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What is the mechanism whereby Crohn's disease makes calcium stones in the kidney? Can you be specific about what type of calcium stones are most associated with Crohn's disease?

Crohn's disease is associated with calcium oxalate stones. Oxalate normally binds with calcium in the bowel lumen to be excreted in the stool. With fat malabsorption, such as with Crohn's disease, calcium in the bowel binds to fat more than it binds to oxalate. Oxalate is then reabsorbed at higher than normal levels and is renally excreted. These can then form calcium oxalate stones.

How does bile contribute to this phenomenon of calcium oxalate stone formation in Crohn's disease?

Bile, which helps with fat absorption, is normally excreted into the small bowel to promote fat absorption and the bile is then reabsorbed in the ileum as part of the enterohepatic circulation of bile salts. In patients with Crohn's disease that have ileal dysfunction and in patients who have had ileal resection, the bile is not reabsorbed, there is a paucity of bile to help with fat absorption, calcium binds to fat instead of oxalate, oxalate is then absorbed and renally excreted, and can form calcium oxalate stones.

Bonus question: Struvite stones classically form secondary to which bacteria?

Struvite stones are classic on board exams and you need to know that they have ammonium that results from urease in the setting of proteous and/or klebsiella.

What are top differential considerations for a spiculated mesenteric mass that is calcified?

Think desmoid tumor, carcinoid, and sclerosing mesenteritis (aka retractile mesenteritis). Carcinoid is most common about the terminal ileum and pancreas and is arterially enhancing. Retractile mesenteritis has a classic "misty" attenuation. Note that all of these have various imaging manifestations but one way they can present is with a spiculated, calcified, mesenteric mass.

What is milk of calcium and how does milk of calcium typically appear on imaging?

Layering sediments of calcium within cysts. On imaging the appearance can show "tea cupping" or a meniscus like effect with calcium sediments layering dependently within cysts.

Milk of calcium is most classic on mammography. What is the classic appearance of milk of calcium on a craniocaudal view and what is the classic appearance of milk of calcium on true lateral view?

CC= round amorphous (smudgy) calcifications. ML or LM or MLO: tea cupping/layering, especially on extended hold views (hold the patient in compression for a minute or so before you take the true lateral image to allow additional time for the calcium to layer. Key for milk of calcium is that the calcifications

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change configuration between orthogonal views. Other types of calcifications in the breast do not classically change configuration on orthogonal views.

What if grouped calcifications on a mammogram appear amorphous on orthogonal CC and MLO or ML views?

If calcifications are amorphous on both orthogonal views you have not confirmed milk of calcium and you need to biopsy. Entities like DCIS can present with amorphous calcifications.

A pathologist is examining core biopsy specimens from a stereotactic biopsy of calcifications, thought to possibly be related to milk of calcium. The pathologist does not see any calcifications in the biopsy specimen under standard microscopy. What could the pathologist consider adding in order to potentially reveal calcifications that are present but not seen on standard microscopy?

The pathologist could evaluate the biopsy specimens with polarized light which may show calcium oxalate which can be seen with benign milk of calcium. Calcium oxalate is seen under polarized light microscopy whereas calcium phosphate is the more common type of calcification seen on standard microscopy and is stained purple with H & E staining. Calcium oxalate is more likely to be benign whereas calcium phosphate can be benign or malignant.

Besides the breast can you think of another organ that can show milk of calcium on x-rays?

First, I think of renal cysts in which calcium can precipitate. Renal milk of calcium can also be seen in calyceal diverticula. The appear layering on layering on an erect film and rounded on a supine film (change configuration as also seen in mammography). You also can have milk of calcium in other places like the gallbladder but that is less likely to be tested on a radiology board exam.

What are Randall's plaques?

These are subendothelial deposits of calcium carbonate or calcium phosphate within the loops of Henle within the kidney. These appear hyperdense on an abdominal CT but are not renal stones. Key is that these are abutted by 50% or greater renal parenchyma—they are intimately associated with the renal papilla and renal parenchyma. These are often thought to denote an increased risk of stone formation.

What is a key sequence that is necessary on a triple phase CT to differentiate contrast from calcium on CT angiography, such as for endoleak evaluation?

Precontrast imaging is key on a triple phase CT to differentiate between calcium and contrast. If the hyperdensity seen on later phase images is also present on precontrast images then it is calcium. If the

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hyperdensity seen on later phase images is not present on precontrast images this is presumed to represent contrast, such as in an endoleak.

What are differential considerations for renal medullary nephrocalcinosis?

HAMHOP

H=hyperparathyroidism

A=acidosis (Renal tubular acidosis)

M=medullary sponge kidney

H=hypercalcemia/hypercalciuria (sarcoid, milk-alkali syndrome, hypervitaminosis D)

O=oxalosis (Crohn's disease, ileal surgical resection)

P=papillary necrosis

Remember a unilateral calcified kidney think TB as nephrocalcinosis would be expected to be bilateral

Bonus: What is a reasonable differential for renal papillary necrosis?

NSAID

N=NSAIDs

S=sickle cell disease

A=acetaminophen

I=infection (TB, pyelonephritis)

D=diabetes/dehydration

The calcifications with renal papillary necrosis may be curvilinear or ring-shaped (arc of calcs around the papillary tips), or triangular in appearance (more extensive calcification of the papilla). Detached triangular calcs should make you consider sloughed papilla in the setting of renal papillary necrosis.

What are characteristic imaging findings of medullary sponge kidney?

