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An Overview on Renal Pathology of Obesity

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Description

Obesity affects the kidneys in a variety of ways, including structural, hemodynamic, and metabolic changes. The majority of these are presumably compensatory reactions to obesity's systemic increase in metabolic demand. Renal damage, on the other hand, can manifest clinically as a result of compensatory failure in some circumstances. The most well-known of these diseases is obesity-related glomerulopathy. The intensity and number of obesity-related illnesses or consequences, including components of metabolic syndrome, as well as the mismatch of body size to nephron mass due to congenital or acquired nephron reductions, may predispose obese people to renal compensatory failure and injury.

Obesity can cause health problems in the kidney, which is one of the organs that might be affected. The kidney disease category of Obesity-Related Glomerulopathy (ORG) is based on a biopsy diagnosis that can emerge as a result of obesity. ORG's detailed clinicopathologic observations have contributed significantly to our understanding of obesity-related renal problems. Glomerulomegaly with perihilar focal segmental glomerulosclerosis is a common renal histopathologic feature in ORG, which has long been thought to be a single-nephron glomerular hyperfiltration state.

This theory was recently proven in ORG patients by employing a combination of image analysis and biopsy-based stereology to estimate single-nephron glomerular filtration rate. Overshooting in glomerulotubular and tubuloglomerular interactions can lead to glomerular hyperfiltration/hypertension, podocyte failure, tubular protein-traffic overload, and tubulointerstitial scarring, forming a vicious cycle of a common pathway to further nephron loss and progression of kidney functional impairment.

Obesity, which has become epidemic in the United States and other Western countries, might be compounded by hypertension, an increased risk of kidney cancer, or proteinuria. Obesity-related proteinuria is characterised by focal glomerulosclerosis and glomerulomegaly on biopsy, limited clinical edoema, and generally normal serum albumin, cholesterol, and blood pressure levels, and can develop to end-stage renal disease. In patients with preexisting nephropathy or diminished renal mass, severe obesity may be an additional risk factor.

Hyperfiltration, increased renal venous pressure, glomerular hypertrophy, hyperlipidemia, and increased synthesis of vasoactive and fibrogenic substances such as angiotensin II, insulin, leptin, and transforming growth factor-1 may all play a role in the pathophysiology of obesity-related proteinuria. These compounds may alter glomerular hyperfiltration, mesangial cell hypertrophy and matrix synthesis, as well as collagen, fibronectin, transforming growth factor- and other fibrogenic mediators of change, separately or in combination. Although angiotensin-converting enzyme inhibition has been shown to have a temporary effect on obesity-related proteinuria in people, weight loss early in the disease appears to be the most relevant therapeutic method.

Albuminuria is related with large glomeruli, thicker basement membrane, and epithelial cellular (podocyte) deformation in those with a BMI more than 40 kg/m_2. Obstructive sleep apnea also exacerbates glomerular damage, most likely due to a vasoconstrictive mechanism. In the obese state, decreased release of high molecular weight adiponectin from adipose cells exacerbates insulin resistance caused by excess fatty acids. Insulin's post-receptor signalling is potentiated by adiponectin, resulting in glucose oxidation in mitochondria. The structural and functional requirements that limit glomerular albumin leakage have been the focus of recent podocyte physiology research. Nephrin and podocin, two proteins that work together to keep slit pores between foot processes capable of retaining albumin, are part of the podocyte's architecture.

Insulin and adiponectin are required for the production of high-energy phosphates. The toxicity of fatty acids to proximal renal tubules is amplified when they bind to albumin. Individuals with obesity-related nephrotic syndrome have higher levels of albumin and fatty acids in their urine. Insulin and adiponectin are inhibited by fatty acid buildup and resistin. A greater knowledge of the association between weight and hypertension has resulted from research into cytokines produced by adipose tissue (adiponectin and leptin) and macrophages (resistin). Leptin is thought to be released after a meal to suppress the midbrain/hypothalamic hunger centres. Resistance to leptin causes an overabundance of signals to the hypothalamic sympathetic nervous system, resulting in hypertension. The discovery of a brain receptor mutation supports the idea that a central nerve reflex arc has a role in hypertension in humans.

Experimental models have lately been used to gain a better understanding of obesity-related renal impairment. Rapid weight loss after bariatric surgery may help to repair obesity-related kidney disease while also restoring normal blood pressure.

Proteinuria is a well-known consequence of morbid obesity; however it only affects a small percentage of patients. Focused and Segmental Glomerulosclerosis (FSG) is the most prevalent histologic lesion in proteinuric obese people, according to biopsies and autopsy investigations. Glomerulomegaly and less severe foot process effacement separate obesity-related FSG from idiopathic cases of FSG. Even when there is enormous and continuous proteinuria, people with obesity-related glomerulopathies do not develop the nephrotic syndrome (hypoalbuminaemia, oedema) clinically.

This clinical feature (which is also seen in other hyperfiltration-related renal diseases like reflux nephropathy or proteinuria due to

renal mass loss) is very useful in distinguishing these patients from idiopathic FSG, in which a complete nephrotic syndrome is usually present along with nephrotic range proteinuria. The reasons why people with ORG do not develop complete nephrotic syndrome are unknown, although they may be related to changes in tubular protein management and a very gradual proteinuria growth over time, which is common in ORG and other hyper filtering illnesses.