



Brief Notes on Air Embolism of the Brain in a Hospitalized Horse

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Abstract

An 8-year-old, 590 kg Belgian draught cross gelding underwent endoscopy and tendon sheath laceration repair under anesthesia. The horse spent several days in the hospital. The jugular catheter unintentionally disengaged from the extension set while the patient was recovering in the hospital after surgery. The horse started to circle to the left the following day and was bilaterally blind. Also seen were elevated cardiac troponin levels and a little tachycardia. Although the horse received supportive care, the neurologic symptoms remained, and humane death was chosen after three weeks of no improvement. The left occipital cortex's massive malacia and haemorrhage, along with generalized cerebral oedema, were the most notable findings on the gross postmortem examination. In the left and right occipital cortices, histopathology showed regionally significant grey and white matter necrosis, which is compatible with an infarctive aetiology. The postmortem examination results and the medical history are consistent with air emboli in the brain. The use of intravenous catheters in horses can result in this uncommon but dangerous problem.

Background: Continuous IV fluid administration, intermittent use during blood collection, and IV drug administration are all common uses for intravenous (IV) catheterization. Thrombophlebitis, exsanguination, catheter fragmentation, and emboli are risks related to IV catheter use in horses. The unusual, potentially fatal condition known as venous air embolism (VAE) in horses has also been observed in dogs, cats, cattle, and humans. Horses that have 3-6 VAE may have an extension set, injection cap, fluid line, or three-way stopcock that has been damaged or that has been

and the horse was determined to pose a serious danger of harm to both people and to itself. On Day 19, euthanasia with compassion was chosen.

Discussion / **H**

An unusual but possibly fatal side effect of using intravenous catheters in horses is venous air embolism. Agitation, colic, pruritus, muscle fasciculations, sweating, tachycardia, tachypnoea, pulmonary oedema, and behavioural and neurologic symptoms are some of the clinical indicators of venous air embolism. Antemortem diagnosis is not often easy. In addition to a history of a detached catheter and the necessary clinical indicators, evidence of a multifocal random infarctive process postmortem and histologically can help in the diagnosis of a venous air embolism. To prevent unintentional venous air embolism, intravenous catheter [6] integrity and their connections must be closely monitored. The entire brain was immersed in 10% neutral buffered formalin and fixed. Immersion fixative was also used on samples of common organ systems. Representative sections were routinely processed into haematoxylin and eosin-stained slides after being routinely clipped. The left and right occipital cortices of the brain's histopathology revealed multifocal and regionally large, discretely marginated areas of cortical loss. The changes ranged from full thickness cortical grey matter necrosis and isolated foci of white matter necrosis to oedema with necrosis and loss of outer cortical lamina. Lytic necrosis with parenchymal loss and Gitter cell infiltration made up the malacia foci. Sections of the heart's interventricular septum, left and right ventricular free walls, and sarcoplasm revealed low levels of lymphocyte and plasma cell infiltration as well as uncommon myocyte necrosis. History further supported VAE, and postmortem examination and histopathologic results were compatible with embolism resulting in multifocal brain infarction. In other tissues, there were no notable discoveries.

Discussion

The jugular catheter of this horse was accidentally disengaged, allowing significant air to be aspirated into the bloodstream. As a result, multiple air emboli travelled through the right heart, pulmonary circulation, left heart, and peripheral circulation before inflicting severe ischaemic injury on the brain. Disconnection of a catheter or fluid line is the most frequent reason for air emboli in horses. 8 The negative pressure in the vein aspirates air into the bloodstream when a jugular catheter or catheter line gets exposed to the atmosphere. 10 VAE has also been documented in one horse after cystoscopy with CO₂ oedema my (result from S j bubable in the)Tj-0.018 Tw09 -1.2 D6ropy w bloodstream tgt blkh the right tsah of the hearr orlodgre in t5e hownh