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# THROMBOTIC COMPLICATIONS IN ACUTE CYTOMEGALOVIRUS INFECTION IN IMMUNOCOMPETENT INDIVIDUALS.

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Human Cytomegalovirus (CMV), a member of the Herpesviridae family, is a widespread pathogen with seroprevalence varying from 40% to 100% depending on demographic and geographic factors. In immunocompetent individuals, CMV infection is often asymptomatic or results in a mild, self-limiting mononucleosis-like syndrome. Although they may be found throughout the body, CMV infections are frequently associated with the salivary glands. Symptoms include fever, pharyngitis, malaise, lymphadenopathy, mild liver dysfunction, and lymphocytosis with atypical lymphocytes. Rare and severe manifestations include prolonged fever, arthralgia, gastrointestinal and neurological involvement, and hematological disorders.

While CMV is typically considered a threat primarily to immunocompromised patients, recent evidence suggests that it might also pose risks to immunocompetent individuals, particularly leading to thrombotic events. Thrombosis has arisen as a potential complication of acute CMV infection, leading to a renewed interest in this association in medical research. It is not generally known that acute CMV infection could lead to arterial or venous thromboembolism

Atzmony et al. (2010) retrospective study consisted of 140 hospitalized patients with acute CMV infection and 140 matched controls. A 6.4% incidence of thrombosis among the CMV- infected group, with 3.6% experiencing arterial thrombosis and 2.9% venous thrombosis was found. Notably, the true incidence may be higher due to the limited use of diagnostic imaging methods. Paran et al. (2013) evaluated 6205 patients six months post-CMV infection and compared them to over 84,000 controls. Their results were a venous thrombosis incidence rate of 3.06 per 1000 individuals- likely another underestimate.

Several theories suggest that CMV infects endothelial cells and enhances adhesion molecule and tissue factor expression, thereby triggering platelet adhesion and aggregation on vessel walls, factor X activation, and thrombin formation. Another suggests that CMV increases levels of Von-Willebrand factor and factor VIII (T. H. The et al.). According to Schimanski *et al* (2011)., who prospectively studied acute CMV infection incidence as well as factor VIII plasma levels among 166 hospitalized venous thrombosis patients, 3 out of 7 (42.9%) patients with venous thrombosis and acute CMV infection also had high factor VIII plasma levels. Lastly, the most accepted theory indicates that acute CMV infection is linked to transient appearance of thrombogenic anti-phospholipid antibodies. In Schimanski's study, 14.3% of CMV-infected thrombosis patients tested positive for anti-phospholipid antibodies.

Acute cytomegalovirus infection, though usually considered benign in immunocompetent individuals, may have underappreciated clinical consequences—particularly the risk of thrombosis. The evidence from multiple studies and case reports supports a non-negligible association between CMV and thrombotic events, even among otherwise healthy individuals.