INTRODUCTION

Antiphospholipid Antibodies (APAs), namely; lupus anticoagulant & anticardiolipin antibodies are a family of closely related immunoglobulins (IgG, IgM, IgA, or mixture) that interact with negatively charged phospholipid (Triplett and Brandt 1988 - McNeil et al., 1991).

Lupus anticoagulant and anticardiolipin are acquired autoantibodies that are characteristically found in patients with systemic lupus erythematosus and related autoimmune diseases. (Feinstin & Rapaport 1972 – Shapiro & Thiagarajan 1982 – Derksen & Kater 1985- Harris et al. 1986, 1988).

About 10% of patient with systemic lupus harbor lupus anticoagulant (LA), that is an immunoglobulin that has the ability to prolong phospholipid-dependent coagulation tests.

The LA is commonly seen in other conditions, including malignancy, lymphoproliferative disorders & viral infections especially HIV infection. (Criel et al 1978 - Coller et al 1981 - Lefrere et al 1988 – Tailan et al 1989 - Kunkel 1992).

It was noticed that patients with this type of anticoagulant did not bleed & in contrarly they are at risk of venous & arterial thromboembolic events (Triplett & Brandet 1988). Patients with APAs are prone to repeated episodes of both arterial & venous thrombosis, Cardiomyopathy, thrombocytopenia, fetal loss, renal failure, neurological manifestations & many skin lesions (Asherson & Cervera, 1993).

The primary antiphospholipid antibodies syndrome refers to the presence of the above clinical manifestations without evidence of an associated autoimmune disorders (Fong & Boey, 1992).

However, the great majority of persons who have either the anticardiolipin thrombosis Antiphospholipid syndrome or lupus anticoagulant thrombosis antiphospholipid syndrome are otherwise healthy & harbor no other underlying medical conditions & are classified as having the primary rather than Secondary antiphospholipid thrombosis Syndrome (Bick & Backer 1992 - Bick 1992 - Kunkel 1992) APAs can occur secondary to many pathological conditions as in; connective tissue diseases (systemic lupus erythematosus SLE, Rheumatoid Arthritis) and in infectious diseases (Acquired immunodeficiency syndrome, infectious mononucleosis & in chronic infection as leprosy) as well as drug-induced Pansidar, Hydralazine, Streptomycin, Quindine & Procainamide. (Smolarski, 1980 - Asherson et al 1985, 1989 - Fauci 1985 - Gastineau et al 1985 - Jeffrey 1986 - Canoso et al, 1987 - Harrison 1987 - Fort et al 1987 - Misra et al 1987 - Keane et al 1987 - Li et 1988 - Jones 1991-, Shortell et al, 1992), stated that in comparison with the population having athersoclerosis patients with arterial manifestations of APL syndrome were more likely to be women, younger, did not smoke and had a higher percentage of upper extremity involvement

Robin et al, 1997 reported that patients with APL and stroke/TIA are younger than the general stroke population.

Young patients with stroke in association with APL seen to have a more frequent history of recurrent episodes tnan similar aged patients with stroke who do not have these antibodies (Levin et al, 1997).

AIM OF THE WORK

Our study aim to Shed light on the prevalence of antiphospholipid antibodies among patients with cerebral ischemia.