ABSTRACTS OF CURRENT LITERATURE

Heparin-associated thrombocytopenia caused by low-molecular-weight heparin (Heparin-assoziierte Thrombozytopenie durch niedermolekulare Heparine). Koninger et al. Unfallchirurg 98:49–51, 1995.

The authors describe a female patient who died after heparin-associated thrombocytopenia type II developed following low-molecular-weight heparin (LMWH) given perioperatively to prevent thromboembolism. The authors emphasize that this case is in accordance with recent studies showing that low-molecular weight heparin is not suitable for treating patients with heparin-associated thrombocytopenia (HAT) type II.

Intracardiac thrombus formation in heparin-associated thrombocytopenia type II (Intrakardiala Thrombenbildung bei Heparin-assozierter Thrombozytopenie Typ II). Scheffold et al. Dtsch Med Wochenschr 120:519–522, 1995.

The authors describe heparin-associated thrombocytopenia type II occurring in a 77-year-old woman after percutaneous cardiac catheterization. Heparin-dependent antibodies were demonstrated that cross-reacted in vitro with all low-molecular-weight heparins as well as the heparinoid Organon 10172. Heparin administration was discontinued and phenprocoumon administered. The platelet count became normal and the patient declined cardiac surgery. She was discharged from the hospital in satisfactory general condition on maintenance anticoagulant dosage.

Successful use of a heparinoid (danaproid sodium) for heparin-induced thrombocytopenia type II in aortic valve reoperation (Erfolgreiche Anwendung eines Heparinoids [Danaproid-Natrium] wegen Heparin-induzierter Thrombopenie Typ II bei Aortenklappeneroperation). Scheffler et al. Z Kardiol 84: 565–568, 1995.

The authors describe a patient with heparin-induced thrombocytopenia type II after aortic valve reoperation that was successfully performed under anticoagulation with danaproid sodium.

Heparin-induced thrombopenia and thrombosis (Heparininduzierte Thrombopenie und Thrombose). Stockli et al. Schweiz Med Wochenschr 126:483–488, 1996.

The authors describe the case of a 53-year-old patient hospitalized with complicated pelvic fracture. Intravenous infusion of unfractionated heparin (15,000 IU/24 h) was given for thrombosis prevention. After 11 days of treatment, deep venous thrombosis of the left calf developed, complicated 2 days later by a massive bilateral pulmonary embolism. Simultaneously with these thromboembolic events, thrombocytopenia, signs of activated coagulation, and antibodies to heparin were found. The authors used danaproid (Orgaran) and succeeded in the treatment of this patient.

Case report of congenital afibrinogenemia (Fallbeispiel einer kongenitalen Afibrinogenämie). Leeners et al. Klin Pädiatr 207:34–35, 1995.

The authors describe a case of hereditary afibrinogenemia in a female newborn. On the 5th day after birth melena was observed and bleeding from an injection site where 24 hours before blood had been withdrawn. The parents of the newborn are cousins; the father’s fibrinogen was in the lowest range of the norm; two sisters of the father died in their earliest childhood. Coagulation analysis revealed values of PT below 10%, aPTT of more than 120 seconds, and no detectable clottable fibrinogen (Clauss). Besides the observed mild bleeding tendency, no bleeding complications occurred in the newborn during the observation period.

Severe acquired protein S deficiency with thrombophlebitis after febrile infection in a 7-year-old girl (Schwerer erworbener Protein S-Mangel mit Beinvenenthrombose nach fieberhaftem Infekt bei einem 7-jährigen Mädchen). Lutze et al. Klin Pädiatr 207: 113–116, 1995.

The authors describe a 7-year-old girl who suffered from febrile infection with purpura-like bruising and lesions on both thighs. A deep venous thrombosis developed and was treated successfully with urokinase. No
laboratory signs of disseminated intravascular coagulation were found; however, an isolated severe degradation of all protein S components and the presence of a circulating autoantibody to protein S were found. After several months the antibody was no longer detectable, and activity and concentration of protein S were found to be normal. The authors conclude from this observation the significance of protein S defects as a risk factor for venous thromboembolic events.

Recombinant activated factor VII (Novoseven, Novo Nordisk) for hemostasis in acquired factor VIII-inhibitor hemophilia (Rekombinanter aktivierter Faktor VII [Novoseven, Novo Nordisk] zur Blutstillung bei erworbener Hemmkörper-Hämophilie). Meili et al. Schweiz Med Wochenschr 125:405-411, 1995.

