Renovascular hypertention(RVH)

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DEFINATION:

- Syndrome of elevated BP(systolic or diastolic)produced by condition that interferes with the aretrial circulation of the kidney.

- Majority of patients have RAS with reduced renal perfusion pressure.

- Most conditions cause reduced perfusion to one kidney while a second “contralateral”kidney is exposed to elevated systolic pressure ,called”two – kidney “hypertention.

- When both kidneys are affected,as may occur with atheroembolic disease or a RA stenosis in a solitary functioning kidney without a normal “contralateral”kidney,the designation”one kidney”RVH is given.

Renal artery stenosis:

- 1) fibromuscular dysplasia(FMD)
- 2)Atherosclerotic renal vascular disease.(ASRVD)

Ischemic nephropathy \ ischemic renal disease

- Refers to decreased GFR associated with reduced renal blood flow beyond the level of autoregulatory compensation.

- Critical RA stenosis can lead to renal atrophy and progressive renal impairment.

- Collateral renal blood supply can preserve renal viability in the face of proximal atherosclerotic renovascular occlusive disease and small vessel and parenchymal disease often coexist with main RA atherosclerotic disease.

- Renal revascularization of ischemic kidneys in some cases can succeed in recovering renal function.

PATHOPHYSIOLOGY

- Renovascular occlusive disease from any cause can activate pressor pathways that tend to restore renal artery perfusion pressures.
- Central to this process is the release of renin from JG apparatus,leading to activation of the RAAS.
- This is mediated in part by stimulation of neuronal nitric oxide synthase and cyclooxygenase-2 in the macula densa.
- Studies in transgenic mice without receptors to angiotensin confirm that development of RVH requires an intact RAAS.

- Mechanisms responsible for sustained RVH differ depending on whether one or both kidneys are affected by significant stenosis,either pathologic or created in animal models using clips.

- The nomenclature distinguishes between a situation in which one clip is present with a normal contralateral or unclipped kidney(“1-clip-2kidney HTN”)and a situation in which the entire renal mass is affected with no contralateral kidney(“1-clip-1- kidney HTN”).

- The presence of a normal contra-lateral kidney allows pressure natriuresis to occur,in which the elevated perfusion pressure mediates natriuresis in one non-stenotic kidney.
- Because the non-stenotic kidney functions to eliminate the excess sodium ,the level of perfusion to the stenotic side remains reduced,leading to sustained activation of the RA stenosis .this sequence of events produces Ang.ll – dependent HTN and secondary aldestrone excess with hypokalemia.

- By contrast,1-clip1- kidney HTN represents a model in which the entire renal mass is exposed to reduced pressures beyond a stenosis.
- There is no normal or nonstenotic kidney to contract increased systemic pressures.
- As a result,sodium is retained and blood volume expanded ,which eventually feeds back to inhnbit the RAAS.
- Therefore,1-clip-1-kidney HTN is typically not angiotensin dependent unless removal of volume is achieved that reduces renal perfusion pressure and activates the RAAS.

- These differences have clinical implications.
- Many diagnostic studied used to evaluate the functional significance of RA lesions depend on comparisons of the different physiologic response of the two kidneys,which may give a false impression if both kidneys are involved.
- Furthermore, diagnostic tests that depend on differences in responses to alternation in sodium status(e.g.,measuring renal vein renin after sodium depletion or individual kidney sodium reabsorption)may be problematic,because high level of Ang ll and aldosterone stimulate sodium reabsorption in both the stenotic and the nonstenotic kidney.

ATHEROSCLEROTIC RNOVASCULAR DISEASE

- Older patients(>50 years)
- Associated with systemic atherosclerosis.
- Most common cause of RVH.
- Atherscleroticplaque often arises in the first or second centimeter of renal artery or may extend from the aorta into the ranal astium.
- Associated with coronary,cerebrovascular,peripheral vascular, and aortic disease.

- Predictores of ASRVD include a history of HTN,presense of renal functional impairment,coexisting vascular or coronary artery disease,the presence of abdominal bruits.and a history of smoking.

- RA lesions are bilateral in 20%TO 40% of such patients.
- Overall prevalence of 4%to 20%with progressively higher rates for those older than 60 years(25%to 30%) and 75 years(40% to 60%).
- Reported to contribute to the decline in renal function in 15% to 22% of patients reaching ESRD.

