

Hematuria evaluation

(MARCH 2008)

TO THE EDITOR: In regard to the article on hematuria by Drs. Rao and Jones in the March issue of the *Cleveland Clinic Journal of Medicine*, I work in the emergency department and see a lot of cases of hematuria associated with indwelling Foley catheters and in patients on anticoagulants. I realize that gross hematuria usually needs a workup; however, in my clinical experience, the presence of a Foley and/or the use of anticoagulants often confuses the clinical scenario. I prefer to wait to refer the patient until either the Foley is removed or the anticoagulation is in the therapeutic range. I'd be eager to hear of your advice or experience with such patients.

DON FERNANDEZ, MD
Co-Director, Emergency Department
Carl T. Hayden VA Hospital
Phoenix, AZ

IN REPLY: Although a Foley catheter can contribute to macroscopic (gross) hematuria, to assume that it is the cause of the hematuria carries some undefined, but presumably small, risk of missing a more serious diagnosis. A more serious potential for misdiagnosis is in the setting of anticoagulation. As we discuss in our review, hematuria is analogous to hematochezia in the setting of anticoagulants. The possibility that anticoagulation has simply exposed an underlying bleeding source such as a tumor in either setting must not be underestimated, and we recommend that all patients with macroscopic hematuria undergo urologic evaluation unless there is a documented benign physical cause of bleeding such as traumatic catheterization.

J. STEPHEN JONES, MD
Chairman, Department
of Regional Urology
Glickman Urological
and Kidney Institute
Cleveland Clinic

PRAVIN KUMAR RAO, MD
Glickman Urological
and Kidney Institute
Cleveland Clinic

Methicillin-resistant *Staphylococcus aureus*: A link to statin therapy?

(MARCH 2008)

TO THE EDITOR: We read the interesting paper by Dr. Susan Rehm on the recently changing epidemiology and increasing incidence of methicillin-resistant *Staphylococcus aureus* (MRSA) bacteremia.¹ We feel these trends may be related in part to the increasing use of statin therapy in both outpatient and hospital settings.

In a case-control study,² skin and soft tissue infections as the source of bacteremia were significantly more prevalent among patients treated with statins compared with patients not receiving statin therapy. Additionally, there have been isolated reports of recurrent community-acquired MRSA skin infections in subjects on therapy with high-dose statins and no obvious risk factors for skin infection.³

The epidermis is a very active site of cholesterol synthesis, and after barrier disruption in the murine model, there is a brisk increase in epidermal cholesterol synthesis.⁴ Moreover, if the increase in epidermal cholesterol synthesis is inhibited by the topical application of statins, barrier function is impaired.⁴

Therefore, it is plausible that systemic statin therapy alters epidermal cholesterol homeostasis, resulting in barrier inadequacy and impaired innate immune function of the skin,⁵ leaving it more vulnerable to external pathogens and resulting bacteremia. Importantly, this needs to be investigated, particularly in light of the increasing and widespread use of high-dose statin therapy.

MARK R. GOLDSTEIN, MD
Medical Director,
Fountain Medical Court
Bonita Springs, FL

LUCA MASCITELLI, MD
Comando Brigata Alpina "Julia"
Servizio Sanitario
Udine, Italy

FRANCESCA PEZZETTA, MD
Cardiology Service
Ospedale di Tolmezzo
Tolmezzo, Italy



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IN REPLY: While there may be some theoretical increased risk of *S aureus* cutaneous infections because of the effect of statins on lipids in the skin, it is not clear what role statin use has played in the changing epidemiology of *S aureus* infections. Other studies suggest that statins may ameliorate sepsis because of their anti-inflammatory effect.¹

SUSAN REHM, MD
Department of
Infectious Diseases
Cleveland Clinic

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