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Editorial

Shunts and security—the polytetrafluoroethylene leak

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Summary Plasma leakage in polytetrafluoroethylene grafts arises through complex interactions between the prosthetic physical/chemical microstructure and patient environment. While many of the precipitating factors can be avoided by careful graft-handling and meticulous surgical technique, this unpredictable complication still contributes significant morbidity to shunt procedures. Protein sealing of the graft offers a possible solution to the problem.

SEROUS LEAKAGE THROUGH POLYTETRAFLUROETHYLENE grafts is a rare but well-recognized occurrence in both peripheral vascular surgery[1-4] and systemic-pulmonary shunts.[5-7] The case described in this issue by Masiello and colleagues (pages 94-95), however, is the first reported instance in a central aortopulmonary conduit, a site previously thought to be invulnerable to this complication.[8] The importance of understanding this phenomenon lies in both effective management (or, ideally, prevention) and the socioeconomic implications of prolonged hospitalization after an otherwise straightforward surgical procedure.

Polytetrafluoroethylene vascular grafts consist of polymerized molecules which have been extruded as longitudinal fibers between solid nodes.[9] The mean interfibrillar distance is 5 mm, and average internodal distance 25-30 mm, while the fibers themselves have a diameter of about 0.5 mm.[10] This compares with a red cell diameter of 7-8 mm and gives the graft a composition of 15-20% polytetrafluoroethylene and 80-85% air by volume.[9,10] Chemically, polytetrafluoroethylene is both stable and inert as the result of its electronegative surface fluorine atoms, which also render the material hydrophobic.

Following implantation, the lumenal aspect of the graft is exposed to blood, while the outer surface lies against solid tissue or within extracellular fluid. A variable pressure gradient is produced across the wall and, in most cases, also along the length of the conduit. During the subsequent 48-96 hours, air is displaced from the polytetrafluoroethylene by penetration of protein-rich fluid, which transforms the opaque, white graft into a transparent, grayish material.[5,11] This so-called "wetting" process occurs because constituents of the blood or interstitial fluid convert the hydrophobic surface of polytetrafluoroethylene into a hydrophilic one. Wetting occurs predominantly from the exterior towards the lumenal surface, probably as the result of higher concentrations of enzymes or surfactants in tissue fluid, as well as flow characteristics of blood within the lumen.[11] During a less-certain time-frame, it is likely that proteinaceous material is deposited within the graft interstices, and cellular elements attach to both the external and internal surfaces, although there is scant clinical or experimental evidence for these assumptions.[1,5]

Leakage through the graft, then, probably occurs when there is a chance interaction among several complex variables. Mechanical deformation increases the space between polytetrafluoroethylene fibers. This has been produced experimentally by circumferential force equivalent to an intraluminal pressure of 100-200 mm Hg,[10] and attributed also to stretching or bending of the graft[11] and ionizing radiation.[9] Although high pressures are uncommon in pediatric systemic-pulmonary shunts, they may arise through compression of a clamped graft or, as advocated by some authors,[12] by rapid filling against a distal clamp.[13] Most distortion of the prosthetic microstructure occurs with the initial stress and, because of polytetrafluoroethylenes inelastic qualities, is not reversible.[9,10] In an elegant computer simulation, Tabata and Associates demonstrated the theoretical relationships among interfibrillar distance, intraluminal pressure, and graft leakage.[9] Plasma leakage, in this model, became inevitable at certain values of surface tension or internal pressure, and could be influenced by as little as 0.001 mm change in the distance between polytetrafluoroethylene fibers.