Pyramid like calcifications of the renal medulla on a noncontrast CT that develop a paintbrush like appearance on delayed post-contrast images. These patients are often asymptomatic but they can present with intermittent flank pain and/or UTI's due to ureteral obstruction by small calculi in dilated renal tubules. The medullary pyramids would also look echogenic on ultrasound.

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Episode 2

In hepatic echinococcal disease what is the significance of complete rim calcifications of the hydatid cysts?

With hepatic echinococcal disease you often get a "cyst within a cyst" or large cyst with multiple daughter cyst appearance. You can also have the "water lily sign" which is the ruptured cyst envelope floating within the fluid filled cystic space. The cysts often get peripheral calcifications and if the peripheral calcification is complete so you have complete rim calcification that often denotes inactive hydatid disease. On a plain film you can see large curvilinear or rounded calcification(s) that should make you consider hepatic hydatid disease. Random fact is that hydatid disease is endemic in countries with lots of sheep.

What is the main difference in internal calcifications between an immature and mature teratoma?

Calcifications in an immature teratoma are often irregular and amorphous. Calcifications in a mature teratoma are coarser and tooth-like. Both mature and immature teratoma have macroscopic fat which are smaller and more scattered in an immature teratoma and larger collections of fat are often seen in a mature teratoma.

If you have a renal cyst with a few thin septa under 1 cm in thickness with thin calcifications, internal blood products or protein, and the cyst is less than 3 cm in size what Bosniak classification is this?

Bosniak II (benign, minimally complex)

If you have a renal cyst with the above features but the septa contain thick calcifications, septa are increased in number, and cyst is larger than 3 cm what Bosniak classification is this?

Bosniak IIF (minimally complex, warrants follow-up, often in 6 months). F=follow-up.

Tip with Bosniak is a 1 is a simple cyst, 2 is benign with no follow-up, 2F is most likely benign but needs follow-up, 3 starts to have thickened walls and mural nodules, 4 has an overt mass associated with the cyst. Bosniak 3 and 4 need an MRI and urology consultation. Treatment is often with something like radiofrequency ablation for a Bosniak 3 and partial/total nephrectomy for a Bosniak 4 lesion.

What percentage of pheochromocytomas are calcified?

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Remember 10%. With pheochromocytomas there are lots of 10% associations to include 10% bilateral, 10% extra-adrenal, 10% malignant, 10% pediatric, 10% clinically silent, 10% autosomal dominant, 10% are calcified. Pheochromocytoma—rule of 10s. If they ask you a pheochromocytoma asking about the percentage likelihood of an association, 10% is statistically likely to be the answer.

How can calcifications help differentiate the true from the false lumen in the setting of aortic dissection?

The true lumen has calcifications around the lumen. This makes sense because we know on CT scans that calcifications, when present, are seen circumferentially in the wall of the aortic lumen. The false lumen opens up in the potential space in the aortic wall, not within the existing lumen with atherosclerotic calcifications.

Bonus question: What are other ways to tell the true from the false lumen in aortic dissection?

The false lumen is usually larger than the true lumen, enhances later than the true lumen. Thrombosis is more common in the false rather than the true lumen. The true lumen more often contains the celiac, superior mesenteric artery and right renal artery. Two facts to definitely know: 1. The left renal artery usually arises from the false lumen. 2. If you are unsure which is the true or false lumen, the larger of the two lumen is more likely to be the false lumen.

What is the significance of atherosclerosis calcifications of the ascending aorta?

Atherosclerosis typically spares the ascending aorta so if you see atherosclerotic calcifications of the ascending aorta, consider a non-atherosclerotic process such as Takayasu and Syphilis.

What are differential considerations for urinary bladder wall and/or urinary lumen calcifications?

Top differential considerations include bladder calculi, schistosomiasis and tuberculosis. Less common entities include things like amyloidosis and alkaptonuria.

What cancer is associated with urinary bladder schistosomiasis?

Squamous cell carcinoma of the urinary bladder.

Rapid fire CNS calcification questions:

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What stage of neurocysticercosis is calcified?

Stage 4 nodular calcified neurocysticercosis. This is the quiescent stage with a calcified remnant of the prior viable parasite.

Bonus: What is the most symptomatic stage of neurocysticercosis?

Stage 2: colloidal vesicular stage which is the stage at which the parasite dies, the cyst membrane becomes leaky, and there is prominent edema and enhancement.

What is the significance of calcifications in neurotoxoplasmosis?

Calcifications denote non-active treated lesions.

Bonus: In the setting of uncertainty regarding toxoplasmosis vs CNS lymphoma, thallium uptake supports which entity?

Thallium uptake would typically be seen with primary CNS lymphoma with no uptake in neurotoxoplasmosis.

What are characteristic imaging features of Fahr syndrome?

The primary imaging feature of Fahr syndrome is extensive and symmetric calcifications of the basal ganglia and thalami and calcifications can also be seen elsewhere in the white matter. Some say Fahr disease is related to the genetic form of the disease and Fahr syndrome is the secondary form of the disease which can be due to things like parathyroid hormone dysregulation, infections like HIV, and carbon monoxide poisoning.

What is a description of the calcifications in the brain associated with Sturge Weber Syndrome?

Buzzword is tram-track calcifications with gyriform cortical and subcortical parenchymal calcifications. Associated features include the classic facial port wine stain in a V1 ophthalmic division of the trigeminal nerve distribution. Childhood seizures are common.

What are some intracranial lesions that frequently calcify in tuberous sclerosis?

Cortical and subcortical tubers and subependymal hamartomas are common lesions in tuberous sclerosis that commonly calcify. Note that calcification of these lesions typically starts in childhood with the cortical and subcortical tubers often showing calcification by age 2.