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REFERENCES

1. Brooklyn TN, Dunnill MGS, Shetty A, et al. Infliximab for the treatment of pyoderma gangrenosum: a randomised, double blind, placebo controlled trial. *Gut*. 2006;55(4):505-509.
2. Feagan BG, Sandborn WJ, Gasink C, et al. Ustekinumab as induction and maintenance therapy for Crohn's disease. *N Engl J Med*. 2016;375(20):1946-1960.
3. Marzano AV, Borghi A, Wallach D, Cugno M. A comprehensive review of neutrophilic diseases. *Clin Rev Allergy Immunol*. 2018;54:114-130.
4. Barbosa NS, Tolkachjov SN, El-Azhary RA, et al. Clinical features, causes, treatments, and outcomes of peristomal pyoderma gangrenosum (PPG) in 44 patients: the Mayo Clinic experience, 1996 through 2013. *J Am Acad Dermatol*. 2016;75(5):931-939.
5. Low ZM, Mar A. Treatment of severe recalcitrant pyoderma gangrenosum with ustekinumab. *Australas J Dermatol*. 2018;59:131-134.
6. Guenova E, Teske A, Fehrenbacher B, et al. Interleukin 23 expression in pyoderma gangrenosum and targeted therapy with ustekinumab. *Arch Dermatol*. 2011;147(10):1203-1205.
7. Fahmy M, Ramamoorthy S, Hata T, Sandborn WJ. Ustekinumab for peristomal pyoderma gangrenosum. *Am J Gastroenterol*. 2012;107(5):794-795.
8. Goldminz AM, Botto NC, Gottlieb AB. Severely recalcitrant pyoderma gangrenosum successfully treated with ustekinumab. *J Am Acad Dermatol*. 2012;67(5):e237-238.
9. Greb JE, Gottlieb AB, Goldminz AM. High-dose ustekinumab for the treatment of severe, recalcitrant pyoderma gangrenosum. *Dermatol Ther*. 2016;29(6):482-483.
10. Cosgarea I, Lovric Z, Körber A, Dissemond J. Successful treatment of refractory pyoderma gangrenosum with ustekinumab only after excision of renal cell carcinoma. *Int Wound J*. 2016;13(5):1041-1042.
11. Marzano AV, Tavecchio S, Berti E, Gelmetti C, Cugno M. Paradoxical autoinflammatory skin reaction to tumor necrosis factor alpha blockers manifesting as amicrobial pustulosis of the folds in patients with inflammatory bowel diseases. *Medicine (Baltimore)*. 2015;94(45):e1818.
12. Acquitter M, Plantin P, Kupfer I, Auvinet H, Marhadour T. Anakinra improves pyoderma gangrenosum in psoriatic arthritis: a case report. *Ann Intern Med*. 2015;163(1):70-71.
13. Zirwas M, Dobkin HE, Krishnamurthy S. Amicrobial pustulosis of the folds and palmoplantar pustulosis simultaneously induced by different tumor necrosis factor-α inhibitors: demonstration of a shared pathophysiology. *JAAD Case Rep*. 2017;3(5):401-403.

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Femoral artery ultrasound for improving the detection of atherosclerosis in psoriasis

To the Editor: The life expectancy of patients with psoriasis is reduced because of cardiovascular disease (CVD), and there is an increased risk of myocardial infarction at a younger age, higher than could be attributed to traditional CVD risk factors.[1] Hence, classic screening methods such as the Framingham Risk Score do not reliably evaluate the risk of coronary artery disease in these patients.[2] Rigorous screening for atherosclerosis has therefore been proposed for these patients,[2] and there is a need for a noninvasive, simple, and widely available technique for this purpose. High-resolution ultrasonic arterial scanning provides information on arterial atherosclerotic plaques and meets the aforementioned criteria.[3] Carotid intima-media thickness was initially used as a biomarker of

Table I. Anthropometric and clinical data in patients with psoriasis and control subjects

