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directions in residency
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Financial planning in residency: Frequently asked questions — Part 2

By Emily Margosian, Senior Editor

David B. Mandell, JD, MBA, answers frequently asked questions about financial planning during medical residency.

Q *Directions in Residency*: Are there financial planners who are experts in dealing with physicians' specific type of debt and subsequent jump in tax brackets between medical school and attending life? Does a resident necessarily need to consult a financial advisor who has expertise working with physicians?

MANDELL: I think it's good to work with an advisor with physician expertise. I say that as someone from a firm that has been working with physicians for 20 years, but there are many other excellent firms out there. On my podcast, which is a free resource, I interview another financial planner who is not from our firm but specializes in helping early-career physicians navigate their student loans. While we do help clients a little bit with that, given all the different options to defer student loans, get them forgiven, or refinance, it may be best to seek out someone who is an expert in that. Some advisors may have expertise in graduate student loans in general, not just medical. That's probably helpful, but there are also folks out there who really know that world of medical school debt well and it doesn't take much research to go out and find them.

Q Where and how should residents be investing?

MANDELL: Let's start with 'where' — or what we call 'asset location.' As a resident or fellow, if your employer offers a 401k or a 403b and has a match — essentially, they're giving you free money — that's a clear place to take advantage. Anything that's tax-deferred, like a 401k or a 403b, is a good place to start. If you're a young physician with just a dollar

see **FINANCIAL** on p. 3

David B. Mandell, JD, MBA, is an attorney and author of more than a dozen books for physicians. He is a partner in the wealth management firm OJM Group. Check out his podcast, *Wealth Planning for the Modern Physician* at **www.ojmgroup.com/wealth-planning-for-the-modern-physician-podcast** or wherever you get your podcasts.

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FINANCIAL from p. 1

to invest, I'd rather have that dollar be in the 401k or the 403b at the hospital because you'll have tax-free growth until your retirement which may be 30 or 40 years down the road. Invest in tax-advantaged vehicles as much as you can.

As far as what to invest in, that's a complex question. The short answer for most people is a well-diversified, low-cost portfolio. Keep costs low; there's what's called exchange-traded funds (ETFs), which are more passive and tied to the S&P 500 and other asset classes. Have it well-allocated so you have diversification, which is a whole other topic. As you build up more assets, you can drill down more. However, for residents, my advice is to choose a location in a tax-advantaged vehicle and invest in a low-cost, diversified portfolio.

How much should residents be saving right now at this point in their career when there may not be a lot to save?

MANDELL: It's ok even if it's zero. Right now, you're just trying to make it work. My brother was a cardiology fellow in Manhattan. It's hard to save money in Manhattan regardless, and when you're not making that much, it's going to be even tighter. I wouldn't get overly stressed about that. There will be a time to save. Now, if you are saving because you're able to live below your means, then it gets back to what we were talking about before, which is trading off between student loan interest payments and putting together a portfolio that is low-cost and tax-advantaged.

Most residents and fellows are not going to be able to save much. However, any dollar that you can save as a young physician will give you the power of compound interest, which Einstein is alleged to have called the eighth wonder of the world. Time in the market beats timing the market. Meaning, if you have some thoughtful, broad ETFs, and you're investing in your 20s or early 30s, at age 60 you will be extremely happy you did. My takeaway is, if you're not able to save, don't stress about it because this is not a time that many can. If you are able, don't overcomplicate it; keep it simple, allocated, and low cost.

Missed part one? Check it out at **www.aad.org/member/publications/more/dir/archive**. DR

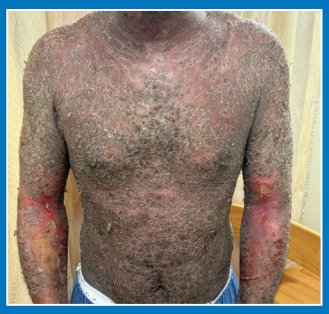
Marita Yaghi, MD, is a PGY-2 dermatology resident at Larkin Community Hospital, South Miami.

Stanley Skopit, DO, MSE, FAOCD, FAAD, is chair and program director of the Larkin Community Hospital, South Miami residency program.

Uros Rakita, MD, is a PGY-4 dermatology resident at Larkin Community Hospital, South Miami. (not pictured)

Race for the Case

By Marita Yaghi, MD, Uros Rakita, MD, and Stanley Skopit, DO, MSE, FAOCD, FAAD



A 54-year-old incarcerated male was referred to our clinic one month after discharge from an outside hospital for evaluation of a pruritic, painful, desquamating rash of one year's duration.

