Hemorrhagic Panniculitis Caused by Delayed Microemboli From Intravascular Device

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Case Report/Case Series

REPORT OF A CASE

A woman in her 70s presented 6 months after a complex aortic aneurysm repair with several large ecchymoses radiating from firm subcutaneous nodules on the buttocks, arms, and thighs. Skin biopsy specimens revealed extensive hemorrhage and a panniculitis with sparse, subtle, intra-arteriole, gray amorphous deposits that, on analysis by scanning electron microscopy with energy-dispersive radiography analysis and infrared spectrometry, were most consistent with a hydrophilic polymer. This type of hydrophilic polymer coats catheters and stents such as those used in aortic aneurysm repair.

CONCLUSIONS AND RELEVANCE

This is an unusual case of microemboli from the polymer coating intra-arterial stents starting months after placement and causing a panniculitis. Prior observations show that polymers coating intravascular devices have the potential to break down gradually and long after the device’s placement, but clinical consideration for delayed microembolization is underrecognized until catastrophic impairment or death.

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F ew articles document intravascular catheter insertion devices as the source of polymer microemboli. These rare cutaneous lesions typically occur at the insertion site within a few weeks of the procedure. These cases further describe a delayed presentation found predominantly in the kidneys, lungs, and brain. To our knowledge, there are no documented cases of a vascular stent shedding polymer microemboli. Here we report an unusual presentation of cutaneous polymer microemboli that presented months after a complicated abdominal aortic surgical repair. This case report did not require institutional review board approval or a waiver. Oral consent was obtained from the patient for publication, and medical records were provided by the patient.
identified atrophy of the left kidney with no other significant abnormalities. Chest radiography findings were normal.

Two punch biopsies from the right and left thighs were performed. The biopsy specimens revealed extensive dermal and subcutaneous hemorrhage, slight thickening of the subcutaneous fibrous septa with increased inflammation composed of lymphocytes and eosinophils, lobular fat necrosis in the pan-niculus lobule, and rare perivascular multinucleated giant cells (Figure 2). However, both specimens showed intravascular and subendothelial gray amorphous deposits (Figure 2 and Figure 3). Staining of these deposits was negative for Alcian blue, von Kossa, periodic acid-Schiff, elastin, and Congo red but unexpectedly positive for nuclear fast red used as a counterstain for the Alcian blue and von Kossa sections (Figure 4). An elastic stain confirmed the amorphous material to be in small arterioles. Further evaluation of the deposits with scanning electron microscopy with energy-dispersive radiography analysis and infrared spectrometry revealed them to be a foreign material, consistent with a hydrophilic polymer.

The patient continued to develop new blue-hued papules on the palms and fingers until 2 months after onset, when she received 2 intravascular stent repairs for a type III endoleak with celiac artery fenestration. On follow-up 6 months later, she no longer developed papules, plaques, or ecchymoses, and the older lesions resolved.

**Discussion**

This case demonstrates an unusually delayed presentation of microembolic disease from the polymer coating of a surgically implanted device presenting with a panniculitis and ecchymoses. With the few reported cases of polymer microemboli, the cutaneous manifestations occurred abruptly, in a localized manner, and were thought to be secondary to the insertion devices and not the stents.1 In this case, it is most likely that degradation from and embolization of the...
The mechanisms behind these polymer microemboli remain uncertain. Insertion devices, such as catheters, are most commonly implicated after vascular surgery, but the emboli in these cases develop days to weeks after surgery.2-5 However, Denardo et al6 demonstrated that stents degrade over time; therefore, this occurrence may be responsible for polymer microemboli that occur weeks to months after surgery. This study included 4 US Food and Drug Administration–approved companies and 5 stents from each company and involved performing microscopic evaluation of stents both before and after balloon inflation. One stent was noted to have preinflation damage, and all tested stents demonstrated degradation after inflation. However, there remain no US Food and Drug Administration guidelines for governing the level of particulate matter that can be generated by coated medical devices.7 We hypothesize that degradation of the polymer used to coat the stents would most likely explain the delayed presentation of months rather than weeks. While this outcome may represent the natural degradation of the polymer in the human body, turbulent blood flow over a free edge of the stent, which is inherent in a type III endoleak, may lead to gradual rather than abrupt stent damage. An endoleak is the presence of blood flow between a graft and the vascular component that is being used. Specifically, type III endoleaks result from a defect in the graft material or inadequate sealing in areas where stents may overlap.5 In our case, the initial aortic aneurysm repair was conducted 6 months before onset of symptoms and marked by multiple significant intraoperative complications that may have predisposed the stent’s polymer coating to degradation and embolize. Repair of the type III endoleak and a new stent resolved her dermatitis, suggesting the polymer from the initial stent was the source of the microemboli.

