# Renovascular Hypertension for the USMLE Step One Exam







Howard J. Sachs, MD Associate Professor of Medicine University of Massachusetts Medical School <u>www.12DaysinMarch.com</u>; Season III <u>E-mail</u>: Howard@12daysinmarch.com





- <u>Background</u>
  - Prototypic condition for hypoperfusion of one kidney



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- Presentation:
  - HTN in (*older*) patient with vasculopathy/ASCVD (i.e. CAD, PVD, TIA/carotid)
  - 'Play Media'  $\rightarrow$  Abdominal Bruit (in a vasculopath)





#### Renovascular Disorders: Renal Artery Hypoperfusion

- Presentation:
  - Young patient with refractory HTN; angiogram shown
    - Beaded appearance
  - Young Asian woman without a palpable pulse; angiogram shown
    - Segmental stenosis with aneurysm formation





Takayasu's Arteritis

- Pathology
  - <u>Gross</u>: unilateral shrunken kidney
  - <u>Micro</u>: <u>atrophy</u> (tubules, glomeruli), <u>fibrosis</u> (interstitium, tubules)





<u>Glomeruli</u>: atrophy, sclerosis <u>Tubulointerstitial</u>: atrophy, fibrosis

- What is RAA doing in the the hypoperfused kidney? Activated
- What is RAA doing in contralateral kidney? Suppressed
- What happens to the filtration fraction? Balanced (*reduced GFR and RPF*)



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- Background
  - *Prototypic* condition for hypoperfusion of one kidney
- Presentation:
  - HTN in (older) patient with atherosclerosis/vasculopathy (i.e.CAD/CABG, TIA/carotid)
  - 'Play Media'  $\rightarrow$  Abdominal Bruit (in a patient with vasculopathy)
  - Fibromuscular dysplasia/Takayasu younger (female) patient
- Pathology
  - <u>Gross</u>: *unilateral shrunken* kidney
  - <u>Micro</u>: *atrophy* (tubules, glomeruli), *fibrosis* (interstitium, tubules)
- <u>Derivatives</u>
  - What is RAA doing in that kidney? *Activated*
  - What is RAA doing in contralateral kidney? *Suppressed*
  - What happens to the filtration fraction? No  $\Delta$  (*balanced reduction in GFR and RPF*)

## Fibromuscular Dysplasia (Non-inflammatory, non-atherosclerotic angiopathy)



## Fibromuscular Dysplasia

(Non-inflammatory, non-atherosclerotic angiopathy)

#### • <u>Background</u>:

- Common cause of renovascular HTN in *children* and *young adults*.
- May be bilateral AND present in other arteries (carotid/vertebral)
- *When to suspect*:
  - HTN that is severe accelerating or refractory in a young patient.
  - Bruit may be present
  - Significant drop in GFR ( $\uparrow$  Cr) with initiation of ACE-I (ATII dependence)

Similar *physiology* and *presentation* to RAS (*so questions can overlap*) **EXCEPT** these are younger patients with different pathology, arteriogram and lack of vasculopathy/ASCVD

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Diagnosis: Arteriogram



Renal Arteriograms for the Boards

## Fibromuscular Dysplasia

(*Non-inflammatory*, *non-atherosclerotic angiopathy*)

- Pathology
  - <u>Vessel</u>: Fibromuscular thickening that may involve the intima, media\* or adventitia.
  - <u>Kidney</u>: diffuse ischemic <u>atrophy</u> (glomeruli, tubules and interstitial fibrosis).



#### Renovascular Disorders (key derivative topics)

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

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