

ACP Internal Medicine Board Review Questions and Answers -Tuesday, May 13, 2025

I thought Raloxifene was the superior SERM due to osteoporosis protection. Is this incorrect?

"In the treatment of breast cancer in pre-menopausal women, Tamoxifen is the preferred SERM.

The Study of Tamoxifen and Raloxifene (STAR) trial directly compared the efficacy of tamoxifen and raloxifene in reducing breast cancer risk. Tamoxifen demonstrated a 24% greater reduction in invasive breast cancer risk compared to raloxifene. Raloxifene showed comparable efficacy to tamoxifen in reducing invasive cancer risk in women with lobular carcinoma in situ (LCIS), but it was less effective in reducing noninvasive breast cancer risk.

Raloxifen is the only one that has FDA approval for prevention and treatment of postmenopausal osteoporosis. Maybe that is what you are recalling."

Soft tissue CT of neck would help define extent. Better ? I order both referral to ENT and CT soft tissue. Yes/No?

As Dr Chugh mentioned, imaging may be needed for staging eventually. This question is asking for diagnostic test, so endoscopic evaluation is the best answer as that should occur before imaging staging.

Is first diagnostic step of anal cancer work up an anal pap?

Anal pap smear is primarily used for screening in high-risk populations, although their routine use is debated. Anoscopy with biopsy is typically used in the diagnosis of anal cancer.

What is the role of prednisone in prostate cancer?

Prednisone is typically used in combination with other agents in the treatment of metastatic castration-resistant prostate cancer

Can you comment on the approach to PSA screening tools?

Recommendations on the use of PSA for screening varies depending on the society- USPSTF recommends using shared decision making for men aged 55 to 69 years (Grade C recommendation). Other societies, such as NCCN or ACS vary slightly.

Do we ever perform a biopsy for renal cancer?

Biopsy is used in diagnosis, especially for confirming RCC and determining its subtype. The radiology guidelines especially highlight the utility of biopsy for indeterminate lesions on imaging: <https://pubmed.ncbi.nlm.nih.gov/33153554/>

What does IGHV stand for?

IGHV- immunoglobulin heavy chain variable, referencing a gene.

Is insurance coverage for BRCA I / II testing improving any? Most policies DO NOT cover it.

This is likely beyond the scope of the board review. A quick review shows that specific criteria must be met for Medicare and private insurances, which may differ depending on the policy. Medicaid coverage varies by state.

When are we going for a biopsy of primary tumor vs something like an FNA of the farthest tumor to aid with staging?

As Dr Chugh mentioned, we typically would like to biopsy for staging (ie, not suspected primary site) while balancing the least invasive approaches.

How about cefepime, instead of Pip-Tazo?

"There is data that suggests all-cause mortality is better if pip-tazo is used in febrile neutropenia."

Clinically, Cefepime would also give you broad coverage and if an answer choice would be relevant.

A high-stakes exam is not likely to ask you to choose between cefepime or pip-tazo."

For treating high-risk febrile neutropenia, how do you choose between antipseudomonal therapy and carbapenems?

In clinical practice, this is likely driven by antimicrobial stewardship programs in the hospital. As an example, if a patient has a history of infection with an ESBL organism, you may need to opt for a carbapenem.

Do you often see MASCC score used to guide the initial approach to febrile neutropenia?

MASCC (Multinational Association for Supportive Care in Cancer) score use is recommended by both ASCO and IDSA to help with risk stratification in febrile neutropenia management-- those with a low score can be managed from home (< or equal to 21)

Is renal ultrasound with Doppler an equivalent screen for fibromuscular dysplasia in a secondary HTN w/u?

Computed Tomographic Angiography (CTA) is often the initial imaging modality of choice due to its high spatial resolution and ability to visualize small calcifications, which helps differentiate FMD from atherosclerotic disease. US with doppler is more user-dependent and can be less sensitive.

To treat hypertension only with ASCVD over 10%, are you considering hiv pts who are now included as a cv risk factor?

The slide was referencing the threshold blood pressure for patients with ASCVD risk above or less than 10% based on the AHA/ACC 2017 guidelines. This is particularly useful in patients who have BP that is close to goal but have a high ASCVD risk, it may help prompt treatment. ASCVD risk is not a perfect tool, and clinical judgement should be used.

