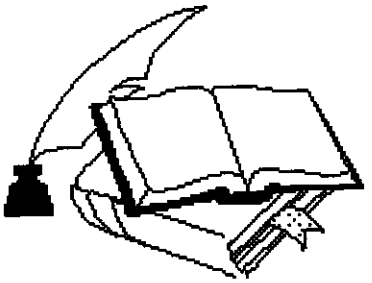
# INTRODUCTION



# INTRODUCTION

Many viruses do circulate in peripheral blood at some stage of disease, but the primary pathology related to infection is in the target organ or cells. Other viruses preferentially infect the blood or lymph glands causing disturbance in the blood components including production, morphology, maturation, and function. Lymphocytes are the target cells of many viruses causing lymphocytosis such as Epstein-Barr virus (EBV), cytomegalovirus (CMV), herpes simplex type II, and varicella zoster virus. Atypical lymphocytes may be associated with lymphocytosis. These clinical conditions are called mononucleosis syndromes (Cantani and Mastrantoni, 1989).

Other viruses may cause lymphocytopenia such as human immunodeficiency virus (HIV) and influenza virus. Acquired immunodeficiency syndrome (AIDS) is caused by (HIV) that infect helper/inducer T lymphocytes and destroy the immune system (Coffin, 1987).

Many viruses cause erythrocyte disorders. Red cell aplastic crisis have been observed in patients infected with viral hepatitis, B19 parvovirus (Sears *et al.*, 1975; Kurtzmam *et al.*, 1987).

A number of viral agents including measles, cytomegalovirus, varicella, herpes simplex, influenza A, Epstein-Barr virus, HIV and

Coxackie viruses have been associated with hemolytic anemia (Heruvitiz *et al.*, 1984).

Certain viral infections may cause neutrophil disorders e.g. Epstein-Barr, infectious hepatitis, Kawasaki disease and HIV cause severe protracted neutropenia, even pancytopenia (Murphy *et al.*, 1987). Also virus associated hemophagocytosis syndrome (VAHS) is a syndrome of exaggerated histiocytic proliferation and activation. Epstein-Barr virus, herpes simplex, cytomegalovirus, varicella zoster and adenovirus have been implicated (Mc Clain, 1986). Some virus infection has been associated with bone marrow aplasia, e.g. hepatitis virus, Epstein-Barr virus, Rubella and HIV (Young and Mortimer, 1984).

As regards oncogenesis, some viruses have been strongly associated with human hematologic malignancies. HTLV-H is isolated from patients with adult T-cell leukemia/lymphoma (Hinuma *et al.*, 1982). HTLV-II was isolated from patients with a T-cell variant of hairy cell leukemia (Rosenblatt *et al.*, 1986).

HIV is also accompanied by secondary neoplasias such as B-cell lymphoma. EBV was detected in tumour cells from patients with Burkitt's lymphoma (Tosato and Blease, 1985).

Infection caused by certain viruses are occasionally associated with evidence of hemostatic impairment such as EBV, rubella, varicella and HIV (Cosgriff, 1989). A small number of viruses produce prominent alteration in hemostasis and so much so that hemorrhage is a hallmark of

these infections, these are the hemorrhagic fever viruses (Johnson and Monath, 1982). There are several mechanisms by which the viruses cause hemostatic abnormalities including thrombocytopenia, platelet dysfunction, reduced levels of coagulation factors, disseminated intravascular coagulation (DIC) and vascular injury (Cosgriff, 1989).

Acute infection with B19 parvovirus produces transient aplastic crisis in the patient with hemolysis and pure red cell aplasia in the immunologically incompetent host. The target cell of the virus is the marrow erythroid progenitor. Thrombocytopenic purpura after vaccination with measles, mumps and rubella has occasionally been reported. Vaccine-associated thrombocytopenic purpura appears to be similar to that occurring after natural measles or rubella infections and is not distinguishable from acute childhood idiopathic thrombocytopenic purpura not associated with vaccination (Hessel *et al.*, 1996).

Measles virus, human immunodeficiency virus, Epstein-Barr virus and other leukotropic viruses can modulate the expression of leukocyte function antigen-I (LFA-I) on the surface of infected and nearby leukocytes. This ability to induce changes in (LFA-I) expression may play an important role in the pathogenesis of these viruses (Nagendra *et al.*, 1996). Measles virus modulation of (LFA-I) expression on leukocytes may be an important step in measles virus pathogenesis (Rossen *et al.*, 1993).