Cutaneous emboli from polymers related to the insertion device have presented as firm violaceous nodules near the vascular insertion site in areas such as the extremities.4-5 Most often, however, polymer microemboli from vascular catheterization have been reported in the lungs, heart, brain, and kidneys,3-5 and these microemboli were predominantly noted with delayed presentation ranging from weeks to years.5 To our knowledge, all polymer microemboli in our case were localized to the skin, and, fortunately, the patient sustained no other organ damage.

Conclusions

This case demonstrates that it is important for both dermatologists and pathologists to be aware that iatrogenic microemboli from stents may occur months after vascular surgery. These emboli may present as panniculitic or hemorrhagic lesions. A thorough medical history review and early consideration of this eventuality in the differential diagnosis may prevent significant morbidity or mortality.
Hemorrhagic Panniculitis and Intravascular Device

NOTABLE NOTES

Three-Dimensional Printing of the Skin

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Three-dimensional (3-D) printing is the creation of a 3-D object from a digital model through the successive layering of material, a process known as additive manufacturing. The first 3-D printer was developed in 1984 by Charles W. Hull, but only since the beginning of this decade has the technology become widely available for a multitude of applications in engineering, industrial design, biotechnology, and medicine. In a process known as 3-D “bioprinting,” researchers have refashioned inkjet printers to layer living cells into a scaffolding medium, constructing blood vessels, tissue, and entire organs.

Skin burns compromise the barrier and physiological functions of the skin and can cause water loss and opportunistic pathogen entry. Autologous split-thickness skin grafts are sometimes used to cover burn wounds; however, their use is limited in large-scale injuries that require extensive grafting. This is an area in which tissue engineering holds great promise. Conventional tissue engineering involves the extraction of keratinocytes from harvested full-thickness or split-thickness skin and culture on a dermal surrogate made of synthetic nylon or polycarbonate. After 3 to 4 weeks of growth, the keratinocytes are stratified by degree of maturation in the epidermis and the engineered skin grossly mimics natural skin. However, conventional tissue engineering still lacks the variety of immune cells and multipotent stem cells of natural skin, which are challenging to both culture and precisely position within the epidermal strata. Three-dimensional bioprinting offers notable advantages over conventional tissue engineering by allowing the precise layering of extracellular matrix, growth factors, and numerous types and sizes of epidermal cells into their optimal positions in a highly reproducible manner. Furthermore, printed skin does not require long periods of incubation and growth. In a proof-of-concept design by Lee et al., a skin prototype printed using fibroblasts and keratinocytes in the epidermis and collagen in the dermis was found to be structurally and functionally similar to human skin tissue by immunofluorescence. Koch et al. demonstrated that keratinocytes and fibroblasts printed by a laser-assisted technique formed their own intercellular adherens and gap junctions, demonstrating that printed cells can generate real tissue. Laser-assisted bioprinting has also created a simple skin substitute that restored skin functionality in mice with full-thickness skin wounds. Skin that can be easily printed and grafted onto large-scale wounds is likely many years away, pending the development of techniques to integrate the vascular systems necessary for the functioning of large, thick grafts. But in addition to its applications in skin grafting, engineered skin provides a superior medium for the investigation of disease pathophysiology and drug delivery, closely mimicking human skin physiology and potentially absolving animal models in pharmaceutical research. Considering all of its future potential, 3-D printing is certainly an exciting new area of bioengineering and dermatological research.

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REFERENCES