Causes of Edema That Result From an Increased Capillary Pressure

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Edema is a medical condition that occurs as a result of the excessive accumulation of body fluid in the body tissues (Wassermann, 2008). It can occur in any part of the body including the limb, skin, or lungs (Wassermann, 2008). There are four factors that precipitate Edema: increased capillary permeability, decreased plasma oncotic pressure, lymphatic obstruction, and increased capillary permeability (Wassermann, 2008). Increased capillary hydrostatic pressure may caused by an increase in venous pressure (Wassermann, 2008). Proinflammatory mediators such as bradykinin, and histamine can also cause capillary permeability and lead to edema (Wassermann, 2008). This paper differentiates between three causes of edema that result from an increased capillary pressure. It discusses the pathophysiologic changes that occur that results in edema.

The three causes of edema that result from an increased capillary pressure are increased vascular volume, venous obstruction, and decreased colloidal Osmotic pressure (Fishman, 2008).

Increased Vascular Volume

The increase in capillary pressure occurs following an increase in the hydrostatic pressure. Increase in the hydrostatic pressure can either retard the movement of the fluid into the capillary or favor its movement out of the capillary (Wassermann, 2008). This promotes the formation of edema.

Normally, the precapillary arteriole resistance is regulated to ensure alterations in the systemic blood pressure do not lead to marked changes in capillary hydrostatic pressure. However, in cases where the regulation of postcapillary resistance does not occur, the pressure in the venous part of the circulation significantly affects the capillary hydrostatic pressure (AnatLoewenstein, & Coscas, 2010). Increase venous pressure alters capillary hydrostatic pressure, which in turn increases the volume of fluid into the interstitial spaces (Wassermann, 2008). Excessive retention of water and sodium can also cause intravascular volume to expand. This can increase the hydrostatic pressure resulting in increased plasma osmotic pressure. Excess water and salt retention are often seen in a variety of diseases including *acute renal failure*, and *poststeptococcal glomerulonephritis*. Common causes of increased vascular volume include heart failure, Kidney disease, venous thrombosis, pregnancy and premenstrual sodium retention (Wassermann, 2008).

Venous obstruction

The movement of fluid in and out of the vascular compartment is controlled by starling forces in the capillary walls (AnatLoewenstein, & Coscas, 2010). Altering these forces can results to increased movement and volume of fluid between the interstitium and the vascular spaces (AnatLoewenstein, & Coscas, 2010). This interstitial fluid accumulation can lead cause the interstitial hydrostatic pressure to increase. This can lead to the formation of the edema fluid. The lymphatic system plays an important role of returning the interstitial fluid to the vascular system. Venous obstruction can interfere with this process and result to the accumulation of the interstitial fluid in the part of the body (AnatLoewenstein, & Coscas, 2010). Impaired venous drainage usually occurs due to a localized obstruction. It is caused by a neoplastic or

inflammatory condition (AnatLoewenstein, & Coscas, 2010). For instance, a massive edema of the external genitalia and lower extremity can occur as a result of a parasitic *filariasis* infection. Obstruction and infiltration of the superficial lymphatics can also cause edema in the overlying skin. Venous obstruction can also occur following a therapy complication (AnatLoewenstein, & Coscas, 2010).

Decreased colloidal Osmotic pressure.

Under normal circumstances, more than half of plasma protein is constituted by albumin (AnatLoewenstein, & Coscas, 2010). Therefore, inadequate amount of albumin in the plasma protein or reduced synthesizing of albumin can lead to decrease colloidal Osmotic pressure (AnatLoewenstein, & Coscas, 2010). For instance, in the nephritic syndrome, the damaging of glomerular capillaries causes the albumin and other plasma proteins to be lost in the urine resulting to edema. Reduced albumin synthesis can occur settings of protein malnutrition, and severe liver diseases such as cirrhosis. Regardless of its cause a reduction in level of albumin leads to reduced intravascular volume, secondary hyperaldosteronism, and hypoperfusion (AnatLoewenstein, & Coscas, 2010).