- Activation of RAAS and endothelial systems stimulate inflammation and drive fibrosis.
- Under acute conditions of reduced blood flow with persistent filtration and tubular function,levels of deoxygenated hemoglobin increase in the renal medulla,representing medullary hypoxia.
- Reduction in GFR and associated energy-dependent solute transport allow “adaptation”to reduced blood flow without development of tissue hypoxia.
- Only when more severe vascular occlusion develops beyond the limits of such adaptation can one identify overt cortical ischemia associated with increased deoxyhemoglobin.
- This results in macrophage accumulation with progressive tubular cell loss and fibrosis.
- Glomerole are preserved but collapsed.

Clinical manifestaions of RVH

- Have higher nocturnal pressures(“nondipper”)
- Have more severe target organ manifestations LVH.
- Rarely may be associated with nephrotic range proteinuria,which can regress with correction of vascular lesions.
- Occlusion may only gradually produce BP increase may cause a repid increase in HTN that precipitates a hyprtensive urgency or emergency.
- Syndroms of polydipsia and accelerated HTN with hyponatremia and hypokalemia , sometimes attributed to the dipsogenic actions of ang II , also have been obsereved.

IRD(ischemic nephropathy)

- Should be considered as a cause of CKD in the ATS age group,particularly when other vascular disease is detected.
- Clues to IRD include asymmetry size or recent deterioration of renal function,as opposed to slowly progressive CKD.
- An asymmetrically small kidney in an adult over age 50 has a 70 % chance of being association with ipsilateral RA stenosisi.
- The development of renal impairment in pt. treated medically for longstanding HTN should raise suspicion for possible IRD.
- The most common presenation of ASRVD is unilateral involvement in a pt. with CKD associated with long standing HTN.

The kidney supplied by stenotic vessels may have reduced blood flow ,resulting in diminished GFR,RVH and atrophy.

The contaralateral kidney with a patent renal vessels often hypertrophies and compensates with hemofiltration.however overtime this kidney developed parenchymal enjury.

Acute kidney injury after starting antihypertensive /RAAS blockade pt.s with hemodynamically significant RA stenosis

- The sudden reduction in SBP in the pt. with critical RA stenosis may reduce RA pressure below levels needed to sustain GFR by autoregulatory.
- This can occur with reduction in BP by any antihypertensive agent,
- With medications that block the RAAS. ,alternations in glomerular hemodynamics may result in acute reduction in GFR, which is independent of effects on SBP.
- Normally activation of ang.II cause efferent arteriolar vasoconstriction,which preserves transcapillary filtration pressures at the glomeruius when preglomrular pressure are reduced ,thereby maintaining GFR.
- The loss of this compensatory mechanism induced by agents that inhibit or block the RAAS can result in functional AKI.

This typically occurs whithin a few days from the start of therapy and is usually ,but not always,reversible.

In pt,s without RA stenosis,AKI can also occure with use of RAAS inhibitors.this most frequently occurs in pt, with cardiac or hepatic dysfunction or pt.s with intravascular volume depletion because in the setting GFR is also ang II dependant

The observation of 20%increase in serum cr. Or more after administration of an ACEI detected most cases of bilateral severe RA stenosis.

Because of the potential chang in GFR kidney function should be rechecked whith in 1 to 2 weeks of instituting therapy with RAAS blockers

“flash”pulmonary edema

- Some pts. Develop sever HTN and ECF excess caused by impared pressure natriuresis.
- Such condition may produce the sudden “flash” onset of pulmonary edema in associaten with rapid development of circulatory congestion.
- This has been attributed in part to rapid loss of contractile strength of left ventricle cause by sudden increases in afterload.
- Up to 41 % of pts. With bilateral RA stenosis had a history of pulmonary edema,compare with 12% with unilateral renovascular disease.

Oligoanuric AKI superimposed on CKD

- When RA stenosis is bilateral or unilateral in the pts. With single functioning kidney ,progression to total occlusion can present as oligoanuric AKI,sometimes associated with a hypertensive emergency.
- A clinical clue to this diagnosis is abrupt onset oligoanuria.
- In this setting,the kidney paranchyma may be viable despite lack of filtration.
- Clues to renal viability include preserved kidney length and evidence of renal contrast enhancement("renal blush")seen on delayed or venous phase images during renal angiography.
- When these factors are present and clinical course is consistent with recent occlusion,there is a chance of retrieval of the renal function if revascularization is feasible clinically and anatomically.
- In this setting,urgent vascular surgical opinion should be sought.

