



## 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 accidentally disconnected from the catheter.

**Keywords:** IV fluid; Brain; Hospitalized Horse; Air

### Introduction

An 8-year-old Belgian draught cross gelding underwent normal anaesthesia for tenoscopic examination, lavage, and closure of a linear incision involving the digital flexor tendon sheath that was about 2-3 cm long and located on the plantar aspect of the right hind pastern. Before surgery and throughout hospitalisation, a 14G extended-use 5.25-inch over-the-needle catheter (Milacath International, Florence, KY, USA) was inserted into the left jugular vein to help provide broad-spectrum antibiotics and non-steroidal anti-inflammatory medicines. Four days following the procedure, the catheter unintentionally detached from the extension set. The catheter was unplugged for no more than an hour. The horse was observed to roll once and to be heavily perspiring. The catheter was reconnected to the extension set after it was discovered that air could not be [1-5] sucked from it. Vital indicators were within typical bounds. The horse was hyperesthetic the following day and exhibited a left head tilt, a positive pupillary light reaction, and a bilaterally negative menacing response. It was head pressing in the stall on occasion and constantly rotating to the left. It displayed diffuse muscle fasciculations and was grade 3 out of 5 ataxic in all limbs. Bilateral ear twitching consistent with partial seizure activity was one of the other findings. Forebrain was the area of injury's neurological localization (predominantly left-sided). It has been determined that ataxia may be a contemporaneous brainstem or spinal cord component. Alternately, ataxia might have been mistaken for fear of moving brought on by sudden blindness. Additional findings included sinus tachycardia (60 bpm), elevated cardiac troponin I (0.28 ng/ml, reference range: 0.00-0.06), hyperglycemia (142 mg/dl, reference range: 80-120), mildly elevated creatine kinase (460 U/L, reference range: 50-400), and aspartate aminotransferase activity (377 U/L, reference range: 100-360). The rest of the chemical panel, venous lactate, and complete blood count were all within normal ranges. The horse was first given water-soluble natural vitamin E (10 IU/kg, Kentucky Performance Products, Versailles, KY, USA) orally (PO) every 24 hours to reduce inflammation, flunixin meglumine (1.1 mg/kg, Merck & Co., Kenilworth, NJ, USA) IV slowly every 12

hours to reduce inflammation, and mannitol (0.5 g/kg, ICU Medical, San Clemente, CA, USA) IV slowly every 6 hours for four doses to reduce it. It also received minocycline (4 mg/kg, Aurobindo Pharma, Hyderabad, India) PO every 12 hours primarily to prevent infection of the surgical wound and also because minocycline is able to penetrate the cerebrospinal fluid in horses and may be helpful for traumatic brain injury. Gabapentin (10 mg/kg, ScieGen Pharmaceuticals, Hauppauge, NY, USA) PO every 12 hours for pain and anticonvulsant properties. The IV catheter was withdrawn after the fourth mannitol dose was given, and flunixin meglumine paste (1.1 mg/kg) was then given PO every 12 to 24 hours. After 8 days, gabapentin was tapered off and ceased. For the rest of the hospital stay, the other meds (minocycline, vitamin E, and flunixin meglumine) were continued. The tachycardia went away after two days. After the third dose of mannitol was administered, the head pressure and tilt became less noticeable. When handled, the horse remained wary and hyper-reactive to any movement. Due to the patient's possible hemi-neglect condition from the left cerebrocortical injury, it was unable to approach the patient from the right side. The patient was able to navigate on Day 15 after the problem, which may have indicated a partial recovery of vision. On Day 18 after the problem, the patient went outside, but as soon as it started cantering in circles to the left without regard for anything or anyone around it, it became agitated and uncontrollable. Hyperesthesia, a lack of threat, persistent compulsive leftward circling, and unpredictable behaviour persisted,

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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.

### Learning Points/Take-Home Messages

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 fixing 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 CO<sub>2</sub> insufflation and in one horse as a side effect of CO<sub>2</sub> insufflation during arthroscopy. In these cases, the entry of atmospheric gas into the venous vasculature was probably facilitated by the injection of pressurised gas into small, enclosed bodily cavities. An open jugular catheter may aspirate a substantial amount of air. Aspiration of air into the catheter in one incidence of VAE in a horse was severe enough to be audible to the unaided ear. 1 A 14-gauge catheter with a 5 cmH<sub>2</sub>O pressure gradient was shown to be capable of aspirating 100 ml of air per second in a different paper. According to reports, 0.5-0.75 and 7.5-15.0 ml of air per kg of bodyweight are the deadly levels for rabbits and dogs, respectively. 10 Although it is unknown how much air was inhaled into the horse's catheter, clinical signs have

been recorded to appear at volumes more than 0.25 ml of air per kg of bodyweight, which translates to a minimum [7-10] estimated volume of about 150 ml for this horse. 14 A different study from 1934 found that giving horses a 500 cc dose of air quickly may kill them. Acute pulmonary thrombosis and oedema may result from gas bubbles in the venous bloodstream that block the right side of the heart or lodge in the pulmonary circulation. As shown in this instance, bubbles can harm other tissues by ischemic injury when they enter the bloodstream. 8 The velocity and total volume of air delivered into the bloodstream, as well as the final distribution and size of the emboli, all have an impact on the clinical outcome.

### Conflict of Interest

There are currently no conflicts of interest, according to the authors.

### Author Contributions

The idea for the case report was developed by Paula A. Schaffer, Rachel Hritz analysed the case information and wrote the first draught, and Paula A. Schaffer, Kathleen R. Mullen, Gary Mason, and others offered feedback and proofreading.

### Ethics Statement

The authors of the article have made significant contributions to the conception and design, acquisition, analysis, and interpretation of the data. They have also written the article or have critically revised it for important intellectual content, and have given their final approval to the version that will be published. The only sources used for this investigation were the patient's medical file and postmortem materials. There was no request for ethics committee permission.

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