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18/MHS01/121

ANATOMY

SYSTEMIC HISYOLOGY ANA 204

Q Critically examine the renal function of desert dwellers and the anatomical basis of their unique adaptation

1. **Desert dwellers tend to have long loop of henle-** long loops of henle operate as counter-current multipliers, producing highly concentrated urine. There is a higher number of juxtamedullary nephrons which have long reach loops of henle that penetrate deep into the medulla than cortical nephrons which barely penetrate the medulla. Desert dwellers such as the Camels which produce highly concentrated urine have more of the juxtamedullary nephrons. Blood first flows along ascending limb of Henle, which is impermeable to water. Solutes can leave the filtrate and enter the blood along this stretch. When this blood flows along descending limb, water is reabsorbed from filtrate but not the solutes. Longer the Henle's loop, more amount of solute will be reabsorbed and hence more amount of water could be removed from filtrate.
2. **The thickness of medulla-** The number of nephrons increases as body size increase, which in turn increases the relative amount of cortex at the expense of medulla. This means that smaller desert dwellers i.e desert rats will have a more concentrated urine than bigger ones i.e camel. The relative thickness of the medulla is related to urine-concentrating ability because the medulla contains loop of henle. The thicker medulla of small desert rodents could therefore be viewed as a desert adaptation superimposed on a body size dependent pattern. Most loops of

Q Write extensively on the clinical importance of the glomerular filtration barrier

Glomerular filtration barrier determines the composition of plasma ultrafiltrate.

It restricts the filtration of molecules primarily on the basis of size. In general, molecules with a radius smaller than 20 Å are filtered freely, molecules larger than 42 Å are not filtered, and

molecules between 20 and 42 Å are filtered to various degrees. For example, serum albumin, an anionic protein that has an effective molecular radius of 35.5 Å, is filtered poorly. Because the filtered albumin and other small proteins normally are reabsorbed avidly by the proximal tubule, almost no protein appears in the urine of persons with normal renal function.

Pathophysiology/Pathogenesis

Nephritic Syndrome

Glomeruli appear enlarged and relatively bloodless. There is presence of mesangial cell proliferation and increase in mesangial matrix. Crescents and interstitial inflammation. There could also be edema that leads to puffy face, ascites and anasarca.

Hematuria proteinuria red blood cell casts is seen. There is eventually a blockage in renal capillaries and decreased glomerular filtration rate.

Management could be directed through antibiotic therapy.

Glomerular filtration barrier

The glomerular filtration barrier functions as a highly organized, semipermeable membrane preventing the passage of the majority of proteins into the urine. The glomerular filtration barrier consists of the fenestrated endothelium, the glomerular basement membrane, and the podocyte foot processes, which are connected by a slit-diaphragm. The filtration barrier normally acts to retain protein inside the lumen of the capillaries separate from the urinary space; however, defects in the podocytes affecting the feet, tight junction (podocin, nephrin), and the slit diaphragm signalling, actin cytoskeleton, and cell matrix interactions have been identified in causing a breakdown of this barrier.

The glomerular filtration barrier has several layers.

The first is a glycocalyx made up of proteoglycans and an adsorbed layer of plasma proteins that is located between the endothelial cells and the capillary lumen.

Fenestrated endothelial cells form the next layer. Next is the thick glomerular basement membrane (GBM), which is synthesized by podocytes and endothelial cells and has an inner layer composed of collagen type IV and laminin sandwiched between layers of heparin sulphate. Podocyte foot processes line the epithelial side of the GBM; the intercellular junctions between adjacent foot processes are closed by the slit diaphragm, a specialized

intercellular junction that acts as a molecular sieve and the final component of the filtration barrier. The slit diaphragm comprises several proteins, including nephrin, CD-associated protein (CD2AP), podocin, the tight junction protein ZO-1 (zonula occludens 1), P-cadherin, catenins, and the calcium channel TRPC6 (transient receptor potential cation channel, subfamily C, member 6), each of which is required for slit diaphragm integrity. Slit diaphragm proteins are supported by the highly dynamic podocyte actin cytoskeleton that in turn is anchored to an integrin complex that fastens each podocyte foot process to the GBM.