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## **A CASE STUDY OF SYSTEMIC LUPUS ERYTHEMATOSUS AND ITS COMPLICATIONS**

### ***Introduction***

Systemic lupus erythematosus (SLE) is a autoimmune inflammatory multisystem disorder. Arthralgia and rashes are the most common clinical features while most serious complications associated with SLE are cerebral disease and renal disease. The rate of SLE varies between countries from 20 to 70 per 100 000. Women of childbearing age affected about nine times more than men and children. Peak age is from 20–30 years [2].

### ***Goal***

The study explore and understand the onset of SLE , co-existence of other diseases and the complications.

### ***Materials and Method of research***

The case study which include clinical features, anamnesis, laboratory investigations and comparison with the standard diagnostic criteria.

### ***Research, Results and discussion***

A 40 years old female bank manager (Colombo, Sri Lanka) with family history of rheumatoid arthritis, in 2006 presented with extreme fatigue, heavy hair loss that did not respond to vitamin intake, loss of weight, periorbital swelling, limp, hip pain, finger and toes become pale and blue when exposed to cold. She experienced an early stage miscarriage in January 2010 and she was with complains due to irregular menstrual bleeding over 6 months. In laboratory investigations, U1 RNP antibody tested positive for the first time, rheumatoid factor, Anti double stranded DNA, Jo-1 antibody were negative, Antinuclear antibody(ANA) positive (1/640), lupus anticoagulant was high (44.00), low complement C3 level (51mg/dl) and complement C4 level (7.00 mg/dl),serum glutamate pyruvate transaminase (S.G.P.T) was increased (36.00 U/L) while serum creatinine and eGFR estimated average. Skin biopsy did not provide evidence about discoid lupus erythematosus. Blood picture, red blood cells(RBC) were normocytic, normochromic and mild Rouleaux formation, white blood cells(WBC) illustrated as leukopenia, neutropenia, reactive lymphocytes not seen, and platelet (PLT) level was normal. C.C.P antibody was tested negative (2.1 U/ml).

In 2011,ANA titer was 1:5120, according to Dexa scan z score was -2.3 with high risk for fractures, Anti smith antibody was equivocal, normal microbiology urine report, Serum creatinine level decreased (0.3 mg/dl), serum protein total decreased (4.9 g/dl), serum albumin decreased (2.0 g/dl). In renal profile, low serum uric acid (3.30 mg/dl),serum calcium (7.8 mg/dl),serum sodium (133 mmol/l), C-reactive protein (CRP) was high (1.4 mg/dl),increased CA 125 (110U/ml), normal random blood glucose level, high level of D. Dimer (0.6 mg/l). According to duodenal biopsy, colonic non specific ileitis were reported, sub conjunctival fluid in both eyes were reported. Examination in department of clinic neurophysiology (NHSL, Colombo), mentioned that absence of features of myopathy or myositis. Cardiolipin antibody (IgM) less than 6.3,cardiolipin antibody (IgG) 92.3 were reported and 12 weeks apart the test was repeated and cardiolipin (IgM) 7.2 and

cardiolipin antibody(IgG) 47.7. In ultrasound (US) guided FNAC, focal lesion in right lobe of thyroid, follicular proliferation and adenomatoid nodule were identified. Internal right jugular vein thrombosis appeared and started treatment with Warfarin.

In 2012, from US scan of abdomen, ascites, right sided pleural effusion and atelectasis and hyperechoic lesion in segment 4 of the liver were diagnosed. US scan of chest reported the pleural effusion was more likely pus and later confirmed mycobacterium tuberculosis(TB). She was diagnosed with extra pulmonary TB, treated with anti TB drugs for 6 months and confirmed absence of TB in fluid. Decreased serum sodium(132mmol/l), decreased chloride (93 mmol/l), normal serum creatinine level, increased serum glutamic oxaloacetic transaminase test (S.G.O.T) (84 U/l), high serum cortisol level (607 nmol/l) and continued to report low level of albumin.

