IDIOPATHIC INTRACRANIAL HYPERTENSION: ASSOCIATIONS WITH COAGULATION DISORDERS AND POLYCYSTIC OVARY SYNDROME.

Our specific aim was to determine if there is an association between idiopathic intracranial hypertension (IIH), polycystic ovary syndrome (PCOS) and coagulation disorders. Thrombophilic hyperestrogenemia of pregnancy, exogenous estrogens, obesity, and hypofibrinolysis of PCOS have been associated with IIH.

PCR coagulation measures were done in 109 healthy white female controls and serologic studies were done in 44 of these adult females. Fifty-two consecutively referred white women with IIH underwent the same PCR and serologic studies. Of the 52 women, 31 (60%) had PCOS, 43 (83%) were obese with BMI >30 kg/m², 15 (29%) were extremely obese (BMI ≥ 40). Of the 52 women, 32% (16/50) were homozygous for the thrombophilic C677T mutation of the MTHFR gene vs 9% (10/109) controls (p = .0003). Thrombophilic Russel’s Viper Venom time (DRVVT) was prolonged (>38.4 sec) in 40% (21/52 cases) vs 9% (4/45) controls (p = .0004); the thrombophilic activated partial thromboplastin time was prolonged (>31.5 sec) in 19% (10/52) cases vs 4% (2/54) controls (Fisher’s p [p_f] = .014). Hypofibrinolytic plasminogen activator inhibitor activity (PAI-Fx) was >21.1 U/ml in 23% (11/47) cases vs 8% (3/37) controls (p = .062). Fasting serum insulin and BMI correlated with PAI-Fx (r= 0.60, p<.0001; r= .33, p=.023). Glucophage, which lowers insulin, PAI-Fx, and BMI, also ameliorates PTC, particularly in women with concurrent PCOS. In a subgroup of 13 women from a single Cincinnati practice, 4 (31%) had thrombophilic high Factor VIII (>150%) vs 0/21 controls (p = .015); 5 (38%) had long DRVVT (>38.4 sec) vs 4/45 controls (9%) (p = .021). IIH first occurred during pregnancy in 10% of the 52 women, with oral contraceptives in 37%, with hormone replacement therapy in 10%, with Depo-Provera in 4%, and with minocycline in 4%. PCOS, perhaps through its association with morbid obesity, high insulin, and high PAI-Fx, and, separately, thrombophilic and hypofibrinolytic coagulation disorders, often augmented by thrombophilic exogenous estrogens or pregnancy, appear, speculatively, to be reversible etiologies of IIH.