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ANHIDROSIS

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SUMMARY

Anhidrosis with emphasis on etiopathogenesis is reviewed in this paper. Anhidrosis, a non-sweating syndrome in horses, first reported in 1925, was mainly observed in temperate horses introduced into the hot humid environment. This phenomenon is believed to be a compensatory mechanism for profuse sweating. Anhidrosis can cause reduced performance, elevated respiratory rate, discomfort, varying degree of anorexia and alopecia. Death may ensue as a result of a heat stroke. Feeding, hypothyroidism, hereditary factors, exhaustion of adrenaline, electrolyte imbalance and atrophy of sweat gland may contribute to anhidrosis. The condition can easily be diagnosed from clinical signs. There is no specific treatment, but transferring the horse to a cooler climate or stabling under air-conditioning, in conjunction with vitamin E or ethylenediamine dihydrioidide, may improve the condition.

Keywords: Anhidrosis, etiopathogenesis, clinical signs, diagnosis, treatment

INTRODUCTION

Anhidrosis is a non-sweating syndrome commonly referred to as dry coat. The condition now widely recognised, was first recorded by Wright and Till in 1925 and McGregor in 1935 (Evans *et al.*, 1957b). Although mainly reported in Thoroughbreds and Thoroughbred crosses (Currie and Seager, 1976; Evans *et al.*, 1957a), it can affect horses of all breeds, both local and imported (Warner and Mayhew, 1982). Equine anhidrosis commonly occurs in the hot, humid environment irrespective of the region of the world. The condition has been reported in India, Tonga, Sri Lanka, The Philippines, West Indies, Indonesia (Evans *et al.*, 1957a), the United States of America (Marsh, 1961)), Australia, New Zealand, Panama, South America and Puerto Rico (Correa and Calderin, 1966), Trinidad (Gilyard, 1944), and Hong Kong (Jenkinson *et al.*, 1985). In Malaysia, equine anhidrosis was first reported in 1935 (Evans *et al.*, 1957a).

The inability of newly introduced temperate horses to acclimatise to the hot, humid environment usually causes the horse to sweat profusely under the new environment. Gradually, they show decreased sweating and subsequently non-sweating. This was observed by Evans (1965), who reported that Thoroughbreds shipped from Britain to Malaya showed profuse sweating when the horses passed the Red Sea in summer. Although not common, this condition has also been reported in non-tropical countries such the West Indies, Australia and the United States of America (Peter *et al.*, 1981) where the climate is dry, hot and humid.

Anhidrosis has also been recorded in horses bred locally in tropical countries from imported mare or stallion (Evans, 1965).

Profuse sweating or hiperhidrosis in horses is believed to be a compensatory mechanism similar to that in humans introduced into a hot and humid environment (Warner and Mayhew, 1982). These horses gradually lose the ability to sweat. The incidence of complete anhidrosis is low; however, if partial anhidrosis or hypoanhidrosis were considered together, the rate of incidence of anhidrosis is as high as 20%. (Peter *et al.*, 1981).

ETIOPATHOGENESIS

The cause of anhidrosis is still not well understood. Thus the role of electrolytes, hormones, sweat glands, environment, stress and other related factors in sweating need to be clearly defined. The single most important factor in anhidrosis, however, is management of horses in the hot and humid environment. There are, however, other suggested etiologies to the failure to sweat, among which are stress under a high humid environment, high concentrate feeding, hypothyroidism, hereditary traits (Currie and Seager, 1976), corn or concentrate feeding without grass (Marsh, 1961), high strung horses, high protein diet (Evans *et al.*, 1957a), adrenaline exhaustion or failed production, reduced skin blood flow, damaged sweat glands, sweat glands not responsive to adrenaline, heat exhaustion, sweat retention syndrome (Beadle *et al.*, 1982), hypochloraemia, hyperkeratinisation of sweat ducts and depletion of water and electrolytes (Correa and Calderin, 1966). This seems to suggest that the underlying etiology of equine anhidrosis is related to adrenaline production, sweat gland function and electrolyte balance.