Incidental renal artrery stenosis

- ASRVD is highly correlated with disease in both the coronary and the peripheral vasculature.
- Disease is usually identified incidentally at angiography or CT for other indications.
- Most of these pts. With coexisting ASRVD and CAD have only moderate degree of RA stenosis(50% to 75%)with minimal hemodynamic impact.
- Since no data support intervention for asymptomatic RA stenosis,the use of screening renal angiograghy at coronary angiography should be limited to pts. Who have demonstrated clinical manifestations,and in whom documentation of RA disease is likely to influence management.

Natural history

- Typically progresses over 2 to 5 years.
- Progression is usually defined as a greater than 25%luminal diameter narrowing or as severe stenosis progressing to vascular occlusion.
- Measurable loss of kidney length(>1 cm)is less common but accompanies progressive vascular occlusion.
- Changing in serum cr. Are often minimal.
- Progression is most likely in pts. With more than 60 % stenosis.it often occurs without changes in BP control.
- Therapy with statins to reduce the risk of progression and occasionally induced regression of atherosclerotic RA stenosis.

Risk of mortality

- Both ASRVD and IRD associated with limited long-term survival
- Report 3 to 5 year mortality rates of 30%to 35%in pts.with RA stenosis,largely caused by cardiovascular events or cerebrovascular accident(stroke)
- The 5 to 10 year survival rates for pts.reaching ESRD caused by IRD are also low as 18% and 5% respectively.
- Whether renal revascularization improves overall survival in pts. With ASRVD remains controversial.

Fibromuscular dysplasia (FMD)

- A noninflamatory nonatherosclerotic arteriopathy and the second most common cause of RVH.
- It usually involves the middle to distal RA or branches
- The vascular distribution of FMD involves primarily the renal and cerebral artries.
- Renal arteres are involved with FMD in 65%to 70 % of cases.bilateral RA disease is seen in 25% to 35 % of adult cases.
- Up to 65% of pts. With renovascular FMD have concominant cerebrovascular involvement.
- Less common extrarenal sites of involvement with FMD include coronary,mesenteric,celiac,splenic,aortic and peripheral vasculature.
- With two sites involved in 30%,three sites in 20 % and more than three sites in 10 %of pts.

epidemiology

- The prevalence of clinically apparent renovascular FMD is estimated at 4 in 1000,with a lower prevalence of cerebrovascular involvement of 1 in 1000.
- Female predilection with about 90% of cases occurring in woman.
- More common in whites than in blacks.
- Mean age of onset of HTN 43
- Familiar FMD occurs in 10%with an AD inheritance pattern.
- FMD may also complicate other hereditary syndromes(e.g.,alport,marfan,ehlers-danlos).

pathophysiology

- Is unknown
- Genes being investigated include collagen lll(col 3 A1),alpa1-antitrypsin,ACE and JAGGED 1(encoding a ligand for notch receptors)
- Smoking
- Hormonal influences(based on female predilection)
- Histologically,abnormal vascular wall structure is associated with irregular bands of collagen deposites and in some cases disruption of elastic membrane.

The three main types of FMD are:

- Medial fibroplasia
- Intimal FMD
- Adventitial or periarterial FMD

- Unlike ATS RA stenosis,FMD generally appears beyond the first 2 cm from RA origin.

Clinical manifestations

- Usually asymptomatic for many years,detected as an incidental finding during angiography.
- The most common adults clinical presentation is early onset HTN between ages 15 to 50 years,more often in women than men.
- Headaches,pulsatile tinnitus and bruits over the carotid arteries,epigasterium,and femoral regions are common.
- Stroke,TIA,may occur.
- It should be considered in pts. With early stroke or SAH caused by cerebral aneurysms or when extrarenal vascular aneurysms or dissection or occlusion should be considered at risk for renovascular FMD and vise versa.
- In addition FMD with associated aneurysms of the renal artery can present as renal infarction from renal artery dissection ,embolism from clot within the aneurysm or retropertoneal hemorrhage with flank pain and shock.

Natural history

- Had not been adequately studied.
- Progression of disease can be manifest as the development of new focal lesions within the same arterial bed,worsening arterial luminal narrowing within a specific lesion,involvement of a new vascular territory or development or enlargement of AVF or aneurysms.
- FMD rarely causes ESRD unless HTN remains uncontrolled

Nutcracker syndrome:

- Nutcracker syndrome is a vascular compression disorder and referes to the compression of the left renal vein between the superior mesenteric artery(SMA) and aorta. This can lead to renal venouse HTN. Resulting in rupture of thin-walled vein into the collecting system with resultant hematuria

Diagnosis of RVH

- Require both demonstration of a critical stenotic vascular lesion and activation of the RAAS.

