Innovative advancements in bedside applications can save, extend, and improve the quality of life for patients worldwide. Innovation addresses specific unmet needs when evaluated clinically for safety and efficacy. However, the process does not stop there. A post-market surveillance program is usually in place to address design and performance issues surfacing post product release, which is further enhanced by case reported outcomes from users [the clinical/surgical community] and their patients. This dynamic is captured in the Case Report featured by the Journal of Materials Science: Materials in Medicine March edition, entitled: “Debris of carbon-fibers originated from a CFRP (pEEK) wrist-plate triggered a destruent synovitis in human” [free article download].

Four investigation units (two surgical and two research units) joined in generating this case report. The report focuses on triggers of localized inflammation at the implant site of carbon-fiber-reinforced-polymer [CFRP] wrist plate. This particular case exemplifies the fine balance between product design and manufacturing versus clinical efficacy. CFRP has been encouraged and promoted in both orthopedic and trauma surgery applications, and minimal clinical inflammatory response to the component materials has been reported in the literature. Further, the composite implant itself is designed to ensure that the carbon fibers are deeply embedded into the host structure. A primary “push” for the use of this product in orthopedic and trauma surgery is based on its inherent X-ray transparency, which is critical in assessing fracture reduction and healing. This featured case, however, reports on the undesired onset of a destruent synovitis in a patient who had received such an implant to stabilize and heal a wrist fracture. The authors propose that this outcome is the result of exposure to carbon fibers. The question the collaborative units set-out to answer focused on the potential mode by which the carbon fibers were exposed leading to the reported inflammatory response. Professor Merolli helped me understand the strategy employed in addressing this question.

It has been documented that the surfacing of carbon fibers [CFs] and exposure to the local implant environment is the result of two primary mechanisms: (a) debris resulting from tightening of the composite plate screws [“stripping”] resulting in bulk aggregates of CFs; and (b) detachment of CF tips exposed during the manufacturing process. The two mechanisms are not mutually exclusive, but rather contribute to a varying extent to the observed outcome. As an ongoing investigative direction, the collaborative units are currently evaluating additional retrieved plates to bring further clarity to the blend of triggers [design + manufacturing versus surgical procedure] in an effort to ascertain their individual contribution to the inflammatory response. The ultimate goal is for the product manufacturers and clinical users to join forces in resolving the documented clinical outcome, especially in this circumstance when additional case reports support the current findings.

In this specific case report, CF debris has been identified as the trigger of a chronic inflammatory response which evolved in a destruent synovitis. This outcome is supported by the histopathological assessment of the retrieved implant that shows disrupted CFs originating from the plate and a clear, early stage chronic inflammatory response to CFs where monocytes/macrophages are recruited to multinucleated giant cells with granuloma formation. In terms of patient follow-up, the collaborative units have postulated that perhaps an earlier retrieval and a more robust anti-inflammatory therapy could have limited the observed damage. It is also recognized that the current inflammatory response could lead to a long term risk of an autoimmune disease onset, at which time the current incident, buried in the patient’s medical history, might not be considered as the actual trigger..

The electron micrograph assessment revealed longitudinal traction along the CFs. During the manufacturing process, pEEK coats the CF surface. However, Professor Merolli explained that when torsion occurs, cracks and blebs are formed within the pEEK coating, thus unmasking the CF fibers. It is currently understood that the exposure of CFs and subsequent onset of inflammation is not the result of a steady CF debris accumulation, but rather of a sudden CF exposure more likely to occur close to the implantation time or in the event of an implant mechanical failure. Professor Merolli further commented on the added risk of composite-plated implants in the proximity of delicate tissue structures, such as tendons and their sheaths, which are more
prone to accentuated damage from the local onset of a chronic inflammatory response. Finally, at the time of the plate retrieval, it was documented that the plate was correctly positioned and fixed.

In conclusion, we have a product where it is too early to estimate its rate of compromised efficacy, leading to destruent synovitis. Evidence is being collected to determine if such an outcome is rare, linked to a specific patient profile/implant location, or if this outcome is a recurrent feature requiring a reconsideration of the product or manufacturing process.

I would like to thank Professor Merolli and his colleagues for kindly agreeing to offer a cohesive clinical and investigational account of their ongoing work in the clinical efficacy of CFRP (pEEK) orthopedic implants.

To our readers: I am looking forward to your comments that can be sent to gabriela.voskerician@case.edu using the heading “Editors’ Choice”. We hope to develop this feature into a dynamic forum think-tank.