A Case Study on the Correlation Between Systemic Lupus Erythematosus and Tuberculosis

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Abstract- The incidence of Mycobacterium tuberculosis infections among Systemic Lupus Erythematosus (SLE) patients is commonly observed due to the immunocompromised state of SLE patients [1]. Recent studies show that a history of tuberculous infections play a key role in the induction and exacerbation of SLE in genetically susceptible hosts thus acting as an immunomodulatory agent [2]. We report a case of a female patient with history of tuberculosis with an incidental diagnosis of SLE. The symptoms of SLE worsened after completing a six month course of Anti Tubercular Therapy (ATT) with standard drugs on par with RNTCP DOTS guidelines.

Index Terms- Systemic Lupus Erythematosus; Tuberculosis; Koch's abdomen; Pleural effusion

I. Introduction

Tuberculosis is an infectious disease caused by acid fast bacilli, Mycobacterium tuberculosis and is typically transmitted via airborne droplet nuclei. The incidence of TB is a grave concern for the world today as approximately one quarter of the population is currently infected with latent TB [3]. Whilst affecting worldwide, tuberculosis tends to have a greater prevalence in developing countries due to community health problems and economic constraints [3]. SLE is an autoimmune disease having multiple organ system involvement. The pathophysiology of SLE is production of autoantibodies by the body's immune system against self-antigens. Renal failure, cardiovascular failure and infections due to immunodeficiency are major causes for mortality among cases of SLE [4]. Most common infections are caused by Gram positive and Gram negative organisms along with Mycobacterium tuberculosis infections among many other opportunistic infections [5].

There is ample evidence suggesting that cases of SLE are highly susceptible to tuberculous infections, mostly due to the hyperactivity of the immune system and high doses of immunosuppressive therapy, both aetiologies leading to an immunocompromised state[2]. Resistance to M tb mediated by cellular immunity is dysfunctional in patients with SLE [2].

II. Case Report

A 26 year old female patient from Eastern India (Cuttack, West Bengal) presented with menstrual irregularities in April, 2019. On enquiring about her medical history, she was diagnosed with Systemic Lupus Erythematosus simultaneously with abdominal tuberculosis in late 2016. She had completed a course of ATT for 6 months and has been taking corticosteroids for SLE since diagnosis. The patient is also a known case of hyperthyroidism since the age of 16 and is on medication for the same.

The patient gives history of an abortion 1.5 months after marriage at the age of 20. She has been anxious to conceive ever since the abortion. 4 years after the abortion, she developed symptoms of cough with expectoration, evening rise in temperature, night sweats and occasional abdominal pain. She also had presented with bilateral pitting pedal oedema, bilateral pleural effusion and mild ascites. Her ultrasound scans of the abdomen and pelvis were congruent with findings of Koch's abdomen (Thickened omentum with mesenteric and peripancreatic lymphadenopathy) . Chest X rays had findings of pleural effusion (Left lower lobe infiltrates along with bilaterally blunted costo-diaphragmatic angles). She gave history of her mother being diagnosed with pulmonary tuberculosis about 15 years ago. Other test results included raised erythrocyte sedimentation rates, strongly positive anti-nuclear antibodies, low serum albumin levels (2.2g/dl) and low albumin: globulin ratio (0.51). Tests for SLE were done out of suspicion due to her multi organ involvement despite the patient not presenting with any evident symptoms of SLE. She was immediately started on ATT, prednisolone 10mg BD and hydoxychloroquine 200mg BD; the latter two drugs being therapeutic for SLE, though she was asymptomatic for SLE. The ultrasound findings of Koch's abdomen were recurring on repeat scans for about 4-6 months after which her ultrasound scans were normal along with subsiding symptoms of tuberculosis. Repeat chest X ray also showed no evidence of pleural effusion or pulmonary infection. Yet, the patient's ESR oscillated between 60mm/hr and 115mm/hr strongly indicative of an ongoing inflammatory process.

In early April, 2019, she presented to the department of Obstetrics and Gynaecology with history amenorrhoea of 3 months and spotting with abdominal pain since 1 week. Serial beta Human Chorionic Gonadotropin values were monitored and pregnancy was ruled out, supported by no evidence of a foetus on ultrasound scans. On admission, she presented with abdominal pain, orthopnoea, bilateral pitting pedal oedema, breathlessness at rest, chest pain, joint pain(mostly small joints- fingers), pallor, crepitations in her lower lung fields, history of gradual weight loss over 2 years and passage of worms in stools. On running some tests, serum Amylase and Lipase were low indicative of pancreatitis. The patient was strongly positive for anti-nuclear

antibodies and anti dsDNA antibodies. Anti-Smith antibodies were negative. Liver function tests showed low total protein (5.2g/dl), low serum albumin levels (2g/dl) and low albumin: globulin ratio (0.6). Ultrasound scans of abdomen and pelvis showed bulky pancreas, peri- pancreatic lymphadenopathy and moderate ascites which puts abdominal TB as one of the aetiologies. Chest X rays showed evidence of bilateral pleural effusion. She was continued on treatment with parenteral prednisolone (40mg intravenous OD) and 200mg hydroxychloroquine. There was no evidence of Lupus nephritis nor was there evidence of involvement of the cardiovascular system.

III. Discussion

Infections are a major cause of morbidity and mortality among patients with SLE [6], mostly due to their immunocompromised state. A study from northern India showed that 2.6% of 309 lupus patients had multiple infections. Approximately 25% had infections and tuberculosis was the most common infection in their study [7]. In this case, the patient was first diagnosed with abdominal tuberculosis and SLE was diagnosed incidentally. The patient later developed symptoms of SLE which gradually worsened over the years though she was already on medication for SLE. There are studies that show that tuberculous infections precipitate the onset of SLE in genetically predisposed individuals [8], [9], [10], [11], especially in endemic areas. There are other studies that show that tuberculous infections are responsible for a flare of SLE symptoms [12]. In this patient, we suspect that tuberculosis precipitated the onset of SLE and also it caused an acute flare in the symptoms of SLE. Over a period of 6 years, we suspect that the patient's medications for SLE have brought upon an immunocompromised state in the patient. This immunocompromised state could have caused a reactivation of tuberculous organisms and may be the reason for the recurrence of abdominal TB which could also be causing an acute exacerbation of symptoms of SLE in this patient. Findings of pancreatitis in the patient is suspected to be a result of usage of steroids in the patient or as a complication of gastrointestinal tuberculosis. Ironically, steroids are the most effective treatment for pancreatitis. Findings of bilateral pitting pedal oedema could be caused due to protein losing enteropathy, also a common complication of gastrointestinal tuberculosis. Reappearance of mild ascites and bilateral pleural effusion could be linked to SLE and to protein losing enteropathy. Nephrotic syndrome and liver failure can be ruled out as causes for ascites, pleural effusion and pedal oedema due to absence of proteinuria and normal liver enzymes on LFT.

Few studies suggest that the incidence of tuberculous infection in SLE patients on steroids (prednisolone) increased by 23% for each gram of prednisolone given [2]. Given that prednisolone is the most commonly used drug in SLE, a delicate balance exists between treating a case of SLE and subsequently not letting the treatment induce or reactivate a tuberculous infection.

Abbreviations

SLE: Systemic Lupus Erythematosus
TB: Tuberculosis
RNTCP DOTS: Revised National Tuberculosis Control Program Directly Observed Treatment, Short Course
BD: Twice Daily
M Tb: Mycobacterium tuberculosis
ATT: Anti Tubercular Therapy

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