If she is 48, why would you use a teratogenic med?

The question highlights that she has Stage 2 hypertension, so it is the best answer as you would need TWO different agents based on the AHA/ACC guidelines.

The ACC/AHA calculators do not take into account all of the risk factors for ASCVD.

Correct. When these AHA/ACC guidelines were released, some of the newer calculators such as PREVENT were not yet widely used.

Are there any benefits of ACE-I versus ARBs?

The data for the reduction of cardiovascular events in some populations is stronger for ACEi; however they are also associated with more adverse events.

In a hypertensive patient on ACE inhibitor or ARB, is there an elevated serum creatinine level after which we need to hold these drugs, or we continue?

In general, the cutoff of a 30% increase in Cr is used.

What about ACS with acute pulmonary edema?

In the setting of HTN emergency it is the afterload that is driving the ACS and the pulm edema so any of these agents would work-- NTG may give you venodilation as well so might a reason to start there but the primary goal is to get the afterload down.

For hypertensive urgency, should you send to ER first to rule out end-organ damage with labs?

This is symptom-based based often in the clinic. If they are otherwise asymptomatic and a new patient, one could try to order labs to get a sense of where their Cr is -- which would be important to know in the medical management.

When asking for the most appropriate next step, should we consider definitive management or the immediate next step in the office?

A high-stakes exam asking about the next step you should think about it as what you would do as the immediate next step.

Fibromuscular Dysplasia - if our 27F had well-controlled BP on a reasonable antihypertensive regimen, would we still pursue renal artery angioplasty?

ACEi/ARB is okay, but angioplasty would be curative.

GDMT? Goal-directed medical therapy?

Yes, GDMT is in reference to goal-directed medical therapy.

In FD is it usually a unilateral disease in the renal vessels or is bilateral common? Is CT needed to follow up if renal vessel velocities are the same on US?

FMD is typically multifocal and bilateral. CTA is the recommended imaging modality and is less operator-dependent compared to US doppler.

What workup is expected from urology? Cystoscopy?

Urologic evaluation may include cystoscopy and possibly imaging for evaluation of renal cell carcinoma

Why is repeating the sample wrong if microscopic hematuria is defined as at least 3 RBCs in two of three UAs?

In the question, the patient is >60, which is defined as high risk by the AUA recommendations, so he requires further evaluation instead of repeating the sample

Does it mean that if the patient has Hematuria and is on AC, we should do our work up and do not rely on AC as the cause?

Yes, you want to rule out other causes.

Why is fibromuscular dysplasia of the renal artery common in females? Is it hormonally mediated? Estrogen?

Fibromuscular dysplasia (FMD) is more common in women due to a combination of genetic, hormonal, and possibly environmental factors

Where does renal artery denervation fit into secondary hypertension?

Renal denervation is definitely an emerging field. It is not widely covered and not currently in the US guidelines as a standard of care.

Several of my patients have aki due to creatinine supplements. What is your approach and recommendations besides stopping the product?

They may not have true AKI. In patients using creatinine supplementation, or patients with muscle mass that may not be a constant state, consider calculating eGFR with other calculators, such as Cystatin-C

What is the role of cystatin C in worsening creatine, especially in the decision to switch HIV medication?

Cystatin C is a relatively steady-state substance in the body, so in circumstances where creatinine is fluctuating due to muscle mass or diet, cystatin C can be used to estimate eGFR

Mechanism of compartment syndrome in Rhabdo ?

Swelling of the tissue from the muscle inflammation-- when the compartment is wrapped in fascia-- which doesn't allow for the full expansion needed in that state-- pressure goes up to a point that it decreases arterial perfusion, leading to ischemia. Treatment is then open the compartment by opening the fascia-- but then can lead to a larger release of CK as the flow into and out of the tissue is restored."

What is the expected recovery time after AIN and stopping the inciting drug?

Great question. This is influenced a lot depending on the age of the patient, baseline renal function, and how quickly the offending agent was identified. One study I found showed that approximately 49% of patients with drug-induced AIN treated with steroids achieve complete recovery, 39% achieve partial recovery, and 12% do not recover within 6 months.

Are we specifically concerned with compartment syndrome abdominal in rhabdo?

"Compartment syndrome in the area of the rhabdo due to the fascia that are around the muscle. The AKI here is due to the CK.

