OCR2

Healing of diabetic defect in patient with PAD and deficiency in Hageman factor (FXII)

J.Venerova, J. Jirkovska, L. Fialova, S. Solar, M. Zavoral
Charles University in Prague, First Faculty of Medicine, Medical Department of the First Faculty of Medicine and Central Military Hospital, Czech Republic

Background: Incidence of congenital coagulation disorders in diabetic patients with PAD and restenosis after PTA is higher then in cases without PAD. The aim of this casuistry is to demonstrate a process of healing a diabetic defect in patient with critical limb ischaemia and thrombophilia. The Hageman factor (FXII) deficiency is a congenital coagulation disorder causing prolongation of the activated partial thromboplastin time (APTT). As the effect of FXII for coagulation in vivo is actually low its deficiency is not accompanied by bleeding. However, FXII helps activate thrombolysis via inner way, i.e. by means of callicrein, and its deficiency results in decrease of fibrinolysis, thus causing thrombophilia. Due to metabolic changes, the coagulation disorder in diabetes is complex and results also in a higher thrombus production. **Case history:** A male (56) examined for the 1st time with anamnesis of a month-lasting painful defect on the left little toe and identification of Type 2 diabetes. Peripheral pulse in the legs was not palpable while in the left leg (LL) a pronounced erythema in the dorsum of foot and necrotic defect, 0.5 x 0.5 cm in size, on the little toe were observed. The defect was treated with a regular debridement, locally applied antiseptics and ulcer off-loading with half-shoe. TcpO2 values proved severe PAD (2 mmHg). Duplex sonography revealed closure of all crural arteries and the patient was indicated for DSA with attempt for PTA. As a prolonged APTT (150 s) was detected, prior to the intervention haematological examination evidenced the congenital FXII deficiency. PTA was accomplished successfully neither with complications, nor with any special preparation. After the operation we commenced a dual anti-aggregation treatment (clopidogrel and acetylsalicylic acid). TcpO2 check showed improvement in perfusion (20 mmHg). The defect healed up within 6 weeks after the PTA. Haematological check combined with aggregation tests proved a substandard efficiency of the clopidogrel therapy. Thus the medication continued with acetylsalicylic acid only. Discussion: Our casuistry affirms that concurrent incidence of diabetes and thrombophilia enhances the risk of PAD and when selecting a PAD therapy this factor has to be considered. Clearly, good co-ordination with a haematologist is crucial as it enables to perform safely a vascular intervention and to assess a long-term anti-aggregation therapy efficacy. The research project was supported by grant MO 0901-8-8140.