At the time of initial admission, he presented with erythroderma, fever, chills, and skin pain. He was treated with systemic corticosteroids and antibiotics without improvement, and his home medications (amlodipine and losartan) were discontinued. Laboratory studies demonstrated leukocytosis (15.7 × 10^3/μL) with eosinophilia (14.6%) and a mild elevation in creatinine that resolved with intravenous fluids. A skin biopsy was non-diagnostic, and direct immunofluorescence (DIF) was negative. He was discharged on an 80-mg daily prednisone taper.

At presentation to our clinic, physical examination revealed widespread eroded erythematous plaques with thick scale and crust involving greater than 90% of the body surface area. Eyelid crusting was noted, with no mucosal involvement. The patient endorsed skin soreness, impaired thermoregulation, and eye dryness.

Repeat biopsies from two sites using a 4-mm punch demonstrated intraepidermal acantholysis on hematoxylin and eosin staining and intercellular deposition of IgG and C3 on direct immunofluorescence.

1. **Based on the clinical, histopathologic, and laboratory findings, what is the most likely diagnosis?**
2. **What are the most likely target antigens involved in this condition?**
3. **What therapies can be initiated in addition to systemic corticosteroids in the acute setting?**
4. **What potential complications may occur if this condition remains untreated?**
5. **What is the most common culprit drug in drug-induced forms of this disease?**
6. **If his condition were determined to be a paraneoplastic phenomenon, what additional serum antibodies may be present?**

Respond with the correct answers at **www.aad.org/RaceForTheCase** for the opportunity to win an Amazon gift card!

Race for the Case winner (Winter 2025)

The winner of the winter 2025 Race for the Case is Marita Yaghi, MD, a PGY-2 at Larkin Community Hospital, South Miami. Dr. Yaghi correctly identified discoid lupus erythematosus (DLE) in our latest Race for the Case and provided the most accurate responses in the quickest time. Congrats to Dr. Yaghi!

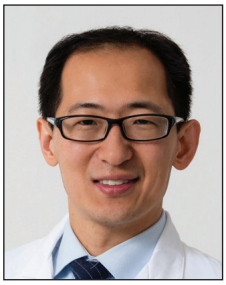
You can read more about this case online at **www.aad.org/race-case-answers**. If you can solve the case above, there may be a $100 Amazon gift card in your future, and you will be invited to contribute your very own Race for the Case. Visit **www.aad.org/RaceForTheCase**.

Keloids

By Albert E. Zhou, MD, PhD, Kelley Sharp, MD, and Hao Feng, MD, MHS, FAAD

Albert E. Zhou, MD, PhD, is a PGY-4 and dermatology chief resident at the University of Connecticut, and incoming Mohs fellow at the University of California, Los Angeles.

Kelley Sharp, MD, is a PGY-2 at the University of Connecticut.



Hao Feng, MD, MHS, FAAD, is associate professor, director of laser surgery and cosmetic dermatology, and fellowship director at the University of Connecticut.

I. DEFINITION & PATHOGENESIS

Feature	Description
Definition	Benign fibrous overgrowths extending beyond original wound margins
Etiology	Dysregulated wound healing → excess type III > type I collagen deposition
Genetics	Associated with HLA-B14, -B21, -BW16, -DR5; more prevalent in Fitzpatrick IV-VI
Cellular players	↑ Fibroblast proliferation, ↓ apoptosis, ↑ TGF-β1, VEGF, PDGF
Triggers	Skin trauma (surgical wounds, piercings, burns, acne), infections, PFB, AKN

Wound healing ➜ prolonged inflammation ➜ ↑ TGF-β1 ➜ excess fibroblast activity ➜ disorganized collagen deposition ➜ keloid

