

Antiphospholipid syndrome is characterized by arterial and venous thrombosis, recurrent pregnancy loss, and intrauterine growth restriction (IUGR). It is associated with antiphospholipid antibodies, which are subdivided to anti-cardiolipin antibodies, lupus anticoagulant antibodies, and antibodies against proteins. Anti-2GPI is the best known antibody against proteins. 2GPI binds to negatively charged phospholipids, like cardiolipin, phosphatidylserine, and phosphatidylinositol. Antiphospholipid antibodies influence several processes, such as hemostasis and cell and complement activation. Complement is the main mediator of tissue injury in the placenta. Initially, the classical pathway of complement activation is induced via C1, then, at the level of C3, the alternative pathway joins and amplifies the damage. It has been shown that interactions between complement anaphylatoxin C5a and its receptors play a key role in causing injury. The murine model has shown that by inhibition of the complement pathway at this level, pregnancies can be rescued. The etiology of placental injury involves three important molecules; these molecules first initiate a proinflammatory state which then allows subsequent induction of the prothrombotic state. The prothrombotic state is the best known complication of antiphospholipid syndrome. The molecules involved are TNF-, tissue factor (TF), and sVEGFR-1. Additionally, complement itself is also capable of inducing thrombophilia. Although increased complement activation is not unusual during normal pregnancies, it is extremely elevated in the presence of antiphospholipid antibodies. Even in pregnancies with no clinical signs of injury, the presence of antiphospholipid antibodies still leads to inflammation and nonspecific inflammatory changes at the placental level.

Today pregnancies can be protected with heparin and aspirin. New studies indicate that treatment with statins and ACE inhibitors can also be effective. In the future, therapies targeted toward complement-induced processes in the placenta will play an increasingly significant role in the treatment of this syndrome.