Post-Catheterization False Aneurysms

Eran E. Weinmann MD and Arie Bass MD

Department of Vascular Surgery, Assaf Harofeh Medical Center, Zerifin [affiliated to Sackler Faculty of Medicine, Tel Aviv University], Israel

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Post-catheterization injury to the access artery is one of the commonest iatrogenic complications inflicted by modern medicine. Bleeding that results from such injury may lead to the formation of an organized hematoma, but if the hole in the artery does not seal, a persistent communication with the arterial lumen may result in the formation of a "pulsating hematoma" or false aneurysm. Despite its frequent occurrence, most physicians seem to be in the dark when faced with it, and experience shows that knowledge regarding its prevention, diagnosis and treatment is limited to very few specialists.

As the number of arteriographies rises exponentially both for diagnostic purposes and as a treatment modality in coronary artery disease and peripheral vascular disease, the frequency of iatrogenic arterial injury increases as well. The size of the catheter or access sheath, the liberal use of anticoagulants, the older age of the patients, and multiple punctures of diseased arteries are but a few of the factors that predict higher rates of arterial injuries and their concomitant complications. Such complications include thrombosis, embolization, bleeding and false aneurysm formation. The current rate of complications – 0.1–2.0% for diagnostic catheterization and up to 5% after interventional treatment [1] – is significant. In most series about 1% of patients will end up with a false aneurysm [2-8].

Up until ten years ago the only treatment modality for such aneurysms was open surgical repair [2-6]. In 1988 Skillman and his group in Boston [2] predicted that the need for operative repair of these lesions will continue to increase as percutaneous cardiologic diagnostic and therapeutic interventions are used more widely. Many surgeons made a plea for an early and aggressive intervention [5-7]. Those grim predictions became obsolete when the first reports of non-surgical treatment for post-catheterization false aneurysms appeared in the literature in the early 1990s, and as a result our whole approach to this disease entity changed and keeps on changing [9,10].

We now know with certainty that if left alone, spontaneous thrombosis may occur in a significant number of those aneurysms, particularly the small ones, in patients not receiving anticoagulant therapy. We also know that it is possible to treat most persisting false aneurysms, by using direct compression on the point of communication between the artery and the hematoma. It was well demonstrated recently that direct injection of thrombin into the lumen of such aneurysms initiates a thrombotic process that obliterates the aneurysm so that the compression becomes unnecessary [11]. Thus, a condition that ten years ago was considered an absolute indication for urgent surgical repair may be solved today by a percutaneous injection of thrombin.

In this publication of IMAI, the paper by Szendro and the group from Beer Sheva is paradoxically both timely and outdated [12]. Their study plays a very important role in bringing to the attention of family physicians, general practitioners, general surgeons and internists this quite common complication that they are bound to encounter a few times a year among their patients. The second salient point that they emphasize is that surgery is no longer indicated in the majority of cases and that simple non-invasive treatment is becoming the gold standard. Yet, this paper is already outdated as the new treatment modality, namely thrombin injection, is rapidly gaining popularity, and the ultrasound-guided compression, the treatment modality that the authors discuss in depth, no longer holds first place. The other point where the authors fall short of current data is their recommendation concerning injuries to the brachial artery. Their limited experience with five patients combined with some unfortunate treatment choices led them to conclude that upper extremity pseudoaneurysms, unlike those in the lower limbs, “present a potentially more serious complication and need early diagnosis with prompt intervention to minimize the high complication rate and serious long-term sequelae.” Several reports in the last few years have addressed this issue and most came up with a different conclusion [13-17]. Khoury et al. [13] reviewed 8,797 cardiac catheterizations, 3,137 of them via the brachial artery, and concluded that although the “frequency of complications was higher with brachial artery catheterizations, ...femoral artery complication were more complicated, difficult to identify and associated with significant morbidity.” It is quite evident today that the antecubital brachial approach is equally suitable for diagnostic and interventional angiography [15,17]. The major complication of this approach is acute thrombosis and limb ischemia, which is easily recognizable and treatable.

The last chapter of the saga of post-catheterization iatrogenic arterial injury is being written at present. Dedicated devices are under development to enable percutaneous sealing of the arterial puncture sites by the end of the procedure, and some
of them are already on the market. Though the existing closure devices do not reduce the incidence of local complications, they seem to be effective in reducing the time required for hemostasis [18]. Further development of such devices may ultimately result in a significant decline in the rate of post-catheterization bleeding complications. Nevertheless, retroperitoneal bleeding as well as ischemic complications due to arterial thrombosis and embolization continue to present a real threat. To combat these, we need further improved catheterization hardware such as smaller diameter catheters and access sheaths, and improved catheterization techniques, e.g., better puncture-site selection. But that is another story.

References

Correspondence: Dr. A. Bass, Dept. of Vascular Surgery, Assaf Haroef Medical Center, Zerifin 70300, Israel. Phone: (972-8) 977-988, Fax: (972-8) 977-9186, email: arbas@post.tau.ac.il

- Capsule

**Smoking and the incidence of diabetes mellitus**

In order to determine the association between cigarette smoking and the incidence of type 2 diabetes mellitus, Manson et al. studied 21,068 American male physicians aged 40 to 84 years in the Physicians’ Health Study who were initially free of diagnosed diabetes mellitus, cardiovascular disease, and cancer. Information about cigarette smoking and other risk indicators was obtained at baseline. The primary outcome was reported diagnosis of type 2 diabetes mellitus.

The results showed that during 255,830 person-years of follow-up, 770 new cases of type 2 diabetes mellitus were identified. Smokers had a dose-dependent increased risk of developing type 2 diabetes mellitus; compared with never smokers, the age-adjusted relative risk was 2.1 for current smokers of ≥20 cigarettes per day, 1.4 for current smokers of <20 cigarettes per day, and 1.2 for past smokers. After multivariate adjustment for body mass index, physical activity, and other risk factors, the relative risks were 1.7 for current smokers of ≥20 cigarettes per day, 1.5 for current smokers of <20 cigarettes per day, and 1.1 for past smokers. Total pack-years of cigarette smoking was also associated with the risk of type 2 diabetes mellitus. The authors conclude that these prospective data support the hypothesis that cigarette smoking is an independent and modifiable determinant of type 2 diabetes mellitus.