- Common noninvasive screening modalities include:
  - RA duplex
  - Ultrasound
  - CT angiography
  - MR angiography.

Renal artery duplex scanning

- Often used to identify and to follow hemodynamic effects of vascular lesions serially.
- ADVANTAGES:
  - Relatively inexpensive
  - Require no contrast
  - Most effective in detecting of main RA near the ostium,making it better screening test for ASRVD than for FMD.
- DISADVANTAGES:
  - However,the reliability of this method depends on the skill of the ultrasonographer and on the body habitus of the pts.
  - It provides little functional information regarding the kidney beyond the vascular lesion,although many important structural features may be determind,including kidney size and presence of ureteral obstruction.

Diagnostic criteria by duplex ultrasound

- Acceleration of blood flow through the stenotic area that exceeds the flow in the aorta as well as abnormal waveforms representing blood flow in the affected vessel.
- Parameters measured using this modality include peak systolic velocity(PSV)at various sites along the aorta and renal artery
- Accelaration time and index
- Intra renal resistive index.
- The resistive index has been associated with intrinsic small vessel renal disease and a value greater than 80 has a strong negative predictive value on likelihood of BP response to intervention.
- PSV in the range of 250 to 350 cm/sec. is considered the threshold for identifying a 60 % to 70 % RA stenosis.

Magnetic resonance angiography(MRA)

- Offers the potential to provide both structural vascular imaging and functional information.
- MRA with gadolinium contrast gives excellent imaging of the main renal arteries.
- Limitation include:
  - Inter- observe variability,
  - A recognized tendency to ovestimate luminal narrowing and limited sensivity for middle and distal vascular lesions.
- Causion is advised in the use of gadolinium agents in pts. With reduced GFR based on report of nephrogenic fibrosing dermopathy in pts. With advanced CKD.

Computed tomographic angiography(CTA) WITH VASCULAR RECONSTRUCTION

- Achieves image qualities almost equivalent to those of angiography but requires more iodinated contrast.
- CTA is becoming the non-invasive study of choice in pts. Whose risk of contrast associated nephrotoxicity is minimal.
- CTA is highly sensitive for identifying lesions associated with FMD and is a good screening test for these pts., who generally have good kidney function.

Angiography:

- Remains the gold standard for defining the degree of stenosis associated with ASRVD and for identification of FMD.
- Aortoghraphy provides important anatomic and functional information in case of tight stenosis or occlusion,including the demonstration of delayed perfusion of the kidney and distal reconstitution of the proximally occluded renal vessel.
- This is important in consideration of surgical revascularization for retrival of renal function.
- In experienced hands ,selective renal angiography to identify significant RA stenosis can be performed with as little as 20 ml of iodinated contrast.
- In very high risk cases carbon dioxide can be used in place of contrast to assess the renal ostium and proximal vessel where ATS. Typically develops.

- Unfortunatly few tests can accurately predict favorable response to intervention.
- Two tests used for this purpose include
- Captopril renography
- Measuement of renal vein renin levels

Captopril renography

- This examination provides no direct image of the renal vessel
- But does provide a view of the rate of isotope appearance and washout reflecting the sequence of renal blood flow and filtration.
- The study provides functional information regarding the size and excretory capacity of the kidney, as well as emphasizing the role of ang.II in maintaining GFR.
- This test has a high negative predictive value when completely normal
- Many intristic renal abnormalities unrelated to main RA may change these curves,which limits its value in the presentce of reduced GFR.(CR.>2 MG/dl)

Renal vein renin measuements

- May help predict the BP response to renal revascularization.
- Previous studies indicaed that lateralization of renal vein level(>1.5:1 stenotic-nonstenotic kidney ratio) predics a favorable BP response for more than 90% of pts.
- Because failure to lateralization also carried a favorable response in almost half pts. The negative predictive value is limited.
- Some clinicians use these measurments to verify the role of a pressor kidney before undertaking nephrectomy

Treatment:

- Medical therapy
- Renal revascularization with PTRA
- Surgical revascularization

Medical therapy

- Most pts. With RVH are treated initially with conventional lifestyle modification,control of metabolic syndrome and anti hypertensive medications.
- Regimens using agents that interfere with the RAAS such as ACE inhibitors renin inhibitors ans ARBs as well as the use of diuretics and dihydropyridine class of calcium channel blockers allow achievement of target BP levelsin most pts.
- RAAS blockade is considered fundamental. Succesful renal revascularization rarly leads to withdrawal of all anti hypertensive.
- Therfore it may be questioned whether the cost and risk of renal revascularization are warranted for pts. Whose BP and kidney function are stable on an acceptable antihypertensive regimen.

