lar crescents were present exclusively in three glomeruli. The crescents were accompanied by foci of fibrinoid necrosis with endocapillary and extracapillary fibrin. Tubular atrophy and interstitial fibrosis were absent. Interstitial inflammation was present, focal and accompanied by tubular degenerative changes. There was evidence of necrotizing vasculitis. IF revealed granular, segmental to global glomerular capillary wall positivity for IgG, kappa and lambda. Weaker staining for C3 was detected. Staining for fibrinogen highlighted areas of glomerular fibrinoid necrosis. Immunofluorescence shows intense staining of the arterial wall for IgG.

The final diagnosis was 'membranous glomerulonephritis with superimposed ANCA-associated vasculitis and extracapillary proliferation'. The patient started a 6-month course of methylprednisolone (1 g i.v.) for three consecutive days at months 1, 3 and 5, followed by methylprednisolone *per os* alternated with cyclophosphamide *per os*. The patient is in partial remission. Creatinine fell to 1.6 mg/dL while proteinuria reduced to 1.2 g/24 h. Treatment ended on September 2010.

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## Coagulase-negative staphylococcus—oftentimes a virulent masquerader

To the Editor,

Septic arthritis caused by *Staphylococcus lugdunensis*, a coagulase-negative species, is a rare entity. As of 2008, there were only 58 confirmed cases [1] of bone and joint infections caused by this particular staphylococcal species, potentially due to the fact that many microbiology labs identify but do not speciate coagulase-negative staphylococcus [2]. Despite the presence of confirmed cases of bone and joint infections due to *S. lugdunensis* in the literature, we believe that this is the first case of septic arthritis caused by this organism in ESRD patients with long-term indwelling venous catheters. We present our observations below.

A 60-year-old African American male with ESRD and an indwelling venous catheter for access presented to his primary care physician with a 2-week history of pain, swelling and warmth in his right knee. He reported a low-grade fever of 100°F. He was initially treated with prednisone by his primary care physician, and due to unresolving symptoms, he underwent an arthrocentesis which showed 46 333 cells per cubic millimeter, no visible crystals, and cultures that grew a coagulase-negative staphylococcal species, later identified as *S. lugdunensis*. As such, the patient was admitted. Blood cultures drawn from the indwelling venous catheter grew identical organisms, and antibiotics were started empirically in the form of vancomycin. Orthopedic consultation was obtained, and the patient underwent arthroscopic irrigation and debridement of the right knee, during which frank pus was noted within the right knee. Knowing that *S. lugdunensis* is a virulent pathogen, we performed an echocardiogram showing vegetation in the mitral valve leaflet which indicated a subacute versus an old, healed vegetation [3].

In our patient with septic arthritis, the source for *S. lugdunensis* was most likely the indwelling venous catheter as it grew the identical organism. What is striking about this organism is that in contrast to other coagulase-negative species, *S. lugdunensis* has been associated with aggressive infections likely due to the fact that it shares many of its virulence factors with *Staphylococcus aureus* such as proteases, lipases and hemolysins [1,4]. A learning point from this case is to ask microbiology labs to speciate out coagulase-negative staphylococci in the appropriate clinical scenario. The likelihood of *S. lugdunensis* isolated from a clinical culture specimen being a pathogen versus a contaminant varies anywhere from 91% to 30% according to published data [5]. Missing *S. lugdunensis* could be catastrophic for the patient, as the bacteria can cause acute endocarditis, septic arthritis and vertebral discitis, especially since the bacteria is usually pan-sensitive to antibiotics. As microbiology labs speciate more coagulase-negative staphylococci, we are likely to identify *S. lugdunensis* more frequently in clinical infection.

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