Acute Compartment Syndrome of the Hand After Transradial Catheterization

Síndrome compartimental agudo de la mano tras un cateterismo transradial

To the Editor,

The routine use of transradial access in cardiology is due to its proven advantages over femoral access. Although hemostasis is easily achieved, bleeding can occur through the puncture site (PS). This bleeding can lead to hematomas and, on rare occasions, acute compartment syndrome (ACS), which can become serious without early and appropriate treatment. Here, we present and discuss an exceptional case of ACS of the hand (ACSh) that was resolved for the first time using a conservative approach involving a quick and simple maneuver.

Preoperative coronary angiography was performed in an 82-year-old woman with severe aortic stenosis via a right transradial approach using a valved introducer (5-Fr Glidesheath, Terumo) and heparin 5000 IU. The procedure was completed without complications and with compression of the PS with an elastic bandage. Swelling immediately appeared, which progressed rapidly to marked edema of the hand. Attempts were made to compress the PS, first with a pneumatic device and then with manual compression. After 5 minutes of unsuccessful compression attempts, the patient was in intense pain, requiring opioids, and had paresthesia in the affected hand. Physical examination revealed a large tension hematoma and cyanotic and ecchymotic fingers. The hand was flexed and very painful upon movement/extension (Figure 1A). All of these signs and symptoms are compatible with the diagnosis of ACSh.

The radial artery was then compressed 3–5 cm proximal to the PS (in an area without hematoma). Although the compression stopped the progression, the tension edema persisted and there was no improvement in symptoms. Thus, we empirically decided to use a scalpel to extend the initial PS, which produced a gush of nonpulsatile blood (Figure 1B and Figure 1C). After 2 minutes of drainage, the signs and symptoms progressively disappeared. The procedure was finalized by compressing the PS (now hematoma-free) with a pneumatic device. The clinical course in the next 3 weeks was excellent and without sequelae (Figure 2).

Acute compartment syndrome, produced by increased pressure in 1 or more fascial spaces, leads to decreased perfusion pressure and muscular and nerve ischemia. Its rapid diagnosis is vital because, without early treatment, it becomes a serious condition with important functional repercussions. There are several “classic” causes, with trauma being the most frequent.

Diagnosis is clinical (involving the “5Ps”: pain, pulselessness, pallor, paresthesia, and paralysis). The most common and characteristic symptom is intense pain. This pain is refractory to analgesia and frequently disproportionate to the visible injuries.

Figure 1. A: right hand with signs of compartment syndrome. B and C: photograph taken 2 minutes after manual compression of the radial artery showing a gush of nonpulsatile blood (because it was from the hematoma and not the radial artery, which would be pulsatile).

Figure 2. Hand and forearm 3 weeks later; virtual symptom resolution (except for mild persistent ecchymosis in the forearm).
Prevalence of Neuroendocrine Tumors in Patients With Cyanotic Congenital Heart Disease

Prevalencia de tumores neuroendocrinos en pacientes con cardiopatías congénitas cianóticas

To the Editor,

Pheochromocytoma and paraganglioma are neuroendocrine tumors (NETs) that produce catecholamines derived from neural crest cells, localized in the adrenal medulla (90%) or in extra-adrenal chromaffin tissue (10%). Their prevalence is between 0.2% and 0.6% in hypertensive adults, 5% in adrenal incidentalomas, and 0.05% to 0.1% in autopsy series.1 Although they usually present as isolated tumors, they can be associated with hereditary syndromes such as multiple endocrine neoplasms, neurofibromatosis, or von Hippel Lindau syndrome. Furthermore, there have been case reports and small series highlighting the association between cyanotic congenital heart defects (CCHD) and NETs.2 Recently, Opotowsky et al.3 reported an increased risk of NETs in patients with CCHD in a multicenter study in which the role of chronic hypoxia in association with genetic susceptibility was proposed as the underlying pathogenic mechanism for these tumors.

A retrospective analysis was performed of 3311 adults with congenital heart defects, 173 with CCHD, and 33 with Eisenmenger syndrome in a national referral center for adult congenital heart defects. The median length of follow-up was 25 years (range, 10.5 years). All patients with suspected NET under follow-up in the endocrinology department underwent computed tomography and metaiodobenzylguanidine scintigraphy, with monitoring of catecholamines in urine.

A total of 8 NETs were identified in 7 patients with CCHD (4.6%) (Table 1). Overall, 48.8% were men and the median age was 40.0 years (range, 19.0–47.0 years). All patients had active cyanosis at the time of diagnosis (mean, 36.0 ± 11.3 years), including 1 patient with Fontan circulation with venovenous collaterals. The mean baseline arterial oxygen saturation was 83.4% ± 6.3%, mean hemoglobin was 18.1 ± 2.0 g/dL, and mean hematocrit was 66.5% ± 7.3%. Three patients had Eisenmenger syndrome (9.1% of all patients had this syndrome). On analysis of all patients with congenital heart defects, including those with noncyanotic disease, 1 additional NET was identified in a patient with partial anomalous pulmonary venous drainage (0.2% overall).

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