to HIV, HBV, and HCV in the dermatology office. *J Am Acad Dermatol*. 2017;77(5):946-951.
4. Charles PG, Angus PW, Sasadeusz JJ, Grayson ML. Management of healthcare workers after occupational exposure to hepatitis C virus. *Med J Aust*. 2003;179(3):153-157.
5. Nwaiwu CA, Egro FM, Smith S, Harper JD, Spiess AM. Seroconversion rate among health care workers exposed to HIV-contaminated body fluids: the University of Pittsburgh 13-year experience. *Am J Infect Control*. 2017;45(8):896-900.

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A randomized double-blind placebo-controlled trial of autologous platelet-rich plasma intradermal injections for the treatment of vulvar lichen sclerosus

To the Editor: We performed a randomized, double-blind, placebo-controlled trial to evaluate the efficacy and safety of autologous platelet-rich plasma (PRP) for the treatment of vulvar lichen sclerosus (LS). A total of 30 patients (mean age, 52.6 years; 29 whites and 1 Hispanic) with biopsy-proven active LS were recruited. One participant withdrew after randomization but before treatment, and 29 completed the study. Patients were randomized to receive either placebo (saline injections) (10 subjects) or 2 separate treatments of PRP separated by 6 weeks (20 subjects). There was no statistically significant difference in participant age or duration of symptoms between the PRP and placebo groups. Each treatment consisted of 5 mL of PRP injected subdermally and intradermally, infiltrating the areas affected by LS. The PRP was prepared by using a US Food and Drug Administration–cleared centrifuge that uses a laser to isolate the platelet-rich fraction of 60 mL of whole blood (Magellan Autologous Platelet Separator System, Isto Biologics, Hopkinton, MA). The PRP was collected in a blackened syringe so that neither the physician administering the PRP nor the study participants knew whether they were receiving the PRP or placebo. The primary efficacy variable was determined by a pathologist with expertise in vulvar pathology (D.H.), who was blinded to the treatment arms and evaluated the inflammatory infiltration on the pretreatment and post-treatment biopsy specimens (on a 0-3 scale). A secondary end point was change in score according to the Clinical Scoring System for Vulvar Lichen Sclerosus (CSS), which is a validated instrument that assesses both the investigator and patient impression of the severity of the LS.[1] Of the 19 women receiving PRP, 5 had improvement in histopathologic inflammation between the pretest and post-test treatment biopsies, 10 had no change, and 4 had more inflammation. Of the 10 women receiving placebo, 5 had improvement, 4 had no change, and 1 had more inflammation (Mann-Whitney U test result, 109.0 [$P = .542$]). The mean difference in the CSS patient domain between the initial and final visits was −7.74 for patients receiving PRP and −9.44 for patients receiving placebo (Mann-Whitney U test result, 80.50 [$P = .654$]. Bruising was the only adverse event reported.

A recent pilot study performed by our group showed that PRP reduced histopathologic inflammation in 7 of 12 patients with vulvar LS.[2] However, the main limitations of that study was its lack of placebo control. In addition, Tedesco et al studied PRP injection in 31 patients with LS.[3] They reported that 62% of patients had improvement in their LS, but their study was not placebo controlled, did not use validated measures of subjective or objective improvement, and did not include histopathologic evaluation. As LS is a premalignant condition and a reduction in inflammation with optimal use of corticosteroids lowers the rate of malignant transformation, it is essential that all studies of LS use reduction in inflammation as the primary efficacy measure.[4] One of the strengths of our current study is that it used this objective criterion as its primary end point. Additional strengths of our study are that it was blinded, was placebo-controlled, and used the validated CSS. A sample size calculation was performed before initiation of the study and determined that our study sample was powered to show a clinically significant effect of a 50% reduction in inflammation with a 2-sided significance of .05 and a power of 0.8. In conclusion, until further well-designed controlled studies with appropriate end points show positive results, the negative results of this study suggest that autologous PRP does not adequately treat vulvar LS.