Characteristic	Patients with psoriasis (n = 51)	Controls (n = 51)	*P* value
Sex, M/F	36/15	36/15	1.000
Age, y	44.73 ± 11.66	43.88 ± 11.19	<.710
Body mass index, kg/m^2	30.10 ± 5.66	28.42 ± 4.73	<.106
Waist circumference, cm	102.37 ± 13.35	97.43 ± 14.17	<.073
Systolic blood pressure, mm Hg	131.45 ± 14.45	129.78 ± 12.14	<.530
Diastolic blood pressure, mm Hg	82.86 ± 8.38	79.94 ± 7.62	<.068
Sedentarism, %	13.72	17.64	<.786
Smoking, %	35.29	21.56	<.128
Hypertension, %	25.49	15.68	<.327
Dyslipidemia, %	25.49	21.56	<.476
Mean time with psoriasis, y	16.65 ± 10.43	—	—
PASI	12.25 ± 4.30	—	—
BSA, %	14.55 ± 8.67	—	—
Glucose level, mg/dL	99.15 ± 16.67	94.67 ± 9.16	<.122
Cholesterol level, mg/dL	188.78 ± 32.74	194.69 ± 39.25	<.453
LDL level, mg/dL	109.88 ± 28.42	117.77 ± 35.64	<.264
HDL level, mg/dL	50.32 ± 12.95	56.14 ± 14.94	<.059
Triglyceride level, mg/dL	146.51± 93.62	102.09 ± 52.99	<.008
25-OHD, ng/mL	18.89 ± 7.24	19.94 ± 8.12	<.536
CRP, mg/L	4.36 ± 4.98	2.25 ± 2.72	<.019
ESR, mm/h	7.43 ± 7.20	5.71 ± 5.16	<.207

Unless otherwise specified, data are expressed as means plus or minus standard deviation.
BSA, Body surface area; *CRP*, C-reactive protein; *ESR*, erythrocyte sedimentation rate; *F*, female; *HDL*, high-density lipid; *LDL*, low-density lipid; *M*, male; *25-OHD*, 25-hydroxyvitamin D; *PASI*, Psoriasis Area and Severity Index.

atherosclerosis; however, it is now known to be a very weak predictor of cardiovascular risk and is no longer recommended in the American College of Cardiology/American Heart Association guidelines.[3] A few studies used ultrasound to assess the presence of carotid plaques and reported contradictory results on their prevalence in patients with psoriasis.[4] Autopsy studies have observed that the presence of femoral plaque but not carotid plaque is a significant predictor of coronary atherosclerosis and coronary mortality.[5] Our study hypothesis was that ultrasound assessment of atherosclerotic plaques in the femoral artery would be more useful than in the carotid artery in screening for atherosclerosis in patients with psoriasis. We would therefore like to report part of the results of our study of 102 white participants: 51 patients with moderate-to-severe chronic plaque psoriasis (psoriasis area and severity index greater than 10 and body surface area greater than 10%) and 51 healthy control subjects matched 1:1 for age, sex, and body mass index. Patients and controls signed their informed consent before study enrollment. The study protocol was approved by the ethical committee of our institution.

Subjects underwent B-mode and Doppler ultrasound examination with a MyLab 25 Gold ultrasound system (Esaote, Florence, Italy). Ultrasound images were acquired with a linear high-frequency 2-dimensional probe (Esaote LA435). All participants underwent the same vascular ultrasound examination as previously described,[3] examining femoral and carotid arteries bilaterally. Atherosclerotic plaque was defined as a focal

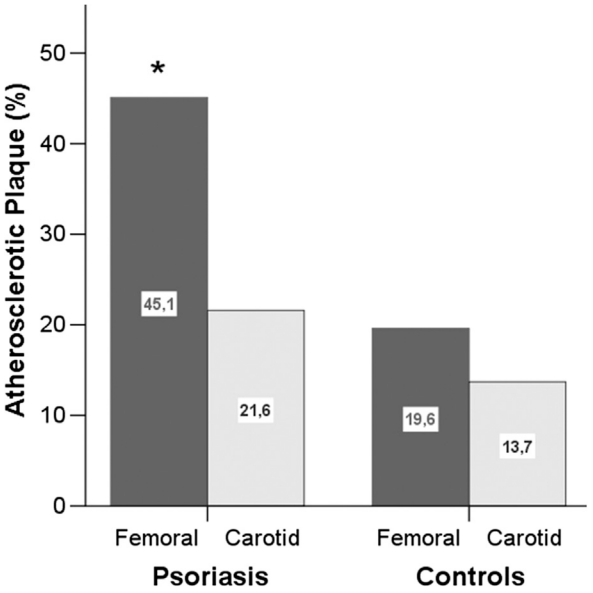

Fig 1. Percentage of atherosclerotic plaque in femoral and carotid arteries in patients with psoriasis and controls. *$P < .05$.

structure encroaching at least 0.5 mm into the arterial lumen or having a thickness greater than 50% of the surrounding intima-media thickness.