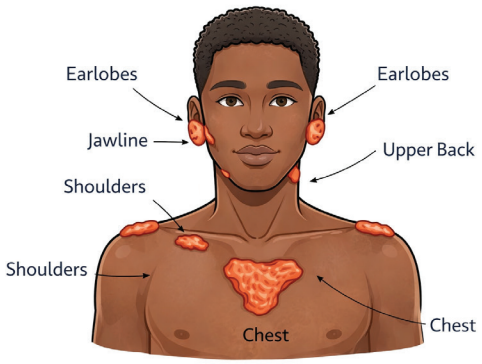
II. DIFFERENTIAL DIAGNOSIS

Condition	Distinguishing features
Hypertrophic scar	Confined to wound; may regress
Dermatofibroma	Firm, dimple sign positive, non-tender
DFSP	Slow-growing plaque; CD34+ on biopsy
Sarcoidosis	Non-caseating granulomas; systemic signs
Colloid milium	Waxy papules; sun-exposed areas
Scarring alopecia	Evaluate LPP, CCCA, AKN if scalp involvement
Pseudofolliculitis barbae (PFB)	Follicular-based papules/pustules in beard area; risk factor for keloid formation
Acne keloidalis nuchae (AKN)	Chronic inflammatory papules on nape of neck → keloid-like plaques; often coexists with true keloids

III. CLINICAL FEATURES

- Pruritic, firm, shiny pink/purple papules or plaques
- Location: Earlobes, shoulders, chest, jawline, upper back
- AKN may mimic or coexist with keloids on posterior neck
- May be painful or tender
- No spontaneous resolution

Most Commonly Affected Sites for Keloids



IV. HISTOLOGY

Feature	Keloid	Hypertrophic scar
Collagen arrangement	Broad, thick, disorganized bundles ("keloidal collagen")	Parallel, wavy bundles (Type III → Type I)
Collagen type	↑ Type III > I; disorganized	Initially ↑ Type III → matures to Type I
Borders	Extends beyond original wound margins	Confined within original wound margins
Vascularity	Decreased in mature keloid	Increased in early hypertrophic scars; more prevalent and vertically arranged
Inflammatory infiltrate	Sparse, chronic	Moderate, active inflammation in early stages
Appendages	Frequently absent	May be preserved or partially damaged
Fibroblasts	Large, plump, active	Spindle-shaped, less active over time
Re-epithelialization	Often normal	Typically normal

Keloids

By Albert E. Zhou, MD, PhD, Kelley Sharp, MD, and Hao Feng, MD, MHS, FAAD

V. MANAGEMENT & TREATMENT OPTIONS

Modality	Mechanism	Notes
ILK (triamcinolone)	↓ Fibroblast activity	10–40 mg/mL every 4–6 weeks
5-FU (fluorouracil)	Antimetabolite	Often combined with ILK
Silicone sheeting/gel	Occlusion, hydration	Best for prevention/post-excision
Cryotherapy	Vascular injury → necrosis	Small lesions; caution in dark skin
Laser (PDL, CO_2)	↓ Vascularity, remodels collagen	Use with ILK/5-FU
Radiation therapy	Fibroblast apoptosis	Infrequently used; post-op only
Excision	Physical removal	Recurrence rate high if monotherapy
Topical clindamycin/ benzoyl peroxide	For PFB/AKN prevention	Use alongside hair removal strategies
*** Combination therapies = most effective approach ***		

Emerging and experimental treatment options		
Agent	**Mechanism/target**	**Clinical notes**
Botulinum toxin A (BoNT-A)	↓ Tension & fibroblast TGF-β1 signaling	Improves scar pliability, ↓ recurrence when combined with ILK
Verapamil	Calcium channel blocker; ↓ collagen synthesis	Intralesional use; mixed results
Imiquimod 5% cream	Immunomodulation via TLR-7	Post-excision topical use; limited efficacy
Bleomycin	Antitumor antibiotic	Intralesional; ↓ collagen & fibroblast activity; good for resistant keloids
Tamoxifen	Anti-estrogenic; ↓ TGF-β	Experimental; topical/injectable forms
Mitomycin C	DNA cross-linker	Topical post-excision use; rare
ACE inhibitors (e.g., enalapril)	↓ Angiotensin II → ↓ fibrosis	Early-stage data; experimental
Insulin	↑ Keratinocyte migration, ↓ inflammation, modulates TGF-β1	Topical or intralesional shown to enhance wound healing and reduce scar formation

VI. TREATMENT ALGORITHM

- Mild (<2 cm) → ILK + silicone → add 5-FU if no response
- Moderate (2–4 cm) → ILK + 5-FU ± cryotherapy; consider laser
- Large/resistant (>4 cm) → excision + post-op radiation ± ILK maintenance

General principles:

- **Avoid excision alone** → Highest recurrence
- **First-line combo**: ILK + 5-FU
- **Best prevention**: Silicone gel + early ILK
- **For ear keloids**: Excision + compression earrings ± ILK
- **For post-shave PFB/AKN**: Treat inflammation (topicals steroids, antibiotics) + ILK early

VII. PREVENTION STRATEGIES

- Avoid elective procedures in at-risk areas (e.g., chest, shoulders, jawline)
- Use silicone sheeting post-surgery and consider ILK immediately post-op in predisposed/high-risk patients
- Manage PFB and AKN early to prevent progression to keloid; early treatment of acne, folliculitis
- Hair grooming education in PFB-prone individuals (avoid close shaving)

References:

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More study charts online!