The normal mean pulmonary capillary hydrostatic pressure ranges between 8 and 10 mm Hg while the plasma colloid osmotic pressure is 25 mm Hg (Wassermann, 2008). Clearly, the mean pulmonary capillary hydrostatic pressure is relatively lower than plasma colloid osmotic pressure. This difference in the two pressures is important because the net fluid absorption is favored by the low hydrostatic pressure within the pulmonary capillaries (Wassermann, 2008). This advantage is offset by Alveolar surface tension (Wassermann, 2008). The result is the net force that favors a continuous flow of fluid out of the pulmonary capillaries into interstitial

spaces (Wassermann, 2008). The excess fluid travels via the interstitium to the peribronchial and perivascular spaces of the lungs (Wassermann, 2008). From the lungs, the fluid travels through the lymphatic channels into the lymphatic system. Pulmonary edema occurs following the accumulation of excess fluid in the alveoli and interstitial spaces of the lungs (Wassermann, 2008). It occurs as a result of capillary filtration exceeding the rate at which the fluid is removed from the interstitial spaces (Wassermann, 2008).

Pulmonary edema is the hallmark of ARDS (Acute Respiratory Distress Syndrome) and it largely associated with high surface tension (Anat Loewenstein, & Coscas, 2010). It is a serious medical condition because it causes excessive flow of fluid into the alveoli hindering gas exchange. This causes the arterial PO_2 to fall below the normal range. It also leads to rises arterial PCO_2 (AnatLoewenstein, & Coscas, 2010). Abnormal high arterial PCO_2 can cause hypercapnia and abnormal low PO_2 can lead to hypoxemia (Fishman, 2008). Edema can obstruct the airflow and increase airway resistance (Fishman, 2008). The compliance of the lung decreases with the decrease in pulmonary edema (AnatLoewenstein, & Coscas, 2010). This is because of increased interstitial swelling and alveolar surface tension (AnatLoewenstein, & Coscas, 2010). Always obstruction and a decrease in lung compliance can greatly increase the rate of breathing (AnatLoewenstein, & Coscas, 2010). This means that in treating pulmonary edema, the focus should on capillary hydrostatic pressure (AnatLoewenstein, & Coscas, 2010). This can be achieved by decreasing the blood volume, increasing the left ventricular functions, and increasing vasodilation (Fishman, 2008).

The two complications more often associated with edema, are respiratory failure and respiratory fatigue (Fishman, 2008). These complications can be prevented through prompt

diagnosis and treatment (Fishman, 2008). However, the physicians should be ready to provide assisted ventilation in case the patient starts shows some signs of respiration fatigue such as worsening arrhythmia, diaphoresis, fatigue and lethargy (Fishman, 2008). Another concern is the cardiac arrhythmia, which if not handled well; it can lead to sudden cardiac death (Fishman, 2008). This condition can be prevented through monitoring of the rhythm of the heart (AnatLoewenstein, & Coscas, 2010). Counseling and educating patients on how to prevent edema can also help prevent the recurrence of the disease (Fishman, 2008).

In conclusion, Edema occurs as a result of the excessive accumulation of body fluid in the body tissues. It is caused by: capillary hydrostatic pressure, increase in permeability of capillary membrane or alveolar and high surface tension. The three causes of edema that result from an increased capillary pressure are increased vascular volume, venous obstruction, and decreased colloidal Osmotic pressure.

References

Anat Loewenstein, C., & . Coscas, J. (2010). *Macular Edema: A Practical Approach*. New York, NY: Karger Medical and Scientific Publishers.

Fishman.P. A. (2008). *Pulmonary edema*. New York, NY: American Physiological Society.

Wassermann, S. (2008). *Acute cardiac pulmonary edema*. New York, NY: Thomas.