- The RAAS blockers remove the vasoconstrictive action of ang.II at the efferent arteriole.
- When preglomerular pressures are reduced for any reason, ang.II preserves glomerular transcapillary filtration pressure by constricting the efferent arteriole prefentially.
- This maintains glomerular filtration despite marginal blood flow.
- Inhibition of this ang II effect under these conditions can lead to AKI.
- The decrease in GFR is apparent clinically under conditions in which the entire renal mass is affected including bilateral RA stenosis or stenosis to a solitary functioning kidney.
- Although the functional decrease in GFR induced by RAAS inhibitors is usually reversible occasionally pts. Do not recover renal function.

- Thus these drugs are a double –edged sword in RVH.
- They have uique properties allowing more effective BP control than previously possible in particular situation.
- But at the same time have the potential for early loss of filtration pressure in pts. With critical levels of RA stenosis.

Renal revascularization

- Restoring the renal blood supply is a rational goal of treating HTN related to renovascular disease
- In a young person with FMD a permanent cure of HTN is sometimes achieved
- Revascularization offers such a pts.the potential for relief from a lifelong regimen of antihypertensive medications and carvdiovascular risk associated with high BP.

In practice however cures are infrequent.

More often renal revascularization allows improved BP control and stabilization of kidney circulation

Percutaneus transluminal renal angiography(PTRA) for fibromuscular dysplasia(FMD)

- currently most centers treat HTN associated with FMD with PTRA without stenting.
- Approximately 86% of pts. Require less antihypertensive medication after technically successful PTRA.
- "complete cuer" defined as normal artrial pressure without medication occurs in 35% to 45 % of cases.
- Although primary technical success rate for PTRA are high(>90%)for FMD restenosis from either inadequate initial treatment or recurrent fibrosis has been reported in up to 34% of cases.
- This appears to be most common with "string of beads"angiographic variant which has multiple areas of stenosis.

- When FMD is associated with large aneurysmal dilation exceeding 1.5 cm in diameter surgical revascularization has been the standard of care.
- Endovascular management of RA aneurysms sometimes can be achieved by use of “covered” stent grafts to exclude the aneurysm.
- Women of childbearing age with RA aneurysms should be treated surgically or with a covered stent graft before pursuing pregnancy because of the risk of ruptured aneurysm during pregnancy or delivery.

Atherosclerotic(ATS) disease: endovascular stents

- Primary endovascular RA stenting has become standard for the interventional treatment of ATS RA stenosis in most centers.
- Establish superior immediate and long term results with stents.
- With current techniques ,target vessel patency rates regularly exceed 95%,
- Short term use of platelet inhibitoes (e.g. clopidogrel) for sevsral weeks to prevent vessel occlusion is standard.
- Functional changes and BP changes may develop over weeks and months when antihypertensive medications can be adjusted.

- BP control rates were improved in 50% with 68 %of pts. Experiencing “stabilization” or “improvement” in renal function over a mean of 17 months.
- Pts. With deterioration of renal function after stenting likely experience complications such as cholesterol embolization or contrast nephropathy .
- Pts. With advanced CKD at intervention tend to progress over time to requiee renal replacement therapy despite technically successful restoration of blood flow.
- Common complication of PTRA include contrast nephrotoxicity which is usually reversible and atheroembolism from which pts. Usually do not recover.that 7.5% to 9% of pts. Have a major procedure related complication including local arterial dissection ,aortic dissection and segmental renal infarction.

Surgical revascularization

- Surgical intervention for RV disease is reserved for pts. Refractory to medical therapy for whom endovascular therapy failed or who have associated aortic disease that is not amenable to endovascular therapy.
- Despite these caveats, successful surgical revascularization in well selected cases provides durable restoration of kidney blood supply and long term survival.
- Overall,the effects of surgical revascularization on BP and renal function response in pts. With ASRVD mirrors those for endovascular therapy.

