



Folliculotropic Mycosis Fungoides Emerging After IL-17 Inhibitor Therapy

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Disclosures

I do not have any relevant financial relationships to disclose.

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Clinical Summary

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- 54-year-old man
- 10-year history of “psoriasis-like” plaques (no biopsy)
- 1 year ago: **secukinumab** started (partial relief)
- 6 months ago: scalp/facial nodules + lymphadenopathy
- Diagnosed as **FMF**, treated with Brentuximab (anti-CD30)
+Gemcitabine → progression → death in 6 months

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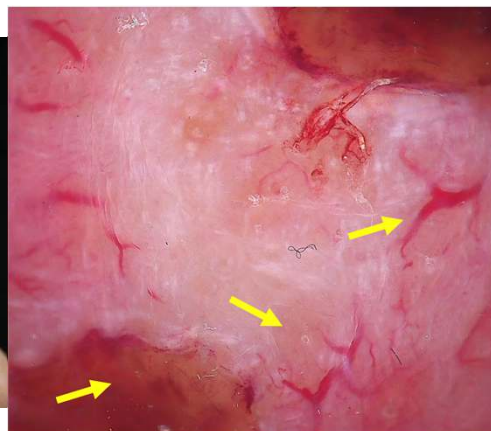
Clinical Images - Trunk



- Trunk: erythematous, scaly plaques
- Dermoscopy: spermatozoa-like vessels, glomerular vessels, orange-white structureless areas, rosette sign

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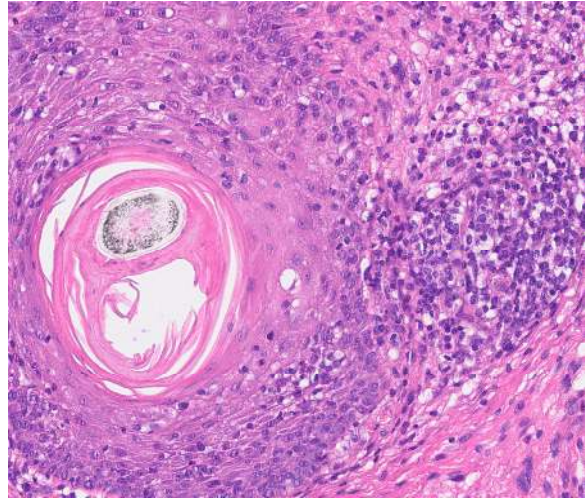
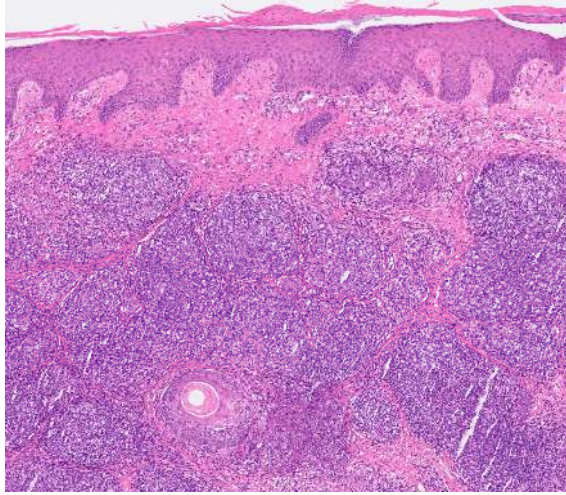
Clinical Images - Scalp



Scalp/face: nodules, tumors
Dermoscopy: orange-white structureless areas, branched vessels

