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Collateral artery pathways of the femoral and popliteal artery

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ABSTRACT

Background: The role of collateral artery circulation in the lower limb is under debate but clinically relevant, particularly when using covered stents for occlusive disease. Covered stents seem to outperform nitinol stents in extensive disease, but collaterals could be essential in case of acute thrombosis. In the present study, we describe the collateral pathways of the deep and superficial femoral artery (DFA, SFA) and the popliteal artery (PA), observed in human cadavers.

Methods: Ten fresh frozen cadaver legs were selected. The SFA and DFA were separately cannulated and injected with a different colored latex mixture simultaneously. After curing of the latex, the circulation was dissected thus visualizing the main arteries and their collateral vessels. The process was photographed and recorded, and collateral pathways were analyzed using a standardized vessel segmentation. The upper leg was divided in three regions, that is, the femoral triangle (F), the adductor canal (H), and the popliteal fossa (P) that, in turn, were split in three segments (1, 2, and 3, from proximal to distal).

Results: Overall, 113 collateral vessels were found; 69 originated from the DFA, 34 from the SFA, and 10 from the PA. The majority of collaterals originating from the DFA terminated in the SFA (57%). Fifty-six of 113 collaterals (50%) ended in either the distal adductor channel (H3) or the proximal PA (P1). Another 28 collateral arteries (25%) had their origin in this segment (H3, P1) and mostly connected to the P2 and P3 segments. Forty-three collaterals of the DFA and H3 segment had a direct or indirect connection to below the knee muscles.

Conclusions: The majority of collaterals originate from the DFA, and the greater part of all collaterals has a connection with the H3-P1 segment. This observation may have clinical implications in the planning of endovascular procedures.

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Introduction

Occlusive disease of the superficial femoral artery (SFA) and popliteal artery (PA) is prevalent with an incidence of 5% in men and 2.5% in women over the age of 60 y and may cause intermittent claudication (IC) and critical limb ischemia (CLI). Conservative therapy of IC consists of (supervised) walking exercise, which is considered to stimulate collateral artery development.[1] The clinical significance of the collateral circulation in patients with SFA occlusive disease, however, is under debate. Studies focusing on collateral artery development in response to exercise training have showed no improvement in lower leg blood flow suggesting that other mechanisms are involved.[2,3] For example, a metabolic adaptation of mitochondria could be an alternative mechanism of action.[4] The anatomical origin and pathways of the collateral arteries in the lower extremity are still largely unknown.

Over the last decades, endovascular techniques and devices for the treatment of SFA occlusive disease have considerably evolved. The main advantage of endovascular treatment is a lower morbidity and earlier recovery, when compared to open surgery. Nitinol stents have significantly improved the outcome of endovascular treatment, but results tend to be worse in more extensive disease (i.e., longer lesions).[5] Covered stents outperform nitinol stents in these longer lesions, as was shown in the Viastar trial.[6] A drawback of endovascular treatment of SFA occlusive disease, particularly when using covered stents, is the potential obliteration of collateral arteries. The concern is that a loss of collateral circulation might reduce limb salvage rate in case of an acute occlusion and may negatively affect the level of an eventual amputation, although a previous study showed no validation for this concern.[7] This emphasizes the clinical significance of knowledge of lower extremity collateral circulation.

In the present study, we assessed the SFA, DFA, and PA collateral artery pathways, obtained from human cadavers, in an attempt to increase knowledge on the anatomical variations in this system. This could attribute to a more scientific-based procedural planning of endovascular procedures.

Methods

Design

An anatomical cadaver study was performed at the Department of Anatomy of the Radboud University Medical Center, Nijmegen, The Netherlands. Cadavers were obtained according to the Dutch body donation program for research and education.[8] Body donation of humans aged 60 years and older with a valid handwritten testament was accepted.

Cadaver specimens

Ten fresh frozen intact cadaver legs (from umbilicus to foot) with a patent external iliac artery and no signs of trauma, prior vascular surgery, and other scars were selected. One leg was excluded and replaced because of excessive leakage of the colored latex mixture from a defect in the common femoral artery (CFA), probably due to femoral puncture. Cadaver characteristics such as age, sex, and cause of death were noted.

Freezing protocol

The fresh frozen cadaver specimens were frozen within 72 hours after death at −40°C for at least 1 day and stored at −20°C. Before use, the cadaver specimens were thawed at room temperature for at least 24 hours.[9]

Selection of the injection fluid

Colored latex (Emadere bvba, Ledenberg, The Netherlands; Bichemie Coatings, Almere, The Netherlands) was selected as injection product, based on a previous study.[9] For each leg, two mixtures of 150-mL latex were prepared and colored with 15-drops/30 mL blue or green pigment, for the SFA and DFA, respectively.

Vascular infusion process

The CFA was identified and dissected distally to the femoral bifurcation. The SFA and DFA were isolated 10-15 mm from surrounding tissue. The inguinal ligament was left intact. The SFA and DFA were cannulated separately through the CFA with curved heparin needles (Van Straten Medical B.V., Nieuwegein, The Netherlands) 2-3 cm into the vessels and fixed with 2-0 silk sutures to prevent dislocation and leakage. The colored latex mixture was simultaneously injected in both the SFA (blue color) and DFA (green color) using a system as illustrated in Figure 1.[9,10] Infusion lasted for at least 15 min at a constant pressure of 19.6 kPa and a maximum of 60 min. Infusion was terminated 5 min after leakage of the latex from a small incision in the digital artery of the hallux. Vessel clamps prevented colored latex leakage from other vessels. The injected legs were covered with wet cloths to prevent dehydration and were left to cure for at least 24 h at 20°C.

Dissection of the arterial system

Dissection was performed after complete curing of the colored latex using a uniform protocol. Skin and subcutaneous tissues were removed, paying attention to subcutaneous perforating vessels. The upper leg was divided in three regions, that is, the femoral triangle (F), the adductor canal (H), and the popliteal fossa (P).

The femoral triangle was defined as a triangular landmark, bounded superiorly by the inguinal ligament, medially by the adductor longus muscle, and laterally by the sartorius muscle. The apex of the femoral triangle is formed by the intersection of the sartorius and the adductor longus muscles. The SFA in the femoral triangle was divided into three equal segments (proximal [F1], middle [F2], and distal [F3]) by marking the midline of the inguinal ligament and the apex of the femoral triangle.[11]

We defined the adductor canal (i.e., subsartorial canal or Hunters' canal) as the canal extending from the apex of the femoral triangle to the adductor hiatus in the tendon of the