Into Thin Air

Bear Creek High School

Period 2

ABSTRACT

Alaska's Mt. Denali can reach up to 17,660 feet in altitude (Lundmark, 2003). At this elevation, Mark McKinley found himself struggling to breath and constantly short of breath (Lundmark, 2003). With his health rapidly deteriorating, his mountain climbing team noticed that he suffered from extreme fatigue (he was falling behind) and he soon fell unconscious (Lundmark, 2003). At the hospital, a diagnosis team diagnosed him as having suffered high altitude pulmonary edema (Lundmark, 2003). His lungs have overworked themselves in the thin atmosphere of Mt. Denali and tore their tissue (Chen, 2013). From the resultant tear came fluids that seeped into the alveoli sacs of his lungs and reduced their oxygen capacity (Wedro, 2014). Oxygen is needed for aerobic cellular respiration, during which a glucose monomer is broken into two pyruvic acids, which then become two molecules of acetyl CoA, which then enters the Kreb Cycle to be further oxidized (Campbell et al., 2014). The electrons and hydrogens (carried by NAD and FAPH2) enter the electron transport chain, which uses a hydrogen gradient and chemiosmosis to produce ATP from ADP molecules and phosphate groups - but this can only happen in the presence of oxygen (Campbell et al., 2014). The presence of ATP molecules is needed for muscle contractions because after the myosin head makes contact with the actin filament, they can only release their bonds if ATP binds to them (Campbell et al., 2014). Mark is suffering from high altitude pulmonary edema because his exposure to the low concentration of air in the atmosphere had caused his lungs to breathe faster, tear themselves, and fill his alveoli sacs with fluids that further reduced his breathing capacity (Chen, 2013). Mark is not suffering from an asthma attack

because while he does have difficulty breathing and while he is coughing, exposure to high altitudes does not cause asthma attacks (Lundmark, 2003).

Keywords: High altitude pulmonary edema, asthma, low oxygen concentration, lung tear, alveoli sacs

Introduction

Mark McKinley is suffering from high altitude pulmonary edema (Lundmark, 2003). Mark had difficulty breathing when he ascended the mountain, more so than his companions (Lundmark, 2003). The fact that he was short of breath even when he rested indicates a problem with his respiratory system (Campbell et al., 2014). More specifically, high altitude pulmonary edema, like all pulmonary edemas, is an ailment that results with fluid in the lungs reducing oxygen capacity (Wedro, 2014). Unlike all pulmonary edemas, however, high altitude pulmonary edema is noncardiogenic, which means that it is not caused by a dysfunctional heart (Wedro, 2014). Rather, it is caused by torn lung tissue (Chen, 2013). High in the mountains, a lower concentration of air supply hinders easy breathing, so Mark's lungs had overworked themselves to supply oxygen to his muscles and as a result, tore their own tissue (Lundmark, 2003; Dugdale, 2012). Torn lung tissue releases fluid (Dugdale, 2012). The fluid eventually seeps into the alveoli and reduces normal oxygen flow through the lungs, which severely worsens the already-scarce oxygen supply that the lungs give to the body (Wedro, 2014). Without oxygen, muscle contraction slows and fatigue sets in, which explains why Mark began to fall behind the pace his other mountaineers (Lundmark, 2003). Also affected are the concentrations of waste in his blood - in particular carbon dioxide and lactic acid from fermentation (Lundmark, 2003). Without efficient means of diffusion in the lungs, high levels of carbon dioxide remained in Mark's blood (Shier, Butler, & Lewis, 2003). Without sufficient oxygen reserves, cells must rely on lactic acid fermentation to obtain ATP (Campbell et al., 2014).

Mark did not suffer from an asthma attack (Lundmark, 2003). Asthma attacks occur when airways become constricted due to swelling of muscle lining such airways, as well as the simultaneous secretion of mucus along such airways (Gibbons, 2014). Although difficulty breathing and perhaps even unconsciousness are common symptoms of both pulmonary edema and asthma attacks, high altitudes trigger only pulmonary edema (Wedro, 2014). Common triggers of asthma attacks, or exacerbations, include pollution, allergens, medicine, sulfites, and respiratory viral infections - none of which were known in Mark's case (Gibbons, 2014; Lundmark, 2003). Moreover, asthma attacks disrupt respiration abruptly and suddenly, rather than gradually, as is the case with pulmonary edema (Gibbons, 2014; Lundmark, 2003). Mark progressively lost his ability to breathe even after acclimation, but had he suffered from asthma, he would have lost the ability instantaneously (Gibbons, 2014; Lundmark, 2003).