Table I exhibits the anthropometric, clinical, and biochemical data of participants.

To our best knowledge, this is the first study to evaluate the presence of femoral atherosclerotic plaques as a screening test for atherosclerosis in patients with psoriasis. The prevalence of femoral plaques but not carotid plaques was significantly higher in the patients with psoriasis than in age-, sex-, and body mass index–matched controls ($P < .006$), whereas the prevalence of femoral plaques among the patients was 2-fold higher than that of carotid plaques ($P < .008$) (Fig 1). According to the present findings, ultrasound study of femoral arteries is more useful than the study of carotid arteries to identify atherosclerosis in patients with psoriasis.

Dr Alvaro Gonzalez-Cantero is a PhD candidate at University of Castilla La Mancha. The authors thank those responsible for the PhD Program in Health Sciences at the University of Castilla La Mancha

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REFERENCES

1. Mehta NN, Azfar RS, Shin DB, Neimann AL, Troxel AB, Gelfand JM. Patients with severe psoriasis are at increased risk of cardiovascular mortality: cohort study using the general practice research database. *Eur Heart J*. 2010;31(8):1000-1006.
2. Lerman JB, Joshi AA, Chaturvedi A, et al. Coronary plaque characterization in psoriasis reveals high-risk features that improve after treatment in a prospective observational study. *Circulation*. 2017;136(3):263-276.
3. Laclaustra M, Casasnovas JA, Fernández-Ortiz A, et al. Femoral and carotid subclinical atherosclerosis association with risk factors and coronary calcium: the AWHS study. *J Am Coll Cardiol*. 2016;67(11):1263-1274.
4. Arias-Santiago S, Orgaz-Molina J, Castellote-Caballero L, et al. Atheroma plaque, metabolic syndrome and inflammation in patients with psoriasis. *Eur J Dermatol*. 2012;22(3):337-344.
5. Dalager S, Falk E, Kristensen IB, Paaske WP. Plaque in superficial femoral arteries indicates generalized atherosclerosis and vulnerability to coronary death: an autopsy study. *J Vasc Surg*. 2008;47(2):296-302.

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Early-stage melanoma and hematopoietic stem cell transplantation outcomes

To the Editor: Hematopoietic stem cell transplantation (HSCT) is an important treatment option for both malignant and autoimmune conditions, but it requires exposure to profoundly immunosuppressive agents and/or radiation. Unlike in patients who receive a solid organ transplant, restoration of a functional immune system and eventual discontinuation of immunosuppressive medications is the ultimate goal in HSCT. How such treatments may affect the risk of a second or recurrent melanoma in patients who had a melanoma before receiving their transplant is not clear.

We searched Duke University Medical Center's historical database of 4528 adult HSCT patients from 1996 to 2017 for patients who had a melanoma before receiving their transplant. One patient whose indication for HSCT was metastatic melanoma was excluded from this study. We included 23 patients (0.5%) with a total of 27 pre-HSCT melanomas (Table I). A total of 7 patients (30%) underwent allogeneic HSCT, whereas the rest received an autologous transplant. A total of 13 patients (56.5%) were exposed to ionizing radiation (Table II).

Only 1 patient (4.3%) developed a new melanoma (in situ) 3.1 years after allogeneic HSCT and 32.0 years after the initial melanoma. This patient did not have graft-versus-host disease (GVHD), had only 1 primary melanoma before HSCT, received a nonablative preparative regimen, and was successfully treated with local excision.

There were no recurrences of melanoma. At the median follow-up time of 2.0 years (range 0.02-13.1 years) after HSCT, 5 patients (26.1%) had died (none owing to melanoma). GVHD occurred following HSCT in 42.9% of patients who underwent allogeneic HSCT. This rate was not significantly