

Long-term results of endovascular treatment for May-Thurner syndrome

Wyniki odległe śródnaczyniowego leczenia zespołu May-Thurnera

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May-Thurner syndrome is a rare condition caused by compression of the left common iliac vein (LCIV) by the overlying right common iliac artery. This syndrome was shown to occur most frequently between the second and fourth decade of life, more often in women, with a prevalence of 2–5% in the general population. The syndrome was first described by R. May and J. Thurner in 1957. Compression of the LCIV causes disability of blood outflow from the left lower extremity,



Figure 1. A 60-year-old female with patent stent in the left common iliac vein with preserved morphologically proper blood flow in a follow-up control three years after operation



Figure 2. Patient aged 33 years. Upper part of the venous stent located in the vena cava inferior (two years after operation)



Figure 3. Patient aged 25 years. Additional stent in the external iliac vein after recanalisation procedure (15 months after operation)

which results in unilateral LCIV thrombosis, chronic venous insufficiency, and the creation of multiple collaterals crossing the pelvis. Seven women, aged from 22 to 60 (mean 38) years were operated in our Department, and the consecutive mean follow-up period was 25.7 (12–36) months. In the preoperative period, in four cases a critical stenosis of LCIV (from 2 to 3 mm) was diagnosed, while in three other patients the stenosis was accompanied by a distal iliac vein thrombosis. In all cases endovascular therapy was performed. Access to the left common femoral vein or popliteal vein — in the case of iliac deep vein thrombosis — was achieved. After initial predilatation and catheter-based aspiration (in patients with iliac vein thrombosis), intravenous stent placement was performed. For this procedure four Veniti, two Zilver Vena, and one Sinus Venous stent/s (14–18 mm × 60–140 mm) was/were used. Three months after stent implantation duplex Doppler ultrasound revealed stent stenosis in the proximal part of LCIV in three patients. In all those cases re-interventions were conducted. Because of the poor radial strength of previously implanted stents, which was insufficient to compensate for the compression by LCIV, additionally 12–20-mm diameter Wallstents were used. This allowed restoration of blood through LCIV in further observations. Directly after the angioplasty procedure, all patients were given clopidogrel 1 × 75 mg and rivaroxaban (initially 2 × 15 mg, thereafter 1 × 20 mg). Such therapy was advocated for six months, and after that period only antiplatelet drugs were used. The follow-up ultrasound examination, to evaluate stent patency, was performed after three months and subsequently every six months. In four cases angio-computed tomography examinations were done from 18 to 24 months after angioplasty, which confirmed the patency of all implanted stents and good blood flow (Figs. 1–3). Endovascular therapy should be considered as a good method of treatment for May-Thurner syndrome, which prevents severe disablement in the patient — left leg chronic venous insufficiency with all its consequences. In addition, the development of collateral circulation may lead to pelvic congestion syndrome, which should be treated as well. We observed such a necessity in one patient after left iliac vein recanalisation, in whom wide pelvic veins were closed by embolisation coils during endovascular procedure.

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