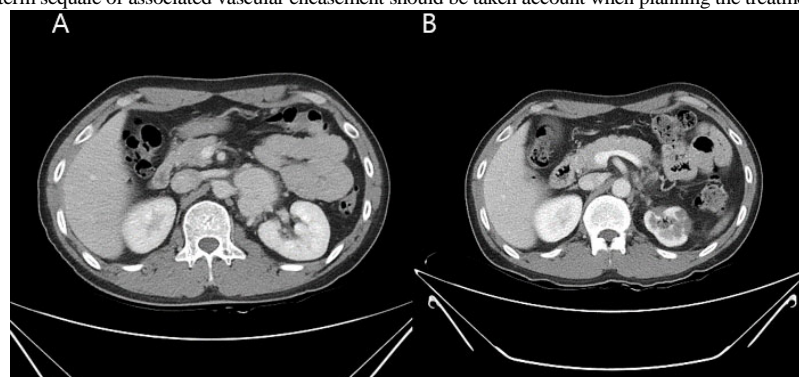


A case of renal artery occlusion in a Castleman's disease patient

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Castleman's disease is a rare lymphoproliferative disease characterized by massive tissue proliferation. Here we report a case of renal artery occlusion related to Castleman's disease which resulted in refractory hypertension. A 33 year-old male visited the endocrinologic clinic with an in suspicious adrenal mass found on computed tomography scan (CT). He had a history of cardiac surgery 15 years ago, due to atrial septal defect and tricuspid regurgitation. CT scan(A) showed 5.5cm sized well demarcated enhancing mass in left anterior pararenal space encasing renal vessels. Hormonal tests to exclude functional adrenal incidentaloma revealed to be within normal range. Lt adrenalectomy with excision of adjacent peria renal lymph node excision was performed. Pathologic result of excised lymph node was confirmed by hyaline vascular type lymphoid infiltrate with HHV-8 negative, Ki-67 positive immunostain as mixed type Castleman's disease. After recovery, he visited the clinic with hypertension which was barely controllable with three anti-hypertensive medications. Renin activity level was 7.8 times higher than prior to the surgery. CT findings(B) revealed occlusion of left renal artery with renal infarction of left upper pole. Angiography showed that the patient had two renal arteries; the left main renal artery, which was incurred with severe stenosis as a result from chronic mass effect of Castleman disease and the left accessory renal artery which supplied lower pole of left kidney. Intervention for angioplasty was failed due to chronic hard stenosis of left main renal artery. Anticoagulation with low molecular weight heparin with subsequent warfarin switching was introduced. This case is a rare case of Castleman's disease with vascular involvement which resulted in renal artery occlusion after successful surgical removal of main mass. Long term sequele of associated vascular encasement should be taken account when planning the treatment of Castleman's disease.



Relationship Between Combination of Anti-Hypertensive Drugs and Adverse Outcomes in CKD Patients.

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Background/Aims: The cornerstone therapy in patients with CKD is renin-angiotensin system blockers (RASBs). However, blood pressure (BP) is not well controlled with RASBs only and additional drugs are required. Accordingly, there has been growing interest in the combination therapy of RAS blockers with other BP-lowering drugs that can improve clinical outcome better in these patients. **Methods:** Among 2,238 CKD patients enrolled in the KNOW-CKD study, we studied 1,907 patients who were taking BP medications. Patients were classified into 4 groups: RASBs only; RASBs+calcium channel blockers (CCBs); RASBs+beta-blockers (BBs); and RASBs+CCBs+BBs. Diuretics were liberally used among 4 groups. Two main study endpoints were 1) a composite of $\geq 50\%$ decline in estimated glomerular filtration rate (eGFR), or the onset of end-stage renal disease (ESRD), and 2) a composite of cardiovascular events (CVEs) or death. **Results:** The mean age was 53.7 ± 12.0 years and 1,194 (62.6%) patients were males. During a median follow-up of 3.2 years, 310 (16.3%) and 132 (6.9%) patients reached the composite renal outcome and the composite of CVEs or death, respectively. In the fully adjusted multivariable Cox models, risk of reaching renal outcome was significantly higher in patients with RASBs and CCBs (HR, 1.55; 95% CI, 1.17-2.04; $P=0.002$) and in patients with triple therapy (HR, 2.00; 95% CI, 1.51-2.66; $P<0.001$) as compared to RASBs only. However, there were no differences in risk of CKD progression among dual or triple combination therapies and combination of RASBs with CCBs had similar risk to that with BBs. In addition, combined use of RASBs and BBs was significantly associated with a 2.95-fold and a 2.67-fold increased risk of CVEs or death as compared to RASBs only (HR, 2.95; 95% CI, 1.68-5.16; $P<0.001$) and combination of RASBs and CCBs (HR, 2.67; 95% CI, 1.51-4.71; $P=0.001$). This association was consistently observed in patients without prior history of CVEs and in those without use of diuretics. **Conclusions:** Risk of CKD progression was similar between RASBs+CCBs and RASBs+BBs. However, risk of CVEs or death was significantly higher in combined use of RASBs and BBs than in combined use of RASBs and CCBs.

Figure 1. A flow diagram of study subjects