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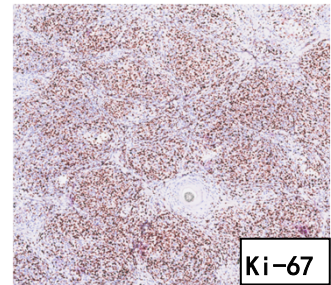
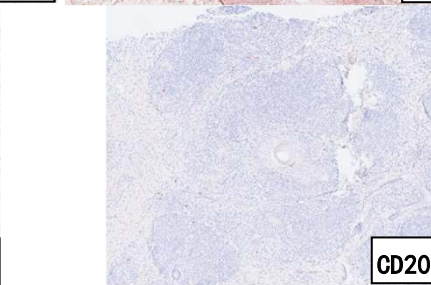
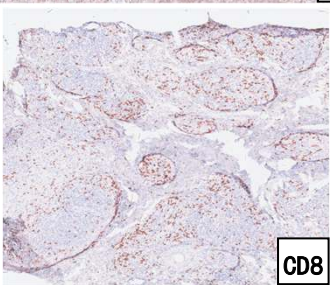
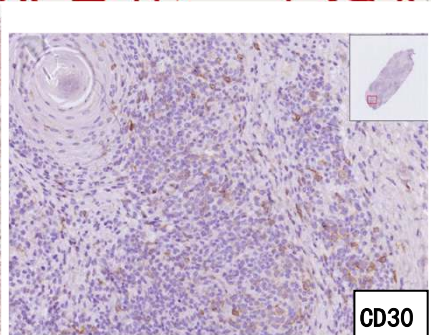
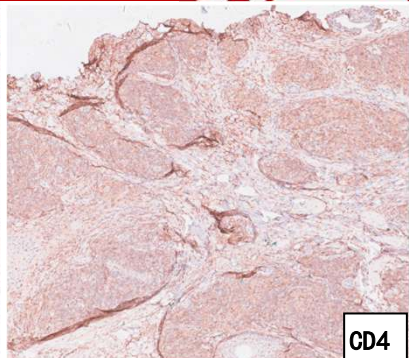
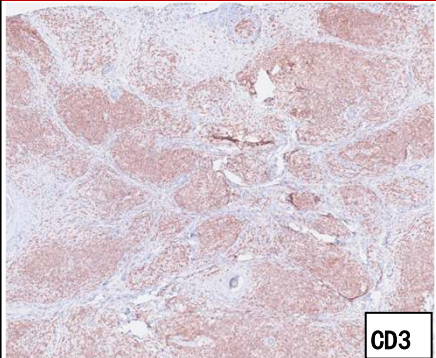
● Histopathology: Scalp Nodule



- Nodular dermal/subcutaneous lymphohistiocytic infiltrates
- Folliculotropism, without apparent epidermotropism
- IHC: CD3+, CD4+, CD30+, Ki-67 80%; T-cell receptor gene rearrangement positive

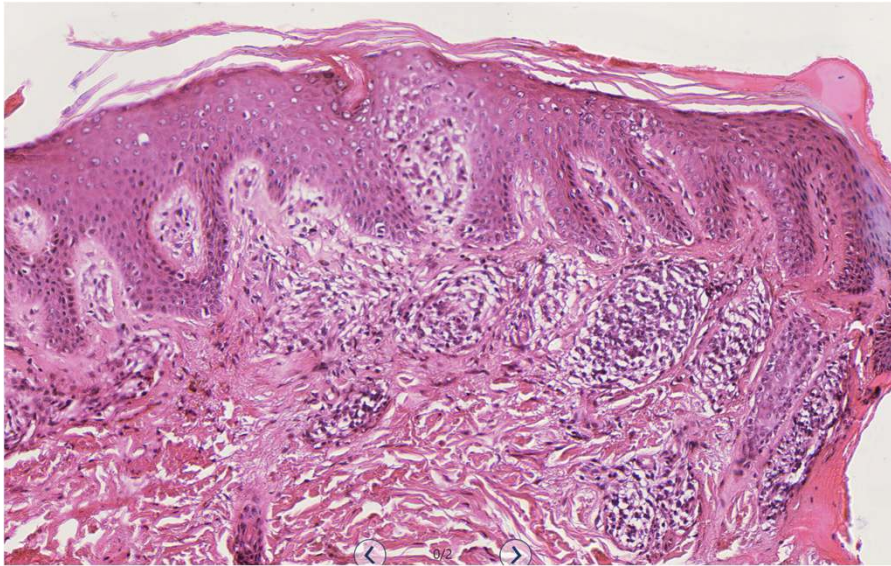
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● Histopathology: Scalp Nodule, IHC



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● Histopathology: Trunk Plaque



- Epidermal hyperkeratosis, acanthosis; Sparse epidermotropism
- **IHC:** CD3+, CD4+, **CD30–**, Ki-67 30%

Heterogeneity
between trunk and
scalp/face lesions

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● Staging Workup

- **PET/CT:** multiple hypermetabolic lymph nodes, splenomegaly
- **Labs:** ↑LDH (582 U/L), ↑β2-microglobulin (17.3 mg/L)
- **Peripheral blood / bone marrow:** no involvement