There are more Boards Fodder charts online! In addition to the chart in this issue, you can view the **Mohs micrographic surgery** chart by Benjamin Cooper, DO, Taha Rasul, MD, and Arianne E. Chavez, MD, FAAD, and the **Guide to actinic keratosis treatments** chart by Nathaniel A. Marroquin, DO, Shannon Hart, DO, and Ryan Ottwell, DO, FAAD.

These and many more charts can be found at **www.aad.org/boardsfodder**.

John Trinidad, MD, MPH, FAAD, is associate professor of dermatology at Massachusetts General Hospital, Harvard Medical School.

Clinical Pearls

Clinical Pearls help prepare residents for the future by providing them with insights about what they should know about a specific subject area by the time they complete their residency.

Measles pearls for residents

By John Trinidad, MD, MPH, FAAD

Pearl #1: Measles remains rare but clinically relevant in the United States, with outbreaks driven by under-immunization. Dermatologists should maintain vigilance despite low current case counts. As of Jan. 29, 2026, 588 confirmed measles cases have been reported in the United States, all outbreak-associated and linked to outbreaks that began in 2025.(1) In contrast, 2025 saw 2,144 confirmed cases across 45 jurisdictions, underscoring the episodic but persistent risk of resurgence when vaccination coverage declines.(2)

Pearl #2: Dermatologists play a critical role in early recognition of measles based on its characteristic clinical progression. Measles classically presents with a prodrome of fever, cough, coryza, and conjunctivitis, followed by Koplik spots and a morbilliform eruption that begins at the hairline and spreads caudally with palmoplantar sparing.(3) Early recognition is essential, as patients are highly contagious before rash onset and during early eruption. Images and clinical considerations can be found at this AAD resource: **www.aad.org/member/clinical-quality/clinical-care/emerging-diseases/measles/clinical-information**.

Pearl #3: Suspected measles is a public health emergency requiring immediate isolation, testing, and reporting. Any patient with suspected measles should be masked, placed in airborne isolation if available, and reported immediately to local or state health departments. Dermatology practices should avoid sending suspected cases to waiting rooms or non-isolated clinical areas and should coordinate testing and evaluation with public health authorities.(3)

Pearl #4: Office preparedness is essential to prevent nosocomial transmission. Dermatology offices should have protocols in place for screening febrile rash illnesses, identifying immune versus non-immune staff, and rapidly isolating suspected cases. Ensuring staff immunity through documented MMR vaccination and having clear escalation pathways reduces practice-level risk during outbreaks. If your office does not have an airborne infection isolation room (AIIR), refer/transfer the patient to a facility where an AIIR is available. Notify the facility and accepting physician of any incoming measles patients. Learn how to prepare for a patient with suspected measles with this AAD resource: **www.aad.org/member/clinical-quality/clinical-care/emerging-diseases/measles/office-preparedness**.

Pearl #5: Vaccination remains the most effective preventive strategy, and dermatologists should reinforce evidence-based counseling. Measles requires approximately 95% population immunity to prevent sustained transmission.(3) A single dose of MMR vaccination is 93% effective against measles. A second dose increases effectiveness to 97%.(4) Dermatologists are well positioned to provide vaccine education, counter misinformation, and encourage appropriate immunization, particularly in communities with known gaps in coverage. DR

References:

