Correspondence

Polycythemia causing Cerebral Venous Thrombosis-A Case Series

Priyanka V Kashyap, Sunil Jee Bhat

1Assistant Professor (Neurology), 2Senior Resident (Internal Medicine), Shri Mahant Indiresh Hospital, Dehradun, Uttarakhand

Sir,

Cerebral venous thrombosis (CVT) is a rare but serious complication of polycythemia causing death in 8.3% patients. Various studies suggest that polycythemia can present in a variety of ways but specific initial presentation of smoker’s polycythemia with CVT is rare.

Smoker’s polycythemia results due to increased red cell mass and reduced plasma volume due to hypoxia. A correlation of polycythemia secondary to high altitude is also seen.

We aimed to determine the association between smoking induced polycythemia and CVT and ruling out common coagulation disorder as etiology.

A retrospective study was done on 100 headache patients of high altitude Uttarakhand. Sixty six patients had migraine and 34 patients had Tension Type headache.

Out of 100, 30 had (CVT) on MRI brain and venogram. Superior sagittal sinus (SSS) was most common involved (78%) followed by Transverse and Sigmoid sinuses (22%). Among them 26 were smokers. Hemoglobin levels ranged 18-26 gm% and Hematocrit levels were 56-60% in these 30 patients higher than rest 70 patients of headache who did not had CVT.

Secondary polycythemia was common in CVT group probably secondary to smoking and high altitude. Four non smokers patients who had CVT, hypoxia induced hemoconcentration may be the cause.

Ten of 30 patients had headache and papilloedema consistent with Benign intracranial hypertension (BIH) and all had thrombosis on MRV.

Upto 25% BIH patients have CVT, with female predominance (1.3:1) as also proved in our series probably reflecting oral contraceptive use.

Smoking creates chronic hypoxia, leading to excess production of erythrocytes from bone marrow, leucocytosis as a result of elevated carboxy Hb and plasma volume reduction, causing stasis and hyperviscosity leading to thrombosis.

Smoker’s polycythemia is best diagnosed by a left shifted O2 dissociation curve (suggesting hypoxia) which was not done in our series due to technical constraints.

Treatment of smoker’s polycythemia is directed towards acute treatment of the disease and cessation of smoking as long term benefit. In previous studies, smoking cessation resulted in significantly decreased hematocrit and hemoglobin levels resulting in clinical and radiologic improvement as seen in our series.

Smoker’s polycythemia is an important entity causing CVT in patient cohort of Uttarakhand, high altitude causing chronic hypoxia multiplies the risk of polycythemia in these patients. This emphasizes role of smoking cessation in treating CVT in smoker’s polycythemia.

This case series conclude CVT a common but rarely correlated presentation of smoker’s polycythemia.

References