and neck became life threatening when complicated with pulmonary oedema and congestion. Pulmonary oedema and congestion were already described complications in AD (5).

From the discussion with the owners it was found out that the bitch did get a raw pork head to eat about a week before the first symptoms appeared. The meat was purchased from the local butcher. The rest of the meat was cooked and used for human consumption. In Slovenia, the last case of AD in a dog was noted more than 20 years ago. Slovenia also tends to acquire AD-free status so thorough epizootic investigation was done about the source of the pork meat in our case. According to our data the pig was imported from Hungary that has already twice acquired status of AD-free country, but has lost it in 2002 (4). Our case shows the possibility of spreading of Aujeszky's disease among European Union members.

References

1. Vandevelde M. Pseudorabies. In: Green CE, eds. Infectious diseases of the dog and cat. Philadelphia: W.B. Saunders company, 1998: 126-8.

2. Kluge JP, Beran GW, Hill HT, et al. Pseudorabies (Aujeszky's disease). In: Leman AD et al., eds. Disease of swine. 7th ed. Ames: Iowa State University Press, 1992: 315-23.

3. Sellon RK. Canine viral diseases. In: Ettinger SJ, Feldman EC, eds. Textbook of veterinary internal medicine. 6th ed. St. Louis: Elsevier Saunders, 2005: 650-1.

4. Aujeszky's disease. Multiannual animal disease status (disease occurrence). OIE International. Paris: OIE International, 2006. http://www.oie.int/hs2/sit_mald_freq_pl.asp?c_cont=4&c_mald=21 (sept. 2006)

5. Greene CE. Infectious diseases of the dog and cat. 3rd ed. Philadelphia: W. B. Saunders company, 2006.

6. Bush BM. Interpretation of laboratory results for small animal clinicians. Oxford: Blackwell science, 1991.

PRIMER BOLEZNI AUJESZKEGA PRI PSICI

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Povzetek: Psica Neca, mešanka, sterilizirana, stara 2,5 leta je bila 3. januarja 2006 ob 10.45 uri pripeljana na Kliniko za kirurgijo in male živali Veterinarske fakultete v Ljubljani. Psica je bila prejšnji dan na videz še zdrava, čez noč zaprta v garaži, od zjutraj pa je jokala, ležala je zvita in iz gobca ji je tekla slina. Ni imela nobenih kroničnih bolezni, bila je tudi redno cepljena proti steklini. Po kliničnem pregledu je bila psica zaradi slabega stanja hospitalizirana. Kmalu so se pojavili še znaki intenzivnega praskanja. 10 ur po sprejemu na kliniko je psica kljub intenzivni terapiji poginila. Po ponovnem izpraševanju lastnikov smo izvedeli, da je psica pred 7 dnevi jedla surovo svinjsko meso. Na podlagi anamneze in poteka bolezni je bila postavljena domnevna diagnoza psevdorabiesa. Patomorfološka preiskava je potrdila spremembe, značilne za to bolezen, etiološka diagnoza pa je bila dokazana z biološkim poskusom na kuncu in z izolacijo virusa.

Ključne besede: psi, bolezni; pseudorabies – etiologija – diagnostika – patologija; herpesvirus 1, suid – izolacija in čiščenje; biološki poskus; psi