Multiple Brain Abscesses and Mycotic Aneurysm in a Patient with Lupus Nephritis: A Challenging Diagnosis and Treatment Course

Saba Zaidi, Fariha Mazhar and Ermish Mahfooz

Department of Neurology, Liaquat National Hospital, Karachi, Pakistan

ABSTRACT

Central nervous system (CNS) septic embolisation is an uncommon but life-threatening condition. Clinicians should be aware of the increased risk of this complication in immunocompromised patients such as those suffering from lupus nephritis (LN). In cases of unexplained fever or neurologic symptoms, septic embolisation should be considered in the differential diagnosis. Early recognition and prompt treatment are essential for optimal outcomes. We report a case of a 21-year female with LN who presented with a 2-week history of fever, confusion, and aphasia. Neuro-imaging showed multiple microabscesses and a left middle cerebral artery (MCA) mycotic aneurysm, consistent with septic embolisation. Blood, cerebrospinal fluid, and bronchoalveolar lavage cultures were negative. The patient was treated with a combination of antibiotics and she responded well to treatment.

Key Words: *Female, Lupus nephritis, Mycotic aneurysm, Brain abscess*.

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INTRODUCTION

Septic embolisation occurs when infectious material, such as bacteria or fungi, disseminate through the bloodstream and lodge in distant organs, resulting in abscesses, mycotic aneurysms, or other complications. Early recognition and appropriate management are critical to prevent serious morbidity and mortality. Systemic lupus erythematosus (SLE) is a chronic autoimmune condition that can involve the kidneys, lungs, brain, among other organs. It is linked to endothelial dysfunction, hypercoagulability, and greater susceptibility to infections because of impaired immunological functions. Clinicians caring for patients with lupus should keep their level of suspicion high for septic embolisation in the presence of unexplained fever, neurological symptoms, or evidence of systemic infection. Prompt imaging studies and appropriate antimicrobial therapy are essential for the diagnosis and treatment of septic embolisation in lupus patients.[1-4]

Herein, a case of a 21-year female with lupus nephritis (LN) who presented with a 2-week history of fever, confusion, and aphasia, is reported. Neuro-imaging showed multiple microabscesses and a left middle cerebral artery (MCA) mycotic aneurysm, consistent with septic embolisation.

Correspondence to: Dr. Saba Zaidi, Department of Neurology, Liaquat National Hospital, Karachi, Pakistan
E-mail: drsabazaidi@gmail.com

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CASE REPORT

A 21-year female, a known case of LN, presented in the emergency department with complaints of fever and cough for two months, and altered mentation for two days. Fever was low-grade intermittent with evening rise, having continuous spikes for four days. She was diagnosed with a lower respiratory tract infection and received intravenous antibiotics. The patient had LN, biopsy-proven to be class V membranous glomerulonephritis (GN). The renal biopsy revealed mild hypercellularity with thickening of the capillary basement membranes, with occasional spikes and granular membranous IgG and IgA focal positivity. The patient was on cyclosporine (5 mg/kg in two divided doses) and steroids.

On examination, she had cushingoid facies. Her blood pressure was 140/100 mm Hg with a pulse rate of 143 bpm, respiratory rate of 44 breaths per minute, and oxygen saturation was 92% on room air. The general physical examination showed pallor and facial flushing. The neurological assessment revealed that the patient was awake but lethargic, not following commands, and globally aphasic. Pupils were 3 mm, reactive, and extraocular movements were intact. The motor examination showed normal bulk and tone, brisk reflexes, right plantar extensor, and left flexor. The respiratory system examination showed left-sided crepitations and bronchial breathing on auscultation. The cardiovascular examination was normal and the abdominal examination was unremarkable. The differential diagnosis of the patient's condition included complicated CNS tuberculosis, vasculitic infarctions, cryptococcal meningitis, pyogenic brain abscess, intracranial suppurative thrombophlebitis, CNS toxoplasmosis, an atypical viral infection of the chest with nocardiosis, lupus cerebritis, and sarcoidosis.