Abdominal compartment syndrome can be seen in the setting of things like bowel ischemia--but this clinical syndrome is separate from compartment syndrome with rhabdo and the AKI here is due to an increase in pressure in the abdomen and compression on the urinary system directly."

Which is better for contrast injury prophylaxis, 0.9% or 0.45% NaCl, and when should we worry about chloride-induced kidney injury?

The American College of Radiology recommends isotonic fluid due to better efficacy as it has better plasma volume expansion

Bladder catheter= Foley cath?

Correct

What are the key/striking features that should make us think acyclovir induced AKI? and vanc induced AKI? (Thanks for reviewing aminoglycoside already)

"Vancomycin-induced AKI involves oxidative stress and apoptosis, acute tubular necrosis, acute tubulointerstitial nephritis, and tubular obstruction by vancomycin-associated casts.

Acyclovir is toxicity from the crystals that precipitate in the tubule and cause direct tubular toxicity and tubular obstruction."

In our case of AKI after PNA, the renal ultrasound shows retention but no hydronephrosis on either kidney - Can I still blame the AKI on a post-renal cause?

You want to think about all the options-- but if you have a full bladder in the setting of AKI then decompression makes sense while you are working through the differential of other causes.

Is Anti PLA2R specific or kidney bx for Mem Neph.

Good question. Technically speaking if the anti PLA2R is positive biopsy is not required, however, in clinical practice, the PLA2R may take a few days to come back, so biopsy may be pursued concurrently. The PLA2R is helpful for determining treatment, as there is great response to Rituximab if PLA2R is positive.

Why is the cholesterol high with nephrotic syndrome? Low proteins rev up liver production of everything in general to try to replete?

This is primarily due to increased hepatic synthesis of lipoproteins and decreased catabolism of lipoproteins.

What about SGLT2 plus something like Kerendia? Is there any benefit? She covered it. Thanks!

In patients with diabetes and CKD, Finerenone may be added if persistent albuminuria despite max RAS inhibition, assuming potassium is not high

On the test, will they use central and nephrogenic diabetes insipidus or will they use the newer terms?

It would be best to be familiar with both terms.

Can you please give us an example of a clinical case of true hypovolemic hyponatremia? Thank you!

This may be seen in someone with a lot of losses from sweating, GI losses, etc

There was an interruption. What was the answer to this query: Can we safely continue ACEi/ARB in hyperkalemic ESRD pt?

Yes, the hyperkalemia here would not be due to the ACEi/ARB if they are anuric. Although depending on severity may consider stopping.

Is it common to see osmotic demyelination syndrome from overcorrection of hyponatremia? Especially with auto rapid correction?

The incidence rate of ODS in the general population is approximately 0.611 per million person-years, it is exceedingly rare.

Could you please explain again metabolic acidosis, coexisting with another acid-base disorder?

"In metabolic acidosis with increased anion gap, the next step is to check that the degree of acidosis matches. This is done using the Δ Anion gap / Δ bicarbonate

<1 → coexisting normal anion gap metabolic acidosis

>2 → coexisting metabolic alkalosis

Subsequently, you check that the CO₂ is appropriately compensated using winter's formula, Respiratory compensation: PCO₂

$= (1.5) [HCO_3] + 8 \pm 2 PCO_2$

> expected → coexisting respiratory acidosis

PCO₂

< expected → coexisting respiratory alkalosis"

For patch testing of hand dermatitis, is that also done on the back?

Patch testing is typically performed on the upper back

For Q3, how do you confirm it's not contact dermatitis from something like new laundry detergent on socks?

Contact dermatitis may appear to be more well-demarcated. The history would also provide significant clues and should be considered in clinical practice.

If ulcer on bilateral dorsum of foot in the uncontrolled diabetic patient with severe PAD. Would it be arterial ?

A high-stakes exam is going to give you a class location and story-- clinically, this may be a case where asking about footwear could be important-- and it is neuropathic from ill-fitting shoes and rubbing the foot in the same way on both sides. Could also be complicated by PAD with impaired healing... but just not the classic presentation.

Which antibiotic/s is usually involved in AGEP?

Classically, beta-lactams, macrolides, and clindamycin.

How to treat acne vulgaris in pregnant women or those plan to get pregnant?