An imbalance between the "wetting" process and sealing of the graft by protein deposition will also result in leakage. Wetting is accelerated by exposure to agents which alter the surface tension of polytetrafluoroethy-

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lene, thus diminishing its water-repellency. These include organic solvents (such as isopropyl alcohol), Betadine,[11] acidosis,[14] blood,[10] antibiotic solutions[15] and tissue fluids.[2] Once rendered hydrophilic, the porous nature of the graft allows immediate filtration or hemorrhage through its wall.[11,15] In this environment, flow velocity, reduced hematocrit, graft bending, and pressure may exacerbate the filtration rate.[11] Leakage, however, has occurred in only one of several grafts implanted simultaneously,[15] suggesting that subtle structural variability in the polytetrafluoroethylene still may be contributory as well.

That the vast majority of polytetrafluoroethylene prostheses do not ultrafiltrate is likely due to timely blockage of the interfibrillar spaces by protein deposition, as well as attachment of fibrous tissue both externally and internally. Histology of leaking grafts has shown sparse development of the protein matrix with little or poorly adherent, acellular surface tissue.[1,5] A humeral fibroblast inhibitor has been identified as one cause of such impaired graft incorporation.[16-18] Production of this substance may be graft-induced, as removal of the prosthesis is followed by its disappearance.[16,18] The approximately ten-fold greater incidence of plasma leakage through systemic-pulmonary shunts compared with peripheral vascular grafts,[3,6,18] could reflect some limitation of fibroblast activity or availability within the pleural space. This would be consistent also with the observed accentuation of shunt leakage by silicone wrapping[6] as well as an apparent genetic predisposition in some patients.[5] Subclinical prosthetic biofilm infection with slime-producing Staphylococcus epidermidis has been shown experimentally to cause sterile exudate and poor graft healing,[19] but its importance in patients is uncertain and difficult to elucidate.

From the foregoing, it becomes apparent that plasma leakage must occasionally and inevitably complicate polytetrafluoroethylene shunts, despite meticulous avoidance of all presently known precipitating factors. Its management will depend upon the clinical manifestations. Immediate weeping of plasma or blood from the graft surface probably indicates extreme structural deformation or inappropriate wetting. Paradoxically, this situation is the easiest to recognize, and appears to respond to reversal of any heparin and transfusion of clotting factors,[11,15] possibly because all of the necessary hemostatic agents pass simultaneously from the lumen into the graft wall. The logical treatment of on-going, chronic, or delayed leakage, as suggested by Massiello and colleagues, would be enhancement or fortification of protein deposition. In their case, this was achieved by wrapping the graft with absorbable collagen hemostat, while others have reported success with topical microfibrillar collagen,[20] collagen fleece soaked in fibrin glue[7] and extrusion of aprotinin and thrombin through the graft wall.[21] Experimentally, precipitation of protein by application of tannic acid also produced an effective barrier.[11] Having sealed the graft, which is facilitated by brief interruption of flow to permit firm adherence of the hemostatic agent, leakage does not recur.

Local containment of the filtrate by surrounding tissues or an acellular protein capsule results in formation of perigraft seroma, the diagnosis and treatment of which are less clear-cut. The seroma may either limit further passage of fluid through the graft, probably coincident with protein sealing,[22] or expand to compress adjacent structures.[6] Compromise of graft function, itself, however is rare,[4] probably because external pressure becomes sufficient to stop the leak before causing compression of the prosthesis. In the absence of complications, observation with serial computerized tomographic scanning is sufficient to exclude important tissue displacement during the time-frame for which most patients require palliation.[22] If there is progressive enlargement before the shunt can be taken down, removal of the seroma and sealing of the graft should be adequate intervention.[18,20] Graft replacement probably is not necessary and, in the authors experience, has been technically challenging, due to the friable nature of the pulmonary artery in this situation. Plasmapheresis in adults has effectively limited seroma formation due to fibroblast inhibition.[17]

Given the socioeconomic penalties of prolonged hospitalization and reoperation effected by this complication, prevention of plasma leakage is highly desirable. This might be achieved by routinely "presealing" the graft with topical application of hemostatic agents at the time of implantation. Alternatively, such a treatment might be usefully incorporated into the manufacturing process, analogous to the collagen or gelatin sealing of Dacron prostheses.

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