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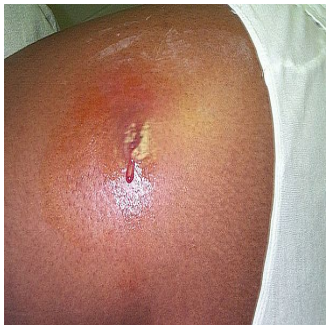
"Bio-floaters" in Antibiotic-Resistant Joint Infections



Results of a new study by Thomas Jefferson University (PA, USA) scientists published in the *Journal of Infectious Diseases* could help explain the joint pain caused by different infections, including Lyme disease, and why these infections are so resistant to antibiotic treatment.

"Our goal was to determine why Staphylococcus aureus, particularly methicillin-resistant Staphylococcus aureus (MRSA), infections of the joint tend to be relatively unresponsive to standard antibiotic treatment," said Sana Dastgheyb, lead author on the study and student at Jefferson's Graduate School of Biomedical Sciences.

It is a common practice for researchers to study bacterial growth in standard growth medium. However, in this particular study, Jefferson scientists wanted to determine whether the bacteria behaved differently in synovial fluid, the liquid that surrounds the joints. Hence, the Jefferson team cultivated several strains of MRSA in human synovial fluid, blood, and typical bacterial growth medium.

The research team found that the bacteria begin to grow as clumps in the synovial fluid, and that these clumps share many of the same properties as biofilms. For example, the clumped bacteria embed themselves in a protective mesh of proteins that resist the penetration of antibiotics. Moreover, as the bacteria slow their growth, they become less susceptible to antibiotics which are designed to target rapidly growing cells like bacteria.

"Biofilm formation has been suspected to play a key role during septic arthritis and prosthetic joint infection," said Noreen Hickok, PhD, Associate Professor in the Department of Orthopaedic Surgery at Jefferson's Sidney Kimmel Medical College. "This study could help explain why these infections have been so difficult to treat and point to therapeutic approaches that could make antibiotics more effective."

To prevent the formation of biofilm clumps or the so-called "bio-floaters", the Jefferson team pre-treated the synovial fluid with a plasmin enzyme that degraded the protein matrix. This pre-treatment enabled the research team to reduce the formation of bio-floater clumps and increase the bacteria's susceptibility to antibiotics.

Physicians have long speculated about the hard-to-treat nature of joint infections. "The study also helps explain why joint infections are so difficult to diagnose, even when there are overt signs of infection," Dr. Hickok said. Current tests for bacterial growth cannot differentiate a single bacterium from a bio-floater clump containing millions of cells, leading to an underestimation of the infection or a lack of detection altogether.

"This study strikes at the heart of one of the most pertinent questions in medicine, namely how the medical community can use antibiotics in the most effective manner to prevent infections," noted Javad Parvizi, MD, the James Edwards Professor of Orthopaedic Surgery at Jefferson's Sidney Kimmel Medical College, Director of Clinical Research at The Rothman Institute, and an author on the study. "Dr. Hickok and her team, by conducting this groundbreaking research, have enhanced our understanding of this phenomenon and opened our eyes to a very novel concept."

Dr. Hickok and Michael Otto, PhD, a Senior Investigator with the Laboratory of Human Bacterial Pathogenesis National Institute for Allergies and Infectious Diseases, National Institutes of Health, served as co-senior investigators on the study.

Although the pre-treatment appeared to stall growth in the lab, the Jefferson team said more research was needed before the results could be translated to patient care.

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