Adrenaline

Sweat glands of horses are of the apocrine type and sweating in horses is dependent on the level of adrenaline in blood. Low plasma adrenaline concentration causing sweating failure may be due to the stores being completely exhausted from high adrenaline utilisation as a result of sudden profuse sweating. This is commonly seen in and associated with either horses worked in the hot and humid environment or heat or work stress initiating high levels of adrenaline production. The plasma adrenaline concentration may also be low due to thyroid insufficiency as in hypothyroidism. Low circulating adrenaline results in restriction of blood supply to the skin, negatively influencing the function of the sweat glands. In horses, blood adrenaline concentration was found to increase during exercise (Evans *et al.*, 1957b; Evans, 1966). However, there is evidence that resting adrenaline concentrations are higher in horses in the tropical environment (Warner, 1982). This suggests that the sweat gland receptors in anhidrotic horses may have accommodated the higher level of adrenaline without causing sweating. Even with exercise, the increased adrenaline stimulation could not induce sweating.

Sweat gland

The sweat gland may fail to respond or be resistant to the normal level of adrenaline in circulation resulting in failure to sweat. The sweat glands may also become damaged or atrophied and eventually exhausted from prolonged stimulation. These phenomena may arise from the initial profuse sweating causing sweat glands to be overworked as the horses are first introduced into the hot and humid environment. The damaged sweat gland can be demonstrated by the hyperkeratinisation of the sweat duct. In anhidrotic horses, approximately 10% of the sweat glands are blocked by keratinous plugs, believed to be a consequence of failure of the sweat glands to function (Evans *et al.*, 1957a). This amount of blocked sweat glands is actually not significant in the etiopathogenesis of anhidrosis. However, structural evidence seems to suggest that there is also sweat gland atrophy in anhidrosis (Jenkinson *et al.*, 1989). Currently, it is not clear whether the ductal occlusion which is induced by excessive epithelial hydration is the primary or secondary factor to the loss of secretory cell activity. Although 60 to 90 % of secretory cells at all levels of the fundus of sweat glands of anhidrotic horses were flattened (Jenkinson *et al.*, 1985), the same number of flattened secretory cells were also observed in 70% of normal sweating horses in the hot humid environment. This seems to suggest that the presence of a higher percentage of flattened secretory cells is in fact a result of stress imposed on the sweat glands from excessive sweating.

Electrolyte balance

Electrolyte is important in thermoregulation and maintenance of numerous biological functions of the body. The electrolyte functions, among others, in nerve conduction, muscle contraction, maintenance of blood flow, water and plasma volume, cell metabolism, acid-base balance, cardiovascular function, blood clotting, and growth.

During exercise, various complex feedback mechanisms are involved in the regulation of electrolytes. This mechanism may be influenced by intake and absorption, excretion, distribution of electrolytes to the body systems and hormones. The equine sweat is hypertonic relative to plasma. The normal horse during excercise is able to produce large quantities of sweat. It has been shown that horses can lose as much as 37 litres of water through sweat compared to 2.8 litres through urination, when ridden for a distance of 80 kilometers. In horses, after 15 minutes of work, at a sweat rate of 27 ml/m^2/min, the sweat may contain 13.6 mmol/L Na^+, 28.1 mmol/L K^+, 149.5 mmol/L Cl^- and 0.32 g/L protein (McCutcheon *et al.*, 1995). In prolonged exercise, however, the electrolyte and protein composition of sweat may not change but the plasma electrolyte concentration may be decreased due to loss of hypertonic sweat (McConaghy *et al.*, 1995). Loss of sweat causes loss of Na^+, K^+, Cl^-, protein and water resulting in reduction in their body contents. The depletion of water, Na^+, Cl^-, during excessive sweating may initiate various feedback mechanisms to conserve the water, Na^+ and Cl^- through non-sweating. This phenomenon is exhibited in anhidrosis.

