CASE STUDY #1

Management of HTN and Proteinuria in CKD Patients

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CASE

- 57 yr old Black male with DM for 4 years and HTN for 13 years.
  SCr 2.1 mg/dl, and BP 156/94 mmHg. Spot urine albumin-to-creatinine ratio = 330 mg/g. Treatment with ACEI and diuretic.
- Subsequent treatment for this patient might include the following options?

Initial Considerations

- Increase ACEI or add ARB
- If GFR < ~35 ml/min change to loop diuretic
- Add non-DHP CCB
  - If BP controlled but proteinuria persists add third RAAS inhibitor
- HR > 84: add beta-blocker or alpha/beta-blocker
- HR < 84: add third RAAS inhibitor or another class of CCB
- At each step revisit non pharmacologic aspects

Importance of Proteinuria in CKD

Microalbuminuria Predicts CV Risk at Levels Below Current Definition

LIFE Study: Composite Endpoint (CV mortality, all-cause mortality, stroke, and MI)

Microalbuminuria assessment in patients with hypertension and diabetes improves CV risk stratification


Six Month Change in Proteinuria from Baseline Predicts Outcome of Kidney Disease: Results from the AASK trial

EsrD Risk Versus Albuminuria Reduction at 6 Months
Stratified by Ethnic Group

(HR with 95% CI relative to 0% change in albuminuria: controlled for all risk markers at baseline and month-6 changes)


RENAAL - Reduction in Endpoints in Non-insulin dependent diabetes mellitus with the Angiotensin II Antagonist Losartan

Therapeutic Considerations – ACEI and Diuretic vs. ACEI and CCB

  - ACEI with a diuretic > UP reduction vs ACEI and CCB. In contrast, blood pressure reduction, particularly the diastolic component, favored ACEI and CCB.

Clinical Practice Guidelines for Management of Hypertension in CKD

Type of Kidney Disease
<table>
<thead>
<tr>
<th>Blood Pressure Target (mm Hg)</th>
<th>Preferred Agents for CKD, with or without Hypertension</th>
<th>Other Agents to Reduce CVD Risk and Reach Blood Pressure Target</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetic Kidney Disease</td>
<td>ACE inhibitor or ARB</td>
<td>Diuretic preferred, then BB or CCB</td>
</tr>
<tr>
<td>Nondiabetic Kidney Disease</td>
<td>Diuretic preferred, then ACE inhibitor, ARB, BB or CCB</td>
<td>None preferred</td>
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Kidney Disease in Kidney Transplant Recipient

CCB, diuretic, BB, ACE inhibitor, ARB


Therapeutic Considerations - HTN and Proteinuria in CKD Patient

- At start-up or change RAAS inhibition, check creatinine and potassium within 2 weeks.
- If eGFR decreases >30% over 4 months, then decrease or discontinue RAAS inhibition.
  - Consider renal artery stenosis (RAS).
- If Ê↑, get dietary counseling, diuretics (adjust for GFR), HCO₃ replacement; avoid NSAIDs & herbals.

Algorithm for Achieving Target BP Goals in Hypertensive Patients with CKD

1. Start RAAS inhibition
2. Titrate upwards
3. If BP still not at goal (130/80 mm Hg)
4. If baseline pulse<84
   - Add low-dose beta-blocker or alpha/beta-blocker
5. If baseline pulse≥84
   - Add Thiazide Diuretic or long-acting CCB

6. If proteinuria present (>300 mg per day) increase RAAS &/or add non-DHP CCB; reduce Na intake

ADDITIONAL CONSIDERATIONS

- Role of Na intake
- Meta-analysis data on ACEI in nondiabetic CKD with low UP
- Role of aldosterone & renin blockade as part of RAAS inhibition
- Role of dual ACEI/ARB vs one alone
- Other?
**SALT REDUCTION ON BP and URINE PROTEIN EXCRETION**


**COMMUNITY HEALTH**

**ALDOSTERONE/RENIN BLOCKADE**

- Strengths: 24 hr bp, Na & Pr intake; Caveats: GFR > 100; UP increased with double therapy

**DUAL ACEI/ARB vs ONE AGENT ALONE**

**Case Study #2**

Keith C. Ferdinand, MD, FACC, FAHA
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Chief Science Officer, Association of Black Cardiologists, Inc.

