Congenital cyanotic heart disease

Cyanotic congenital heart disease (CCHD) develop secondary erythrocytosis with elevated hematocrit and low Po2 that provides an hypoxemia suitable for abscess proliferation.

Cyanotic heart disease accounts for 12.8–69.4% of all cases of brain abscesses with identified risk factors in several series, with the incidence being higher in children.

Brain abscesses can especially with Tetralogy of Fallot and pulmonary anterior venous fistula with large right-to-left shunt.

The pathophysiology of secondary erythrocytosis associated with CCHD is different from primary erythrocytosis associated with polycythemia vera (PV). In PV, the serum erythropoietin level is usually low or normal,1 and the erythroid progenitors are capable of proliferation in vitro in the absence of erythropoietin.

Conversely, secondary erythrocytosis associated with CCHD is a physiological response to tissue hypoxia with resultant increase in serum erythropoietin level, thereby stimulating the bone marrow erythropoiesis causing an elevated red cell mass, hematocrit, and whole blood viscosity.