CLINICAL SIGNS

Anhidrosis is best recognised by the inability of the horse to sweat whether at rest under the hot and humid environment or when heavily worked. Semi-anhidrosis horses may show some localised sweating mainly at the shoulder, brisket and under the saddle. In these horses, the nature of sweat is thin and watery compared to the watery and lathery sweat in normal horses. Horses introduced into the hot and humid environment may first show abnormally high respiratory rate or blowing with exaggerated dilatation of the nostril and laboured breathing. The condition may be preceded or occur together with profuse sweating at rest. The hyperpnoea and dyspnoea may also be accompanied by an increase in body temperature, pulse and respiration or panting. The horse may also show signs of discomfort, irritability, lost of appetite and refusal to drink (Snow and Vogel, 1987). Not all horses introduced into the hot and humid environment with the above signs become anhidrotic. Anhidrosis in these horses can only be confirmed when these signs are accompanied by reduced sweating or complete non-sweating.

Anhidrotic horses may show signs of reduced performance that may vary from slight to severe. The affected horse becomes easily exhausted and fatigued during and after work. Anhidrosis does not cause motility at rest, however, if overworked, the horse may be somnolent, collapse, comatose and die of heat stroke due to circulatory failure. In general, anhidrotic horses usually have poor to fair body condition, difficulty in gaining weight with possibility of losing weight, dull and dry hair coat, alopecia especially at the face and shoulder with continuous lost of hair. The skin may also be dry and coarse with brittle hair. Sweat analysis would show low concentrations of NaCl. The sweat ducts may be blocked. Histologically, there may be evidence of increased thyroid activity. At post-mortem, signs of dehydration may be observed. Post-mortem findings include pulmonary emphysema, enlarged heart and lungs, rib impression on the lung surface, hyperaemia, venous congestion and congested right auricle and ventricle.

DIAGNOSIS

Equine anhidrosis is easily diagnosed from the history of reduced performance and inability to sweat. Clinical examination usually reveals varying degree of non-infectious, non-pruritic alopecia at the face and shoulder. The alopecia is usually oily. The anhidrotic horse has a high respiratory rate at rest. When worked, the horse may exhibit non-sweating ranging from completely dry to wetness at the shoulder, brisket and seat of the saddle only. The sweat character is usually thin, watery and non-lathery compared to the wet lathery sweat of normal horses. The overall condition of anhidrotic horses is usually not satisfactory. It is difficult to diagnose anhidrosis from the laboratory examination of blood parameters. However, it has been shown that the NaCl in sweat of anhidrotic horses is lower than in normal horses (Correa and Calderin, 1966). Examination at post-mortem may not provide a diagnosis of anhidrosis since the anhidrotic horses may show the same changes as horses which die from heat exhaustion or heat stroke not caused by anhidrosis.

TREATMENT AND CONTROL

The treatment of equine anhidrosis and its response to treatment can not be accurately assessed due to lack of information on its etiology and pathogenesis. However, several reports have shown varying degree of response with the use of vitamin E at 2000-3000 i.u./day (Peter *et al.*, 1981; Warner, 1982). Good response to vitamin E treatment has been observed when at the same time the horses were transferred to cooler climatic areas or air-conditioned stables. Transferring to a cooler climate or air-conditioned stable alone, however, did not seem to alleviate signs of anhidrosis in the majority of cases. Thus, at this juncture, it is not clear whether treatment with vitamin E alone will provide a positive response or not in anhidrotic horses. Other treatments that have been used in conjunction with air-conditioned stabling or change to cooler environment with varying degree of success were ethylenediamine dihydroiodide at 3 gram per day, oral electrolyte (Marsh, 1961) injection of thyroid extract at 50gm for 20 days (Correa and Calderin, 1966.) and intravenous NaCl at 1 to 2 litres per day for 10 to 15 days. Treatments that showed limited success included increasing alfalfa and reducing concentrate in feed (Peter, 1981), increasing salt intake, hypertonic drinking water, and sea water bath for 10 minutes.

Heat stress, exhaustion and heat stroke can be reduced in anhidrotic horses by ensuring work is carried out during cool days only and monitoring, temperature, pulse and respiration, water intake and signs of stress.

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