Claim

All of Mark's symptoms point to high altitude pulmonary edema, which is the tearing of lung tissue and the seepage of fluids into the alveoli (Lundmark, 2003). Whether he was moving or resting, Mark had a had time catching his breath (Lundmark, 2003). This reflects the influence of fluids in the lungs because as alveoli volume is occupied, it is even more difficult for oxygen that is already scarce to diffuse into the blood steam (Chen, 2013). For the same reason, both of Mark's arterial oxygen and carbon dioxide pressure fell below average; they measured to 52 and 30 Torr, respectively, when the normal pressures are 80-100 and 35-45 Torr, respectively

(Lundmark, 2003). With fluids in the lungs, gases cannot diffuse efficiently from the pulmonary capillaries to the inhaled oxygen in the alveoli, so unoxygenated blood in the artery remains unoxygenated and carbon dioxide remains low (Wedro, 2014). Mark was also coughing because his nervous system had detected the unnatural seepage of fluid in the thoracic cavity (Shier et al., 2003). At 7.23, Mark's arterial pH is lower than the normal value, which has the range 7.38 to 7.44 (Lundmark, 2003). This is because in the absence of sufficient oxygen, Mark's cells must rely on lactic acid fermentation to supply enough ATP for muscle contraction, and the build up of that acid in the bloodstream results in a lower pH (Campbell et al., 2014; Wedro, 2014; Mayo Clinic, 2013). Mark has a higher hematocrit percentage at 58%, when the normal percentage is between 42% and 52% (Lundmark, 2003). This is because when the body notices a deficiency in its oxygen supply, it triggers its bone marrow to produce more red blood cells to compensate, which constitutes the hematocrit percentage (Shier et al., 2003; Wedro, 2014). Finally, Mark's bicarbonate concentrations are lower than normal because it stands at 18 meq/L whereas normal concentrations are between 20 and 24 meq/L (Lundmark, 2003). This is because bicarbonate serves as a buffer (that is, it regulates pH in the blood) and since there are so many lactic acids floating around in the blood, it has exhausted a great portion of its supply attempting to restore the proper blood pH (Shier et al., 2003).

Counterclaim

Although the symptoms may appear similar to those of an asthma exacerbation, Mark did not suffer from an asthma attack because there were no probable allergic triggers in the high altitude environment of Mt. Denali, nor do his internal homeostatic values quite match up with those of asthma (Lundmark, 2003; Mayo Clinic, 2013). Mark had struggled to catch his breath, which matches the description of pulmonary edema more than an asthma attack (Gibbons, 2014; Lundmark, 2003). Mark was taking deep breaths without feeling well-respirated, as is common with pulmonary edema, but if he had asthma, he would not have been able to take deep breaths at all because the smooth muscle along his respiratory tract would have swollen up (Gibbons, 2014; Mayo Clinic, 2013). Moreover, asthma attacks tend to occur instantaneously, constricting the breathing of the individual instantly (Gibbons, 2014). Mark gradually lost his proper breathing over the course of hours due to the gradual seepage of fluids into his alveoli sacs (Lundmark, 2003; Wedro, 2014). Moreover, allergens and pollution trigger asthma attacks, but none of these were present in the high altitudes of Mount Denali (Lundmark, 2003). If Mark had suffered an asthma attack from any trigger, it would have been strenuous exercise, but had he known that strenuous exercise causes him to suffer asthma attacks, he would have brought along an inhaler (Lundmark, 2003). His internal homeostatic measurements reveal inefficient gas diffusion rather than the insufficient inflow of oxygen. If Mark had not been able to breathe at all, as would be true in an asthma attack, there is no reason why mark's blood carbon dioxide would be lower than normal - at 30 Torr (Lundmark, 2003). Because he had pulmonary edema, carbon dioxide

could diffuse outward into the fluid, but oxygen could not diffuse inward across the fluid into the capillaries in the alveoli (Lundmark, 2003; Wedro, 2014).

Conclusion

Mark suffered from high altitude pulmonary edema, which is the disruption of the respiratory system caused by the flooding of the alveoli sacs by any fluid, because he had been exposed to high altitudes, because he had exerted great muscular effort at that altitude, and because all of his homeostatic values - blood oxygen concentration, blood carbon dioxide concentration, blood pH, and blood buffer concentration - all reflect inefficient gas exchange in the lungs rather than a cut-off in the airways (Lundmark, 2003). Mark's concerned team rushed him down to base camp, where he fell unconscious, and presumably summoned a helicopter to deliver him to a hospital (Lundmark, 2003). While at the camp, they gave him a Gammow bag, which contained pressurized oxygen that introduced more oxygen supply to his damaged lungs (Lundmark, 2003). At that hospital, Mark was treated with more oxygen and presumably pharmaceuticals to reduce the inflammation of his lung tissue (Lundmark, 2003). Unfortunately, being a victim of high altitude pulmonary edema, Mark is susceptible to recurrence, so now he cannot climb mountains without special precautions (Lundmark, 2003). From this case study, one can reflect that dysfunctionality in the respiratory system can also cause dysfunctionality in adjacent systems. Without a functioning respiratory system, oxygen is insufficient, so aerobic cellular respiration cannot transpire, which means there is less ATP for muscles to contract, this disrupting the muscular system. Moreover, the formation of lactic acid from provisionary fermentation also lowers the pH of the blood. A more acidic blood can can cause problems with the circulatory system, if insufficient oxygen flow to the heart already hasn't.

References

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