Cerebral venous thrombosis in paroxysmal nocturnal haemoglobinuria

Paroxysmal nocturnal haemoglobinuria (PNH) is a rare acquired disease of the haematopoietic system manifested by chronic haemolytic anaemia, leukopenia, and thrombocytopenia. Patients with PNH have an increased risk of developing systemic venous thrombosis, and obstruction of the hepatic veins, or Budd-Chiari syndrome (BCS) which is a highly fatal complication in about 30% of cases. PNH is also a well-established though extremely uncommon cause of cerebral venous thrombosis (CVT).1-3 We describe a patient with PNH and BCS in whom CVT was angiographically documented. We comment upon the risks of anticoagulants in this unusual situation and the need to remember PNH among the possible causes of CVT.

A 34-year-old woman was admitted to hospital because of malaise, low-grade fever, pleural effusion, hepatomegaly and ascites. The diagnosis of PNH was established by the presence of haemosiderinuria, positive Ham acid haemolysis and sucrose lysis tests. Hepatic venogram confirmed the existence of BCS. She received repeated blood transfusions and a year later a portocava shunt was performed. Three years later she complained of intense frontal-occipital pain and clumsiness of the left limbs. Examination showed a mild distal paraparesis and edema leg with only minimal slowness of alternating movements in the left arm. There was impairment of position and pain perception with sensory extinction on the left side. The optic discs were slightly blurred. The visual fields were full with a moderately enlarged blind spot on both sides. CT scanning showed a right parietal infarction. Cerebral angiography revealed thrombosis of superior sagittal sinus, straight sinus, lateral sinuses, and internal cerebral veins. The patient made a complete recovery with dexamethasone and coumarin therapy.

In a comprehensive review of 38 cases, Bousset et al4 failed to list PNH among the possible causes of CVT. PNH is commonly undiagnosed for a period of 1-3 months, and its true incidence among patients with CVT is probably underestimated because detailed coagulation studies are not performed in most cases. This may be of the utmost importance when considering treatment for such patients with PNH.4-6 and thus might be deleterious in this particularly difficult situation.

PNH should be considered among other possible causes of CVT and ruled out by the appropriate laboratory investigations, especially when treatment with heparin is contemplated.


Toxic reaction following the combined administration of fluoxetine and phenytoin: two case reports

Fluoxetine is a new antidepressant agent unrelated to the tricyclic antidepressants, whose structure corresponds to a straight chain phenylpropylamine. The drug selectively inhibits reuptake of serotonin but not noradrenaline and has a minimal muscarinic, dopaminergic, histaminergic or serotoninergic effect. The only described interaction is with L-tryptophan which enhances its therapeutic effects, but produces symptoms and signs of intoxication.7 Presently there is no published work on the possible interaction between fluoxetine and anticonvulsant drugs.

We describe two patients, who developed symptoms and signs of intoxication with phenytoin a few days after initiating the use of fluoxetine.

An 84-year-old woman was treated with phenytoin 300 mg daily, after removal of a chronic subdural haematoma. Two months later she developed a depressive syndrome. CT showed no alteration, and the plasma level of phenytoin was 15 μg/ml. Treatment with a dose of 20 mg/day of fluoxetine was given, increasing the dose to 400 mg/day after 10 days. Five days after the beginning of treatment she developed gait ataxia, vertigo, diplopia and alteration of consciousness. Examination also showed dysmetria of the limbs, multidirectional nystagmus, and alteration of judgement with visual hallucinations.

The plasma level of phenytoin was 35 μg/ml. The dose of fluoxetine was reduced gradually and there was progressive recovery from the signs and symptoms.
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