Page Kidney: A Case of Spontaneous Sub-Capsular Hemorrhage Secondary to Anticoagulation

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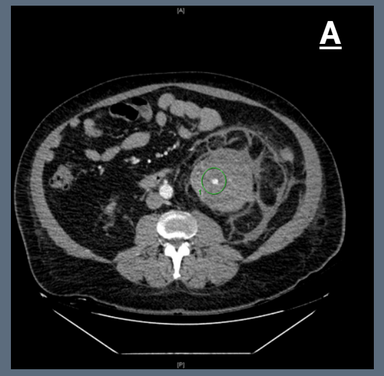
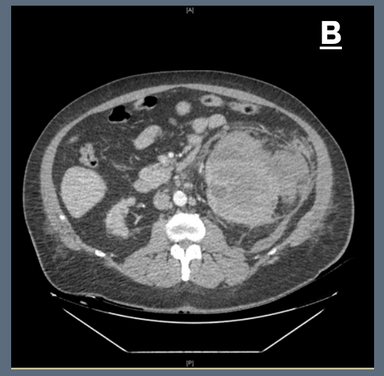
BACKGROUND

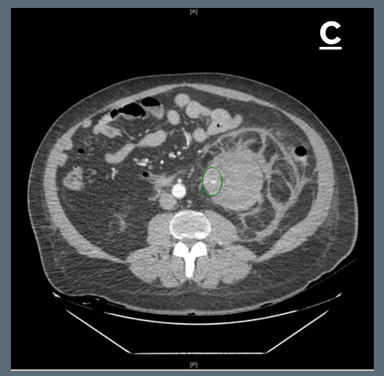
Page kidney is a rare condition caused by external compression on the kidney resulting in renal hypo-perfusion, subsequent activation of the renin-angiotensin-aldosterone system, and secondary hypertension. The etiologies of Page kidney include blunt trauma leading to a hematoma around the kidney, iatrogenic following a kidney biopsy, spontaneous from AV malformation or anticoagulation, and external compression from lymphocytes or large simple cysts. Definitive treatment relies on treating the underlying cause.

CASE PRESENTATION

52yo M with a PMH of ADPKD on HD, PHTN, a fib, CAD, and aortic stenosis s/p mechanical valve replacement on Coumadin, who presented to ACMC for sudden onset of left flank pain. Pain was described as a constant, sharp, stabbing pain, 10/10 in severity that started in his left flank and radiated to his left abdomen. He was hemodynamically stable. Labs were notable for Hgb 8.6 (bl ~10), and INR 2.4. Repeat hemoglobin was 7.4. CTA demonstrated a large left hematoma originating from the left kidney measuring 14.3 x 12.7 cm. There were multiple punctate foci of arterial blush on the arterial phase, which spread out on the venous and delayed phase, concerning for active arterial extravasation-hemorrhage and compression of renal parenchyma. He was transfused 1u of PRBC's and was admitted to the ICU for further management.







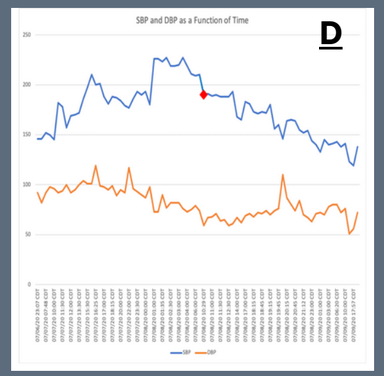


Figure A, B, and C: CTA as described in case presentation
Figure D: Graphical representation of average blood pressure readings before and after intervention. Red dot represents coil embolization

HOSPITAL COURSE

While admitted, he became progressively more hypertensive despite escalating anti-hypertensive therapy. He ultimately underwent angiography and coil embolization to stop the bleed. Following successful coil embolization, the compression of renal parenchyma was alleviated and slowly his blood pressure normalized. Anti-hypertensive medications were titrated off. He resumed Coumadin 7 days after discharge with normalization of hemoglobin to pre-admission values (~10).

CONCLUSION

Page kidney is a cause of secondary hypertension that results from external compression of the kidney, causing compression of the infra-renal vessels, hypo-perfusion, and activation of the RAAS. Our case identifies an autonomous bleed caused by anti-coagulation, causing compression of the infra-renal vessels and activation of RAAS. Our patient developed refractory secondary hypertension that is difficult to manage and can prove to be fatal. Our case is unique in that it is in a setting of polycystic kidney disease, which can already affect the architecture of the renal capsule prior to the extrinsic compression from a hematoma ever showing its effects. The combination of PKD in a patient with significant need for ongoing anti-coagulation results in a dramatic increase in incidence of this rare condition.