Fate of the snake: A parable of blame

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A few months after I became Chair of the Department of Surgery at the University of Michigan, I received a call from the Medicine Chief Resident advising me that they had inserted a Greenfield filter in a pulmonary embolism patient only to have the patient die a few days later of recurrent embolism. My response was to request an autopsy. At the autopsy table, a long snakelike clot was found trapped by the filter, completely obstructing the vena cava. Review of the chart indicated attempted resuscitation using vasopressors and inotropic agents alone.

When I discussed this with the resident, he raised the legitimate question of how to make the diagnosis in an emergent situation. I reminded him that recurrent pulmonary embolism would cause right-sided heart overload and an elevated jugular venous pressure, whereas an acutely obstructed vena cava sharply reduces venous return with the opposite effect on jugular pressure. Such a functional hypovolemia would much more likely respond to fluid resuscitation than to inotropic agents.

Of course, most emboli are smaller and clinically silent, making a diagnosis more difficult. In the early 1970s when we began using the filter, there was no way to know what was happening to it short of contrast-enhanced venacavography. In lieu of royalty payments, Medi-Tech had agreed to fund patient follow-up, and I persuaded many of them to return for those studies. After finding occasional evidence of clots entrapped, I asked hematologists at the University of Oklahoma about their fate but heard only pessimism about the chances of resolution. So it was a pleasant surprise to see venographic clearing of the filter after 6 months in most of them and without correlation with long-term anticoagulation. As ultrasound brought better opportunities for visualization, I envisioned more studies of both clot and filter behavior. Although we had observed long-term patent filters in >90% of patients followed up at 20 years, other groups were reporting higher levels of filter "thrombosis." The quotation marks are appropriate because a large assumption is made when a filter is found to be obstructed. Instead of acknowledging that the filter may have actually done its job and trapped migrating clots, it is more likely to be deemed thrombogenic. Such an assumption ignores many years of favorable intravascular experience, not only with filters but also with other well-tolerated catheters and devices. Now that it has become routine to weigh the risk of such "thrombosis" as greater than the risk of recurrent embolism, the cost and risk of filter removal are assumed justified. Furthermore, devices designed to be retrieved must compromise on security of fixation, rendering them less stable. Until we achieve the ultimate control of intravascular coagulation that makes filters a historical footnote, we should acknowledge the mystery of in vivo clot behavior and question the conviction of filters as thrombogenic when they may simply be guilty of performing their intended function.

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