Lower Airway Disease in the Athletic Horse

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KEYWORDS

• EIPH • IAD • Equine asthma • Respiratory • Exercise • Performance

KEY POINTS

- Exercise-induced pulmonary hemorrhage (EIPH) and inflammatory airway disease (IAD) are the two most important lower airway diseases of the athletic horse.
- EIPH may be considered, at least at the onset, as a problem of physiology rather than a disease, and IAD is a disease primarily of domestication.
- Both EIPH and IAD are widespread among the athletic horse population and account for an impressive number of horses that fail to perform to their potential.
- Because of the high demands for oxygen in the athletic horse, even minor insults to the oxygen-carrying capacity of the body can affect performance, so it is of critical importance to keep the lungs as healthy as possible.

EXERCISE-INDUCED PULMONARY HEMORRHAGE

Exercise-induced pulmonary hemorrhage (EIPH), or bleeding that comes from the horse's lungs during exercise, is certainly the most dramatic of the lower airway diseases that affect equine athletes and the one that causes most consternation in the lay population. It is commonly held by both veterinarians and the lay population that EIPH is a major cause of wastage for equine athletes, especially in the racing industry, and that, when severe, it can result in sudden death. Despite this accepted knowledge, in reality, the connection between performance and EIPH is still poorly understood and sudden death due to EIPH occurs in less than 0.029% of the racing population. There is, indeed, debate as to whether EIPH is even a disease or whether it is an inevitable outcome of the remarkable physiology that allows the horse to be an elite athlete.

Epidemiology: Who Gets It and What Are the Risk Factors?

Bleeding Childers, the grand progenitor of Eclipse, who is ubiquitous in the pedigree of almost all modern racehorses, was eponymously named because he bled so often.

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Although EIPH is well-recognized in the equine racing population, with the vast majority of racing Thoroughbreds and Standardbreds affected,[1] it has become increasingly clear that this is a disease that any horse that performs strenuous exercise at speed has. Populations of horses that perform bursts of work at speed are those that have most recently been identified as experiencing EIPH, including barrel horses whose runs generally last less than 20 seconds[2] and polo ponies that run for short bursts of speed during a 7-minute chukka.[3] Reported prevalences depend on the method used for diagnosis. For instance, if tracheobronchoscopy is used within 2 hours of racing, up to 75% of racehorses are found to be bleeders,[4] whereas with examination of cytology from bronchoalveolar lavage (BAL) to detect evidence of recent and more chronic hemorrhage from the lungs, the number approaches 100%.[5] One population of highly competitive horses that does not regularly experience EIPH, however, is the endurance horse even at high levels of competition.[6] The putative reasons for this will be more fully explored in the later discussion of pathogenesis.

The biggest risk factor for a horse developing EIPH is going at high speed or undergoing very intense exercise. This is most easily appreciated in Thoroughbred racehorses, where it has been found that more horses have evidence of pulmonary hemorrhage after racing than they do after breezing.[7] Horses jumping at speed, such as steeplechasers, are more likely to develop EIPH than horses that race on the flat.[8] Although older horses are commonly thought to be more susceptible to EIPH, it is years spent racing rather than age itself that is associated with EIPH. Other risk factors that have been identified include exercise when temperatures are less than 68F and wearing bar shoes.[9]

Pathogenesis: Why Do Horses Bleed from the Lungs?

The prevailing theory for the cause of EIPH posits that stress failure of pulmonary capillaries occurs because of very high transmural pressures or the pressure that develops across the wall of the pulmonary capillary. It is easiest to think of this as a push and a pull—the push comes from a very high pulmonary artery pressure at intense exercise, and the pull comes from a negative alveolar pressure at the same time as the horse breathes in. This, in turn, is mandated in order to accommodate the horse's phenomenal exercise capacity; in order to supply the amount of oxygen that the horse needs to perform at its extraordinary levels of $V_{O_{2max}}$, the heart rate must be high and pulmonary capillary pressures skyrocket from approximately 25 mm Hg to up to 90 mm Hg because high left ventricular filling pressures are necessary to maintain cardiac output in these conditions. Simultaneously, in order to accommodate the need for increased ventilation that is coupled to the horse's stride, the respiratory system is forced to exert tremendous negative pressures in the pleural space—up to -60 cm H_2O or approximately 45 mm Hg. These pressures summate to more than 120 mm Hg transmural pressure, making it easy to understand why pulmonary capillaries then rupture.[10] For perspective, in human athletes, 20 to 25 mm Hg threshold for pulmonary capillary pressures is associated with interstitial lung edema and altered ventilation/perfusion relationships, and maximum pulmonary arterial pressures (PAPs) of 40 to 50 mm Hg, which elite human athletes can achieve at maximal exercise, are considered to correspond to the extreme of tolerable right ventricular afterload.[11] These numbers seem positively puny with respect to the exercise physiology of the horse.

Anything, therefore, that either increases pulmonary artery pressures or decreases (creates a larger negative pressure) alveolar pressures will potentiate EIPH in the equine athlete. The most logical comorbidities that would contribute, therefore, would be dynamic upper airway obstructions, such as laryngeal hemiplegia or dorsal