Life-threatening hemorrhages were treated with recombinant activated factor VII (rFVIIa) concentrates in a patient with a postpartum acquired inhibitor against factor VIII. Ninety micrograms of rFVIIa per kilogram of body weight were administered as an intravenous bolus injection every 2 to 3.5 hours. The treatment periods were 22.5 days and 11 days in each of the two treatment periods, and hemostasis was promptly achieved and maintained. High doses of activated prothrombin complex concentrate as well as porcine factor VIII concentrates had previously been found to be ineffective. The authors did not observe side effects despite the administration of a high dose and frequent and long-lasting treatment with a total of 234 × 4.8 and 46 × 3.6 mg of rFVIIa. No signs of systemic activation of coagulation were observed. No inhibitor against the patient’s factor VII induced by rFVIIa was detected. Immunosuppressive treatment with cyclophosphamide and prednisone promptly decreased the inhibitor titer.

Local thrombolytic treatment of spontaneous intracerebral hemorrhage with plasminogen activator (rt-PA). Indications and limitations (Die lokale Lysebehandlung spontaner intrazerebraler Blutungen mit Plasminogenaktivator [rt-PA]. Indikation und Grenzen). Schaller et al. Nervenarzt 66:275-281, 1995.

Twenty patients with spontaneous intracerebral hemorrhage (ICH) underwent stereotactic puncture and consecutive local lysis. Administration of rt-PA was performed via a stereotactically placed silicone catheter according to a hematoma size-related formula. The rt-PA dose ranged from 5 to 14 (mean, 8.5) mg. Administration of rt-PA was performed once in one patient, twice in seven, three times in 11, and four times in one patient. On follow-up after a mean of 7.2 months, three patients had died (Glasgow Outcome Score [GOS]I), another patient was GOS II, 10 were GOS III, five were GOS IV, and one had made an excellent recovery (GOS V). The authors conclude that patients who were somnolent or stuporous on admission or who exhibited secondary deterioration of their level of consciousness benefited from the treatment protocol. Comatose patients did not benefit, and they should be treated conservatively.

Therapy of cerebral aneurysms and arteriovenous vascular malformations in hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber disease) (Therapie zerebraler Aneurysmen und arteriovenöser Gefäße malformationen bei der Therapie zerebraler Aneurysmen und arteriovenöser Gefäße malformationen bei der hereditären hämorrhagischen Teleangiektasie (Morus Rendu-Osler-Weber). Helmchen et al. Nervenarzt 66:124-128, 1995.

The authors describe a successful embolization of an arteriovenous fistula and multiple aneurysms of the posterior inferior cerebraller artery (PICA) in a patient with Rendu-Osler-Weber disease after subarachnoid hemorrhage. Two years later, the patient suffered another severe intracranial hemorrhage. Angiography revealed an aneurysm in the same artery (PICA), which spontaneously disappeared within 2 months. The authors conclude that spontaneous regression of aneurysms may occur in Rendu-Osler-Weber disease and that a successful embolization of an arteriovenous fistula may be performed.

Risk of thromboembolism in patients with inflammatory disease (Thrombophiler Status bei Patienten mit entzündlichen Erkrankungen). Lins et al. Dtsch Med Wochenshr 121:855–859, 1996.

Inflammatory reactions are taken to be nonspecific defensive measures of the organism and are associated with complex changes at the cellular and humoral levels. Biochemical markers of coagulation activation, such as prothrombin fragment 1+2 (F 1+2), thrombin-antithrombin III complex (TAT), and fibrin formation (D-dimer), were measured in 130 patients (61 men, 69 women; mean age, 56.9 [20–89] years); 44 had pneumonia, 44 bronchitis, and 42 urinary tract infections. A healthy control group for comparison consisted of 11 men and 15 women (mean age, 48.7 [23–79] years). Levels of F 1+2, TAT, and D-dimer were significantly increased, compared with the controls, in all three patient groups (p <.01). The greatest rises occurred in the patients with pneumonia: F 1+2, median 1.2 vs. 0.6 nmol/L; TAT, 6.2 vs. 2.1 μg/L; D-dimer, 2476 vs. 223 ng/mL. The authors conclude that activation of blood coagulation plays an important role in inflammatory reactions as indicated by measuring sensitive markers of coagulation activation. Especially when these findings are accompanied by an increase of thromboembolism, thrombosis prophylaxis may be important.

Temporary cava filter: Effective prophylaxis of pulmonary embolism in venous thrombosis in the region of the pelvic vascular system and the inferior vena cava? (Temporärer Kava-filter: effektive Prophy-
Recurrent thromboses in a 32-year-old pregnant patient with permanent vena cava filter (Rezidivthrombosen bei einer 32-jährigen Schwangeren mit permanentem Cava-Filter). Kroger et al. Vasa 24:385–388, 1995.

