Renal Sinus Adiposity and Hypertension

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Obesity, a global health problem, has an ever increasing prevalence. Because excess weight is a major risk factor for high blood pressure, this results in a continuous rise in the prevalence of hypertension. In this issue of Hypertension, Chughtai et al identified a potential pathogenic link between renal sinus adiposity and hypertension in humans.

Adipose tissue can be divided into compartments that carry a different metabolic risk. Thoracic and intrabdominal adipose tissue have a strong relation with hypertension, whereas subcutaneous and thigh intermuscular fat tissue seem less important. Chughtai et al assessed the relation between the amount of renal sinus fat and hypertension. The renal sinus is the cavity within the concave border of the kidney. Other than fat, this sinus is composed of renal vessels, calices, nerve tissue, and lymphatic channels. In middle-aged and elderly individuals at risk for cardiovascular events (N = 205), Chughtai et al showed a quantitative relation between renal sinus fat, measured with single-section MRI, and hypertension. The study had a cross-sectional design, so cause-effect relations could not be identified. Potential pathophysiological mechanisms may, however, be deduced.

Obesity can lead to hypertension via pathways that stimulate sympathetic nerve activity (SNA), the renin-angiotensin-aldosterone system, and physical compression of the kidneys, all of which can cause sodium retention. Fat tissue has been identified as an important endocrine organ in which adipocytes secrete angiotensinogen, angiotensin II, and leptin. Leptin stimulates SNA. Norepinephrine levels are elevated in obese patients, partly because of a high caloric intake that leads to stimulation of SNA but perhaps also because of renal hypoxia. In patients with sleep apnea, SNA, as well as the renin-angiotensin-aldosterone system, may be further stimulated by renal hypoxia. Experimental evidence exists that sodium retention in obesity might be driven not only by increased renal SNA but also by increased responsiveness of the kidney to renal SNA. This suggests that, during increased renal SNA, renal sinus adiposity, for instance, by amplifying the decrease in medullary blood flow, might exacerbate renal hypoxia. Because renal hypoxia as such increases renal SNA, it is conceivable that renal sinus adiposity initiates a detrimental positive feedback on blood pressure control.

Insulin resistance might also form a link between obesity and hypertension, because it may lead to sodium retention and an increase in SNA. However, the evidence to support this is mainly from experiments in rodents, and conflicting data have been found in humans. Perivascular adipose tissue may have a direct effect on vascular function and insulin sensitivity. However, other than such general effects, renal sinus adiposity may influence renal function and blood pressure directly by local physical constraints (Figure). Experiments using renal vein constriction have demonstrated that sodium retention can also be the result of increased renal interstitial pressure.

Weight gain is associated with renal hyalinosis. Expansion of the renal extracellular matrix can lead to compression of renal blood vessels and tubules attributed to the increased interstitial fluid hydrostatic pressure. This may slow the flow of blood and urine through the papilla, which could lead to a rightward shift of the pressure-natriuresis relation. A vicious circle can subsequently arise as the higher blood pressure again induces renal hyalinosis. Renal sinus fat may also cause hypertension by compression of the collecting ducts or veins. Venous constriction has been shown to lower glomerular filtration rate, urine volume, and osmolality and solute excretion.

Finally, differences in adipocyte function have been described between subcutaneous and visceral fat. It might be that renal sinus fat as an exocrine tissue differs from other intraperitoneal fat, but it seems more plausible, as extensively discussed by Chughtai et al, that its specific relation with hypertension is attributable to mechanical rather than exocrine effects, as suggested previously.

Assessment of renal sinus fat might help to identify patients with hypertension who would benefit most from weight loss. It has been suggested that dietary changes, notably, reducing salt intake, render more effect than weight loss itself. However, studies by Rocchini et al have shown that weight loss corrects the pressure-natriuresis relation. Recently, a large population study from China demonstrated that salt sensitivity was linearly related to the number of metabolic risk factors and, thus, to the degree of obesity. Hence, salt sensitivity of blood pressure may be a function of renal sinus adiposity.

To assess whether a cause-effect relation indeed exists, future clinical studies should use weight loss programs to evaluate the relation between renal sinus fat and blood pressure in a prospective design. Experimental studies should assess whether decreasing renal sinus adiposity, for instance,
by careful local liposuction, lowers blood pressure independent of changes in other fat compartments.

Disclosures
None.

References
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