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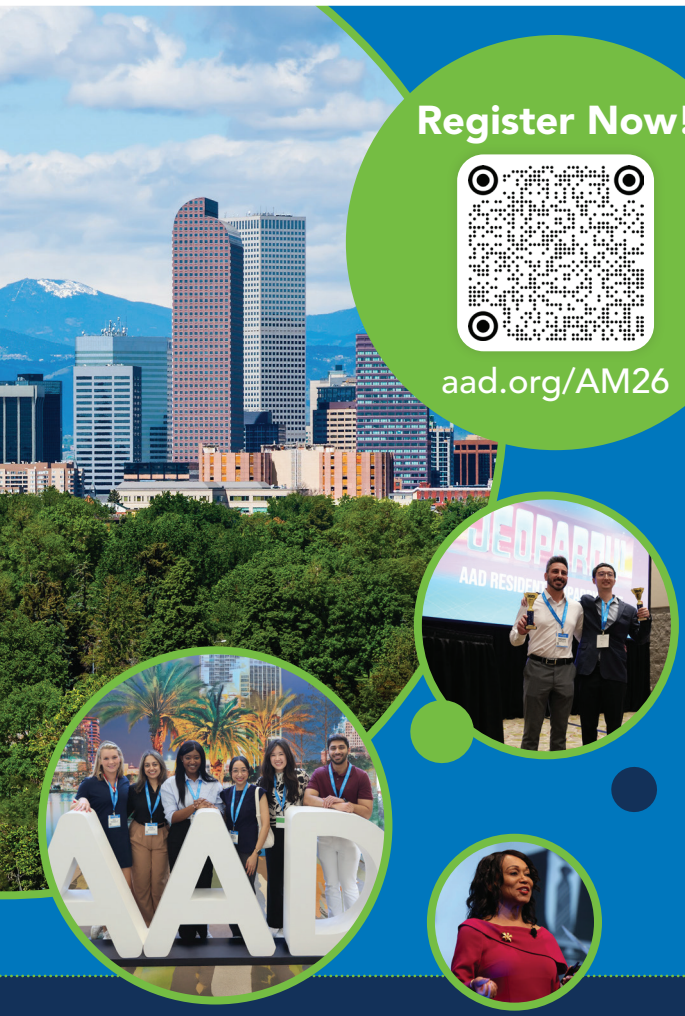

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Learn More!

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Inside this Issue

Chirag Vasavda, MD, PhD, is a PGY-3 at Harvard Combined Dermatology Residency.

Mentee as mentor

Residency is a period of transitions in nearly every sense. We are growing as adults and as physicians, taking on new responsibilities at home and at work, often before we feel fully prepared for either. I often look to my mentors on how to chart the best route forward, burdened by my own imposter syndrome and with so many questions of my own. It is easy to forget that I have accumulated experience worth sharing. Somewhere along the way though, we have graduated from being mentees to mentors too.

Mentorship can make or break a career. Most of us can point to a mentor who shaped how we see ourselves or just as easily to an experience where the wrong kind of mentorship made progress harder than it needed to be. One of the most important lessons my graduate mentor taught me was that it takes just as much effort to answer a minor question as it does to answer a big one. He pushed me to spend my time wisely on things that matter, and I hope to be as thoughtful a mentor to my own mentees as he is to me.

Here, I have distilled a few lessons that I have collected over the years from conversations with my own mentors, colleagues, and friends. Additional resources are listed below.

On being a good mentor: Always put the mentee first. Your perspectives are valuable, but your own aspirations and biases are your own; our job is not to shape our mentees into our own image, but to help them become the best version of themselves. Do not compare mentees, as they each have different strengths and weaknesses. Recognize mentees' wins by sharing the news widely, acknowledging their contributions in presentations, and writing letters of support. Lastly, be available; no matter how good your intentions, a mentor who is not accessible ends up being a useless one.

On being a good mentee: Build a diverse portfolio of mentors and avoid relying on a single individual. Determine if your mentor prefers verbal communication or written materials in advance. Approach mentors with a plan rather than a vague request for help. Explicitly agree on the structure of the relationship, including meeting frequency and key responsibilities. Finally, recognize that the nature of mentorships naturally change over time. We learn to mentor by being mentees, as there is always more to learn. DR

Resources:

1. "Making the Most of Mentors: A Guide for Mentees" – a readable guide that explicitly frames the mentee as the driver of the relationship: Zerzan JT, et al, *Acad Med*. 2009 Jan;84(1):140-4.
2. Nature's Guide for Mentors: Lee A, et al, *Nature*. 2007 Jun 14;447(7146):791-7.
3. Stanford "Quick Guides for Mentees" - tips on how to prepare for meetings, understand your mentor's context, and map the local power structure; good for learners at any level.
4. Duke University "Mentor/Mentee Tools and Resources": curated hub of mentoring tools and references.

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