Stage IIB folliculotropic
mycosis fungoides with large
cell transformation



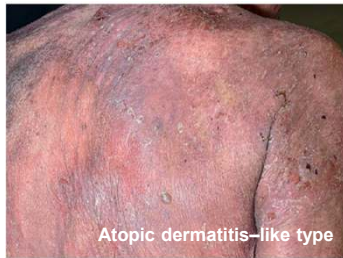
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● Discussion

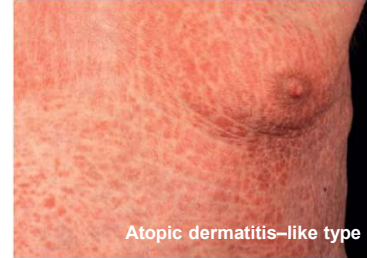
- Mycosis fungoides = “**great mimicker**” (psoriasis, eczema, etc.)



Chronic eczematous type



Atopic dermatitis-like type



Atopic dermatitis-like type



Psoriasiform type



Psoriasiform type



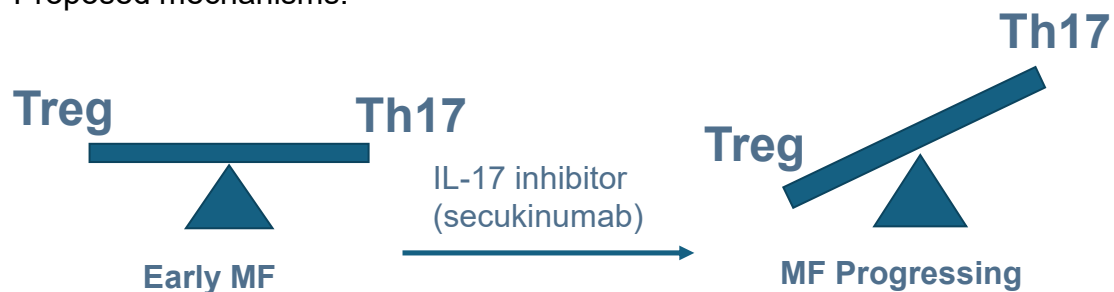
Psoriasiform type

Lorenzo Cerroni, Skin Lymphoma: The Illustrated Guide, 5th Edition

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● Discussion

- Folliculotropic variant: scalp predilection, poor prognosis
- Large cell transformation: CD30+, high Ki-67
- Rare cases reported after IL-17 inhibitor (secukinumab)
- Proposed mechanisms:



(1) Yoo J, et al. *Clin Exp Dermatol.* 2019;44(4):414-417.

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● Agents and CTCL

(1) A systematic review published in March 2023 analyzed 28 case reports and series including **62 patients** who developed or were newly diagnosed **with CTCL after biologic therapy**. Among these, **dupilumab** was the most frequently reported agent (42%).

(2) Espinosa et al. reported **7 patients** who were initially misdiagnosed with **atopic dermatitis (AD)** or had established **CTCL** treated with dupilumab. All experienced disease progression, including 3 patients who evolved to Sézary syndrome, with 2 deaths.

1.Schaefer L et al. *Am J Clin Dermatol.* 2023;24(2):153-164.

2.Espinosa ML et al. *J Am Acad Dermatol.* 2020;83(1):197-199.

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● Reports of MF Progression after Secukinumab



Case 1	Case 2	Case 3
42 F	79 F	71 M
Misdiagnosed as psoriasis	Misdiagnosed as psoriasis	Misdiagnosed as psoriasis
Secukinumab + ixekizumab + etanercept	Secukinumab For 12 weeks	Secukinumab for 8 weeks
Diagnosed as MF (stage IB → IIB)	PASI 8 → 16, later diagnosed as MF	Later diagnosed as erythrodermic MF



1.Amitay-Laish I et al. *Acta Derm Venereol.* 2020;100(16):adv00277.

2.Yoo J et al. *Clin Exp Dermatol.* 2019;44(4):414-417.

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● Take-home Messages

- Always biopsy atypical, Rx-refractory, or progressive lesions before using biologics
- Folliculotropic MF has distinct pathologic and clinical features
- Pathologists are central in early recognition and guiding management

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● Acknowledgments

- **Dr. Zhiyu Pang**, my undergraduate classmate, now a PhD student in dermatology, and her mentor **Dr. Jie Liu** at **Peking Union Medical College Hospital**, for their help in collecting the case information.
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