Andrew T. Goldstein, MD,[a,b] Leia Mitchell, MS,[a] Vaishnavi Govind, MS,[a] and Debra Heller, MD[c,d]

From the Center for Vulvovaginal Disorders, Washington, DC[a]; Department of Obstetrics and Gynecology, George Washington University School of Medicine and Health Sciences[b]; and Department of Pathology and Laboratory Medicine[c] and Department of Obstetrics, Gynecology, and Women's Health, Rutgers New Jersey Medical School, Newark, New Jersey[d]

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Correspondence to: Andrew T. Goldstein, MD, The Center for Vulvovaginal Disorders, 3 Washington Circle NW, Suite 205, Washington, DC 20037

E-mail: Obstetrics@yahoo.com

REFERENCES

1. Gunthert AR, Duclos K, Jahns BG, et al. Clinical scoring system for vulvar lichen sclerosus. *J Sex Med*. 2012;9(9):2342-2350.
2. Goldstein AT, King M, Runels C, Gloth M, Pfau R. Intradermal injection of autologous platelet-rich plasma for the treatment of vulvar lichen sclerosus. *J Am Acad Dermatol*. 2017;76(1):158-160.
3. Tedesco M, Pranteda G, Chichierchia G, et al. The use of PRP (platelet-rich plasma) in patients affected by genital lichen sclerosus: clinical analysis and results. *J Eur Acad Dermatol Venereol*. 2019;33:e58-e59.
4. Lee A, Bradford J, Fischer G. Long-term management of adult vulvar lichen sclerosus: a prospective cohort study of 507 women. *JAMA Dermatol*. 2015;151(10):1061-1067.

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Molecular epidemiology of locally acquired Hansen's disease in Central Florida

To the Editor: From 2010 to 2017, 90 cases of Hansen's disease (HD) were reported in Florida, with some cases believed to have been acquired locally from 9-banded armadillos (*Dasypus novemcinctus*), which are the only known zoonotic reservoir of *Mycobacterium leprae* in North America.[1,2] However, their role in transmitting HD in Central Florida has been questioned owing to a lack of definitive evidence.[3] Previous literature classified global strains of *M leprae* by using single-nucleotide polymorphism (SNP) types and variable number of tandem repeats (VNTR), resulting in the identification of 2 strains of *M. leprae* able to infect humans (zoonotic strains).[4]

Our study protocol was approved by the University of Central Florida institutional review board and involved interviews and genotyping of *M. leprae* isolates from patients with newly diagnosed HD to assess for risk factors and potential exposures. All patients with biopsy-proven HD in a Central Florida dermatology clinic were approached regarding the study, and 5 out of 5 subjects were recruited. The subjects were interviewed using a list of questions regarding their history of exposure to potential risk factors. The HD-causing bacteria in the diagnostic skin biopsy specimens were genotyped by the National Hansen's Disease Program to assess whether the identified strains were zoonotic. The molecular genotyping data were then compared against the reported history of disease exposure to examine for correlation.

All 5 subjects reported no known contact with infected humans and minimal exposure to international travelers (Table I). They routinely participated in outdoor activities and observed armadillos on a regular basis, but only subject 1 recalled direct physical contact.

Of the 5 subjects, 4 were infected with *M leprae* SNP 3I-2, which is a known zoonotic SNP type. Additionally, the strain isolated from subject 4 matched both the SNP and VNTR found in armadillos. Subject 1 was infected with SNP 3K, which is a strain not found in previously tested armadillos.[5]

Through a combination of interview and molecular genotyping, our findings provided preliminary evidence that at least 4 of the 5 subjects had acquired HD from armadillos. SNP3I-2 has been identified in all sampled armadillos with HD and in up to 70% of human cases from the southern United States.[2,4] Because of the rarity of HD, it can be concluded that patients infected with the same *M. leprae* SNP type found in local armadillos likely acquired HD through animal exposure. Although subject 5 reported travel to areas with endemic HD, her *M. leprae* strain is found primarily in North America, suggesting that she likely acquired HD locally.