The authors describe the case of a 32-year-old woman who became pregnant 7 years after the implantation of a Kimray-Greenfield filter and gave birth to a healthy child. During pregnancy and after the birth she suffered two new thrombotic events. After the delivery, a completely occluding thrombosis of the inferior vena cava, both common iliac veins, and the right renal vein were revealed by computed tomography and phlebography. Despite these long-term complications, pregnancy is not contraindicated after implantation of a permanent vena cava filter. However, the authors claim that permanent vena cava filters can induce various complications during the remainder of the patient’s life, and patients have to be monitored carefully after implantation of vena cava filters.

Ultrastructural characteristics of cellular reaction to experimental catheter-induced lesions of arterial blood vessels (Ultrastrukturelle Charakteristika der zellulären Reaktion auf experimentelle katheterinduzierte Läsionen im arteriellen Gefäßsystem). Gonschior et al. Vasa 24:325–332, 1995.

The authors investigated cellular alterations immediately after DA using peripheral atherectomy in normal femoral as well as carotid arteries of 30 pigs. DA was used to remove material. The arteries were assigned to two groups according to the depth of vessel injury: group 1, lesions to the intima; group 2, lesions to the media. Sixty-eight arteries with 41 intimal and 27 medial lacerations were excised 4 to 24 hours later and processed for transmission electron microscopy, histology, and immunohistochemistry. Immediately after DA, thrombus formation at the site of the altered segment was found. A transient infiltration of polymorphonuclear leukocytes (PMN) occurred, especially if the media was lacerated, followed by the transformation of contractile smooth muscle cells (SMC) into the synthetic subtype. A marked myeloproliferative response was found in group 2, whereas only moderate tissue hyperplasia was seen in group 1. The authors conclude that cellular alteration of the atherectomized vessel begins immediately after atherectomy. Subsequent to the initial temporary PMN infiltration, an activation of local SMC occurs at a very early stage. These effects and, in particular, a myeloproliferative response were found when lesions injured the internal elastic membrane, whereas only minor effects were seen when the lesion affected the intimal layer.

Complications in HELLP syndrome due to peripartal hemostatic disorder (Kompplikationen beim HELLP-Syndrom infolge einer peripartalen Hämostasestörung). Tanner et al. Zentralbl Gynäkol 118:213–220, 1996.

Forty-three cases of severe pre-eclampsia with HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome were observed in 14,890 deliveries from 1980 to 1993 in the Department of Obstetrics and Gynecology, University of Mainz. In 17 cases there were severe complications (14 cases of renal failure, three of them requiring dialysis). Six patients had bleeding complications: abdominal wall hematoma in five patients after cesarean section, one rupture of the liver. Three cases of pulmonary complications were observed. Nine patients had cerebral complications: seven eclamptic convulsions, one amaurosis caused by partial infarction of the posterior cerebral artery, one venous sinus thrombosis. The plasmatic blood clotting factors were in the normal range except in two cases. TAT complex and D-dimer levels were determined in 27 patients. In 17 of these 27 patients, TAT complex and D-dimer levels were increased (TAT >20 µg/L and D-dimer >1000 µg/L). In 12 of these 17 patients (70%), complications occurred in the course of the disease. On the other hand, only three of 12 patients (30%) with a low activation of blood coagulation (TAT >10 and 20 µg/L; D-dimer >800 and 1000 µg/L) had complications. The authors conclude that the early determination of TAT and D-dimer levels may help to avoid complications.

Spontaneous factor VIII inhibitors (Spontene Faktor-VIII-Inhibitoren). Huhmann et al. Hämostaseologie 16:164–170, 1996.
Spontaneous factor VIII inhibitors ("Hemmkörper-Hämophilie") are antibodies to factor VIII that arise in previously normal persons. The patients have a bleeding tendency similar to hereditary hemophilia A. The pathogenesis is heterogeneous. The aPTT is prolonged and factor VIII activity is usually severely depressed. Most spontaneous factor VIII inhibitors are type II inhibitors (titers can be quantitated by the Bethesda method). Control of acute bleeding can be achieved by treatment with high-dose human factor VIII concentrates, porcine factor VIII, activated prothrombin complex concentrates, or recombinant activated factor VII. The inhibitor titer can be rapidly reduced by extensive plasmapheresis or immune adsorption with protein A Sepharose or Therasorb. Long-term reduction or elimination of the inhibitor can be achieved by immunosuppressive treatment.—EW