**PATIENT:** D.B.
**SEX:** M
**AGE:** 45
**HEIGHT:** 57 inches
**WEIGHT:** 198 pounds

**PROBLEM LIST**
1. Hypertension with recent presentation with hypertensive emergency on 4/20/2009 requiring IV drugs which were then converted to p.o. medications.
2. Cardiomyopathy with ejection fraction of 10-20%.
3. Left heart cath with coronaries scheduled in 2 days.
4. Chronic renal insufficiency. Baseline creatinine 1.8 in 2008 is now 2.4 to 3.0 on multiple examinations.
5. Iron deficiency anemia with an iron saturation of 16%, hemoglobin 12 and MCV in the 80s.
6. Dyslipidemia.
7. Prior tobacco use, quit years ago.
8. Stroke in 05/2009 for which he received tPA and had a positive CT head and MRI scan.

**MEDICATIONS**
1. Warfarin
2. Carvedilol
3. Amlodipine
4. Clonidine
5. Rosuvastatin
6. Iron sulfate
7. Aspirin

**HISTORY OF PRESENT ILLNESS**
The patient suffers from severe hypertension for over 20 years. He has intermittently been on medications until he recently presented with hypertensive emergency and began aggressive medical regimen. Despite his aggressive regimen presented with a stroke subsequently one month later in May. For this he was started on warfarin after receiving tPA and being diagnosed with left atrial smoke. The patient has a cardiomyopathy as well. The patient is doing well with no chest pain, shortness of breath, dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea or lower extremity edema. He has no palpitations, lightheadedness or dizziness either; however, he does have some limitation in his exercise tolerance and early fatigue. The patient reports compliance with his medications except for the clonidine which he found cumbersome and is not using. His INR checked regularly and is therapeutic. He has limited insight into his disorder and does not even recall being told what is wrong with his heart. He thought it was pumping "too much."

**REVIEW OF SYSTEMS:** Negative except for HPI
**FAMILY HISTORY:** Sister, brother, mother and father all have or had hypertension. His mother died at 43 from a CVA. His father died at 59 from a myocardial infarction.
**SOCIAL HISTORY:** Prior tobacco. No alcohol or substance abuse. He is unemployed, used to detail trucks. He is unmarried and has one child who is grown.
**PAST MEDICAL HISTORY:**
**PAST SURGICAL HISTORY:** No other remarkable findings.

**PHYSICAL EXAMINATION:**
**GENERAL:** He is a well-appearing African American male in no acute distress. HEENT exam: No bruit. JVP less than 8 cm. Cardiovascular exam: regular rate and rhythm. Normal S1, S2. Precordial S4. Grade 2/6 holosystolic LSB to apex murmur. No rubs or S3 gallop. Lungs are clear to auscultation bilaterally. Abdomen is soft, nontender with negative McBurney. Lower extremities have no edema and 2+ DP and PT pulses. Nerves exam is non-focal today.
**LABORATORY DATA**

Echocardiogram from 04/2009 with an ejection fraction of 5-10%, global hypokinesia. Severely decreased LV function, ejection fraction, mild mitral regurgitation. He has an abdominal ultrasound with no aortic aneurysm, renal ultrasound with mild atrophy. Head CT 04/2009 with old lacunar infarct. MRI from 05/03/2009 with small infarct posterior lobe. Chem-7 potassium of 4.3 and creatinine of 2.6. CBC remarkable for hemoglobin of 12.4 and MCV 86. He is HIV negative. His INR was 2.16 on 5/11/2009.

**ASSESSMENT/PLAN**

The patient suffered consequences from severe uncontrolled hypertension including a cardiomyopathy and stage 3 chronic kidney disease. The patient is willing to undergo aggressive medical therapy and close follow-up for this purpose.

Does he need to cancel the cardiac cath? Change BP meds? Any other interventions?