In 2013, C reactive protein increased (13.4 mg/l), high S.G.O.T (62U/l) and S.G.P.T (38 U/l), decreased serum calcium (7.3 mg/l) were indicated. Both CMV IgM and CMV IgG were positive. Epstein-Barr virus and Hepatitis C antibody were tested negative. Bone marrow (BM) biopsy illustrate, presence of mild hypocellular BM with active hemopoiesis and reactive changes, mild leukopenia probably due to hydrochloroquine, anti TB drugs and autoimmune nature of disease, moderate hypochromic microcytic anemia. 2014, she experienced hypoglycemic coma for 4 to 5 days. Normal serum creatine level indicated. Increased level of lactate dehydrogenase (LDH) (800 U/l) and C reactive protein (35.3 mg/l) were observed. Decreased serum iron (17.4 µg/dl) and transferrin saturation (4.7 %). US scan of abdomen done in 2015, illustrate the presence of hepatomegaly and splenomegaly. In 2016, blood picture reported, RBC normocytic normochromic and hyperchromic with Roulex formation, WBC were leukopenia with marked lymphopenia and mild neutropenia and suspected with anemia of chronic disease or drug induced cytopenia. Decreased serum iron(18.6 µg/dl), total iron binding capacity (241.5 µg/dl), transferrin saturation 7.7 % were reported. Normal foetal hemoglobin level was identified. In 2017, C reactive protein increased (27.5 mg/l), serum creatine decreased (0.5 mg/dl). Blood picture illustrated, RBC mild hypochromic microcytic with moderate Roulex formation predominant neutrophils.

In 2021, presented with complaints of dry cough. In pulmonary function test, thickening of lungs and airways were reported. She was prescribed with Seretide 250 IU inhaler. 2022, intracranial hemorrhage occurred with sudden fits and unconscious. Absence of memory and hospitalized for more than 2 months, stopped Warfarin. Persistent low PLT were reported. In 2023, experienced with left patella fracture, dry gangrene in toe observed. Although the PLT count is 126 000, Warfarin started. Blood picture reported, mild hypochromic anemia, mild iron deficiency anemia and moderate thrombocytopenia. Decreased TSH level (0.327 µIU/ml) were identified. Increased activated partial thromboplastin time (42.2s) and ESR (40 mm) observed. Bleeding and clotting time test, eGFR, C reactive protein, liver profile, urinalysis were without significant changes from norm.

Based on a study it was proven that Anti U1 RNP is not a must to fit into a diagnostic criteria [1], therefore this explained the possibility of tested negative Anti U1 RNP, initially. Digitals appeared to be pale in the exposure of cold, which illustrate the Raynaud's phenomenon. According to laboratory investigation, presence of leukopenia, anemia, persistent low PLT, increased ESR, normal C reactive protein and persistent low serum albumin, increased titer of ANA antibody (1:5120), equivocal reported in anti-smith test and decreased level in complement C3 and C4 indicated that she is presented with SLE. According to diagnostic criteria for antiphospholipid syndrome[3], she fulfilled clinical criteria, with presence of internal right jugular vein thrombosis and miscarriage in 2010, presence of high titer anticardiolipin antibody(IgM and IgG) and high level of lupus anticoagulant completed the laboratory criteria which confirm the diagnosis of anti-phospholipid syndrome. Co-existing health conditions increase the risk of treatment in intracerebral hemorrhage, this will lead to cause temporary

memory loss. Family history of rheumatoid arthritis more likely to be the cause of her connective tissue disease [4]. Although she required aggressive treatment with immunosuppressive, she has already experienced critical side effects of long term therapy of corticosteroids which was implicated by increased cortisol level that caused adrenal crisis and drug induced osteoporosis.

### **Conclusion**

40 year old female with clinical features and evidence of laboratory investigation confirm the diagnosis of SLE. Antiphospholipid syndrome coexisted with SLE. The onset of autoimmune connective tissue disorder due to the presence of family history with rheumatoid arthritis. She experienced complications from the disease and long term therapy of aggressive treatment.

### **LITERATURE**

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## **INSTRUMENTAL AND LABORATORY CHANGES IN POST-MYOCARDIAL INFARCTION**

### **Introduction**

Myocardial infarction and the following sequelae remains the most frequently reported cause of death globally. Assessment of the early post-MI changes is of huge impact on the further therapy plan and the prognosis of MI victims. One of the early specific markers of myocardial infarction is the elevation of cardiac lactate dehydrogenase 1 and troponins, including troponins I and T. However, systemic changes are rarely assessed in early post-MI patients [1]. The role of these changes is unclear in determining the prognosis of patients with MI and the potential risk of developing complications such as renal failure. In the current retrospective analysis, we covered the potential role of these systemic and the regularly checked cardiac markers in the prognosis determination and prognosis of the patients.

### **Goal**

The study sought to assess the laboratory and instrumental changes in patients with myocardial infarction.

### **Material and Methods of research**

A retrospective analysis included 154 patients with myocardial infarction for the period 2014–2019. The study analyzed 76 parameters including indicators of post MI complications associated laboratory and instrumental changes. The presented t-test results are all statistically