- Diabetic pts. From an important subgroup of those with IRD. Surgical revascularization in this group is associated with similar renal function responses but an inferior rate of BP response and higher postoperative mortality risk or eventual dialysis dependence.
- Some dialysis-depente pts.and some with advanced CKD with IRD experience recovery of renal function after surgical revascularization.
- The best predictor of successful and sustained withdrawal from dialysis is a rapid and recent peroperative decline in GFR,often associated with occlusion of critically stonosic main RA and a kidney preserved size and extensive collateral supply.
- Some pts. Develops RVH associated with total occlusion of a preexisting RA stenosis resulting in nonfunctioning of the kidney.

Realistc outcomes and contravrsis with renal revascularization

- For some pts. Technically successful renal revascularization leads to better BP control and improved kidney function.

- Antihypertensive medication requirement decrease ,although rarly are eliminated entirely.

- Remarkably, the limited prospective data comparing medical therapy with renal revascularization in the current era at most have demonstrated modest benefit regarding renal or cardiovascular outcome for ATS.disease
- Three small randomized controlled trials in the 1990 could identify only minor differences in BP and renal outcomes.
- Despite the lack of prospective trials,application of endovascular RA stentingin the united state rose more than fourfold between 1996 and 2005.
- Most series report stabilization of renal function meaning that average serum cr. Levels do not change.

- Some pts. Experince improved renal function whereas others have clinically significant loss of renal function .in most series this occurs in up to 18% to 20% of pts.treated either with PTRA or surgery .

- Although group average value do not change ,some pts. Experience advese effects on renal function that should be considered for management decisions.

DRASTIC

- The most prominent was dutch renal artery stenosis intervention cooperative study group(DRASTIC).it included 106 pts. With relatively resistant hypertention randomized to medical therapy or PTRA.
- The lack of difference in BP after 1 year between pts. Treated with PTRA and treated medically led the authors to conclude that "angioplasty has little advsntage over anti hypertensive drug therapy"
- The results of this study were analyzed under "intention- to – treat" statittcal rules, but were confounded by 22 of 50 pts. Assigned to medical therapy(44%)who cross over to the PTRA arm because of uncontrolled BP levels at 3 months.
- Despite their inclusion in the medical arm,many authorities reviewing these data argue that this group offers compelling evidence of medical treatment failure in some cases and benefit of renal revascularization for such individuals.

ASTRAL TRIAL

- Report from prospective registry data from UK and Germany suggest that revascularization provides measurable slowing of renal functional loss and a survival adventutage compare with pts. Managed with medications alone.
- ASTRAL compared medical therapy and stenting to medical therapy alone in more than 800 subjects for whom clinicians were uncertain as to whether they would benefit from revascularization.
- Results demonstrated no differences in renal function,BP,CHF, or mortality end points over several years.

CORAL TRIAL

- The recently completed CORAL trial was the largest randomized controlled trial comparing medical therapy to medical therapy plus stenting in pts. With significant renal artery stenosis and HTN or CKD.
- CORAL randomized 974 pts.to well-defined medical therapy including RAAS blockade with or without stenting.
- CORAL results showed no differences in the composite end point of mortality,cardiovascular and renal events or any of the individual components.

STAR TRIAL

- The stent placement in pts. With ATS. Renal artery stenosis(STAR) trial randomized 140 pts. To medical or stent therapy to evaluate loss of cr. Clearance over 2 years.
- This trial was limited by imprecise definition of stenosis so that 28 % of pts. Assigned too stent therapy were untreated because only trivial stenosis was found at angiography.
- A small randomized trial extending more than 9 years failed to detect a mortality benefit with surgical revascularization.

- Clinicians caring for pts. With ASRVD should embark on aggressive cardiovascular risk factor reduction,incorporate RAAS blockade in the management of HTN, individualize consideration of endovascular treatment to avoid unnecessary loss of kidney function and to limit futile interventions.

Integrated approach to treating renovascular disease

- Central to the management of pts. Renovascular disease is recognition of distinctive clinical syndromes,liking acceleration of hypertension with deteriorating renal function and occasionally episodis circulatory congestion("flash"pulmonary edema).
- Many pts. Can be managed effectively by medical means including vigrorous measures to prevent ATS progression with statin therapy and smoking cessation

- It must be emphasized that long term care of such pts. Is an ongoing process that should be rewed at reular intervals.
- When peogressively more complex antihypertensive regimens become required or renal function deteriorates or for pts. With recurrent flash pulmonary edema despite adequate medical and diuretic therapy consideration of critical vascular lesons affecting the kidneys.

- Thank you