Renovascular Hypertension for the USMLE Step One Exam





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Stenosis Physiology and Related Pathology

The Key Players in *Reno-vascular* HTN

Vascular Pathology

Hyperplastic arteriolitis





Arteriolar Hyalinosis



Nephrosclerosis

Renovascular Disorders (key derivative topics)

- Malignant HTN (physiology, pathology, pharmacology)
- Arteriolosclerosis → Nephrosclerosis (pathology)
- Renal Artery Stenosis (physiology and consequences)
 Fibromuscular Dysplasia (diagnostics, pathology)
 - Takayasu's Arteritis (pathology)









Arteriolosclerosis -> Nephrosclerosis



Arteriolosclerosis - Nephrosclerosis Outside *Inside (renal parenchyma)* Hyalinosis Granular Tubular atrophy, Interstitial fibrosis Cortical scarring Glomerulosclerosis

Arteriolosclerosis - Nephrosclerosis

- Background
 - **Nephrosclerosis** is a pathologic diagnosis characterized by:
 - Reduction in renal function secondary to parenchymal ischemia
 - **<u>Primary Lesion</u>**: Hyalinosis typically as a result of HTN and DM
 - <u>Presentation</u>: HTN/DM with reduced GFR (+/- *mild* proteinuria).
 - *In the clinic*: 'Longstanding diabetic/HTN with CKD *presumably* 2° to nephrosclerosis'







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Malignant → HTN Hyperplastic arteriolitis





Glomerulosclerosis



Thickening → Narrowing of lumen (obliterative arteriopathy) <u>Result</u>: Parenchymla Ischemia

*AGE: advanced glycated end products

- Pathogenesis
 - <u>Small arteries</u>: medial and intimal thickening related to hemodynamic injury. Lumen becomes narrow → focal parenchymal ischemia with reduction in functional renal mass.



Thickening → Narrowing of lumen (obliterative arteriopathy) <u>Result</u>: Parenchymla Ischemia

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• Pathogenesis

- <u>Small arteries</u>: medial and intimal thickening related to hemodynamic injury. Lumen becomes narrow → focal parenchymal ischemia with reduction in functional renal mass.
- <u>Hyalinization (*due endothelial injury*)</u>: extravasation of proteins (AGE) and increased deposition of BM matrix (*elaborated by smooth mm cells*)



*AGE: advanced glycated end products

Thickening → Narrowing of lumen (obliterative arteriopathy) <u>Result</u>: Parenchymla Ischemia



Glomerular sclerosis Tubular atrophy Interstitial fibrosis

- Pathology
 - <u>Artery</u>: homogenous, acellular thickening of vessel wall
 - <u>Kidney</u>: Tubular atrophy, interstitial fibrosis, glomerular sclerosis

Macroscopic



Nephrosclerosis Reduced size, cortical scarring and a granular appearance

Microscopic (nonspecific reflecting chronic vascular injury)



Arteriolosclerosis → Nephrosclerosis





Arteriolosclerosis -> Nephrosclerosis



Arteriolosclerosis → Nephrosclerosis











What happens to filtration fraction if you relieve the 'stricture' with an ACE-I?





Hypertensive (DM) Nephrosclerosis

- <u>Background</u>
 - Ischemic parenchymal injury 2° to *hemodynamic injury* (typically in setting of *HTN/DM*)
- <u>Pathogenesis</u>
 - Elaboration of *extracellular matrix* and/or *advanced glycation end products* (DM) by vascular smooth mm cells
- <u>Pathology</u>
 - <u>Micro</u>: Efferent arteriole hyalinosis (acellular, homogenous thickening) resulting in *tubulointerstitial fibrosis* and *glomerulosclerosis*
 - <u>Macro</u>: *Cortical scarring* with granular appearance and reduction in renal mass.
- <u>Physiology Derivative</u>
 - Impact on GFR and renal plasma flow (= *filtration fraction* with and without ACE-I)

Primary (Essential) Hypertension for the Boards

Older patient with diastolic heart disease

`...an extra pre-systolic heart sound is heard at the apex

'Patient with HTN is started on which of the following agents...'

LVEDP LVEDV LVEF

Α.	Inc	Inc	Dec
В.	NI	Inc	Dec
С.	NI	NI	Dec
D.	Inc	NI	Dec
E.	Inc	NI	NI
F.	N	Inc	NI



Atrium contracting against a poorly compliant ventricle (at limit of compliance)



Primary (Essential) Hypertension for the Boards



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