Topical treatment with benzoyl peroxide is preferred

What kind of emollient in your experience, do you recommend for dermatitis, any preference?

As I mentioned, I can't identify products without naming brands (and I do not get any compensation from personal care product companies :)). I like plain petrolatum ointment, CeraVe from a jar (not the pump), Aquaphor, Eucerin. In addition, I like some thick ones for the hands after each hand washing, like Neutrogena Norwegian Formula. In general, the thicker the emollient the better, i.e., ones from a jar rather than a squeeze bottle or pump

So does it mean that in a patient with a psoriasis history, the steroids taper course for asthma or copd exacerbation should be shorter in duration?

Yes, the general rule is the lowest effective dose and shortest duration possible

Approach for onychomycosis?

This depends on severity and co-morbidities. For severe disease, oral terbinafine is the preferred first-line treatment due to its higher efficacy and shorter treatment duration compared to topical therapies. Oral itraconazole is another option. Topical treatments are generally less effective but are suitable for mild to moderate onychomycosis or for patients who cannot tolerate oral antifungals

Do SKs show up occasionally very dark with a skirt on the back of a 19 yo female?

SKs are typically seen in older patients, though there can be a genetic predisposition for them to occur earlier

To clarify, if breslow depth is greater than 0.8cm then do SLN bx?

yes

Is chondrocalcinosis specific for pseudogout?

It is not specific for pseudogout. It can also occur with increasing age, osteoarthritis, previous joint trauma, and hyperparathyroidism.

Does transcutaneous electrical nerve stimulation = (TENS)?

Yes

Any role of platelet-rich plasma in the treatment of OA? Like knee OA?

The data for platelet-rich plasma is mixed and likely beyond the scope of the Board Exam

Regarding question 4: if it had been a diagnosis question, should we have even obtained a knee X-ray or should we have made the diagnosis clinically?

Good question. Typically the presence of an effusion is an indication for radiographs.

When would you start with Plaquenil instead of methotrexate?

Plaquenil is primarily used as combo therapy b/c it has only modest efficacy in preventing structural disease. It is in guidelines as a conditional recommendation over other DMARDS if the patient is DMARD naive and has low disease activity, b/c it is better tolerated with a more favorable risk profile.

Is positive CCP more common than positive rheumatoid factor in patients with rheumatoid arthritis?

RF is more common, though only slightly. Some patients may be negative for both.

What's the underlying physiology of the triad neutropenia, splenomegaly and RA seen in Felty Syndrome?

The pathophysiology of Felty syndrome is complex. It involves the presence of anti-neutrophil antibodies and immune complexes that bind to neutrophils, leading to their destruction. There is also a strong genetic component that we likely do not fully understand.

Seronegative RA has less extra-articular manifestation as compared to seropositive RA?

Yes

Can we diagnose GCA by Temporal artery US? It is specific?

This is an emerging utility of US. However, as of 2021, the American College of Rheumatology/Vasculitis Foundation Guideline for the management of GCA conditionally recommends temporal artery biopsy (TAB) over TAUS for establishing a diagnosis of GCA in the United States

How might features of jaw claudication in GCA differ and overlap with trigeminal neuralgia pain with chewing?

"Jaw claudication in GCA is a dull, aching pain that occurs after prolonged chewing and is due to ischemia of the masticatory muscles. It is often progressive and can lead to difficulty in completing meals.

Trigeminal neuralgia is characterized by sudden, severe, electric shock-like pain that is brief and can be triggered not only by chewing but light touch or something else very small"

How might features of the vision loss in GCA differ and overlap with amaurosis fugax?

"Vision loss of GCA is often permanent due to arteritic anterior ischemic optic neuropathy (AION) or central retinal artery occlusion (CRAO)-- is can be severe and progress rapidly to bilateral blindness if not treated quickly.

Amaurosis Fugax is often a transient vision loss- and typically brief bc it is embolic in nature-- described classically as a shade being pulled over the eye, unilateral and lacks the systemic symptoms of GCA."

How often do you see purpura in serum sickness? Follow up from the quiz question.

It is often seen but unfortunately I cannot find great data on the exact prevalence

Pre medications with corticosteroids and antihistamines can reduce the risk of serum sickness?

yes

What is HLH/MAS?

Hemophagocytic Lymphohistiocytosis or Macrophage Activation Syndrome