The peripheral blood smear showed microcytic, hypochromic anaemia, anisocytosis, poikilocytosis, dimorphic red blood cells, and reactive neutrophil leukocytosis. Blood chemistry showed a creatinine of 0.42 mg/dl (normal: 0.6-1.1 mg/dl), sodium of 132 mmol/L, potassium of 3.7 mmol/L, and bicarbonate of 28.4 mmol/L. The patient's C-reactive protein was 308 mg/dl (normal: 0-0.5 mg/dl), and ESR was 140 mm/1st hr (normal: 2-10). The urine examination revealed protein, haemoglobin, numerous red blood cells, and granular casts. The patient's urinary sodium was 25.8 mEq/l (normal: <20 mEq/l) and creatinine was 25.25 mg/dl (normal range: 28-217 mg/dl). The urine protein to creatinine ratio was 13.3 mg/mg (normal: 0.2 mg/mg). The patient's C3 level was 182 mg/dl (normal range: 90-180 mg/dl), while the C4 was 18.5 mg/dl (normal range: 10-40 mg/dl). The serum albumin was 2.62 g/dl, while calcium, phosphorous, and magnesium levels were 8.55 mg/dl, 3.78 mg/dl, and 1.48 mg/dl, respectively.

MRI brain showed multiple brain abscesses in the supra- and infra-tentorial brain parenchyma, haemorrhagic infarcts in the left parietal-temporal lobe, subarachnoid haemorrhage, meningitis, and vasculitis (Figure 1 A,B). Cerebrospinal fluid (CSF) showed low glucose (38 mg/dl) compared to the serum glucose (156 mg/dl), an increased level of proteins (129 mg/dl), elevated white blood cell count (28/ mm^3) with predominant neutrophils (84%), and a few lymphocytes (14%). The gram stain and culture results for CSF were negative for the growth of any bacteria or fungi. The presence of red blood cells in the CSF could indicate haemorrhage, and these findings were consistent with acute bacterial meningitis.

Injections of Meropenem, 2 g, q8 hourly, injection Vancomycin, 750 mg, q6 hourly, dexamethasone, 10 mg, q6 hourly, and levofloxacin, 500 mg, q12 hourly were started. The total leukocyte count (TLC) on that day was 46.9/mm^3. On the second day, the patient was clinically less irritable. A bronchoalveolar lavage (BAL) was planned and blood cultures were sent. Tests for antinuclear antibody (ANA) and anti-double-stranded DNA (anti-dsDNA) were negative. Echocardiogram showed no vegetation, and the ejection fraction was 60%. The TLC was 38.2/ mm^3. On the third day, the patient was shifted from ICU to the floor. Clinically, she was aphasic but less irritable and started following single-step commands. BAL showed bacterial/acid fast bacilli (AFB) cultures with gene expert to be negative. Fungal stains and culture including candida albicans and cytology were negative. The repeat MRI brain (Figure 2 A,B) showed a re-demonstration of multiple small abnormal intensity areas of diffusion restriction, indicating subacute micro abscesses due to septic emboli. A subacute ischemic infarction was observed in the left posterior frontal and anterior temporal region, along with a small subarachnoid haemorrhage in the basal cistern. CT angiogram of the head showed a partially thrombosed saccular aneurysm arising from the posterior division of the left middle cerebral artery (MCA). The aneurysm measured 1.2 × 2.0 cm with a central non-thrombosed lumen of 5.2 mm and mild spasm in adjacent vessels. The aneurysm was identified as a mycotic aneurysm. Based on the MRI findings and clinical presentation, the final diagnosis was made of CNS septic embolisation leading to abscess formation and mycotic aneurysm. The patient responded well to the treatment.

Meropenem and Vancomycin were stopped after 2 weeks and the patient was switched to Septran and Linezolid considering the possibility of nocardiosis. However, she again started running a fever due to which the same antibiotics were restarted for another 2 weeks. Her clinical condition got better, and she was discharged for home.

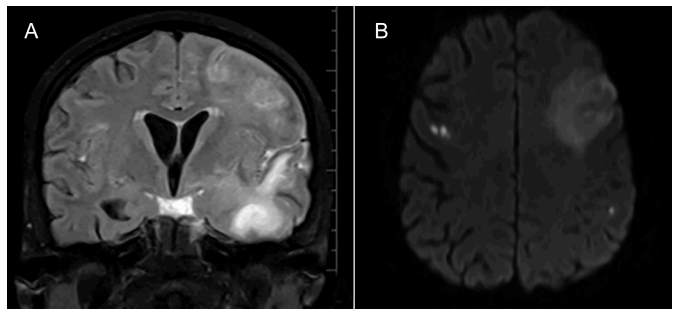

Figure 1: (A) FLAIR sequence hyperintense focus on left temporo-parietal region. (B) DWI restriction in left MCA and patchy in right MCA territory.

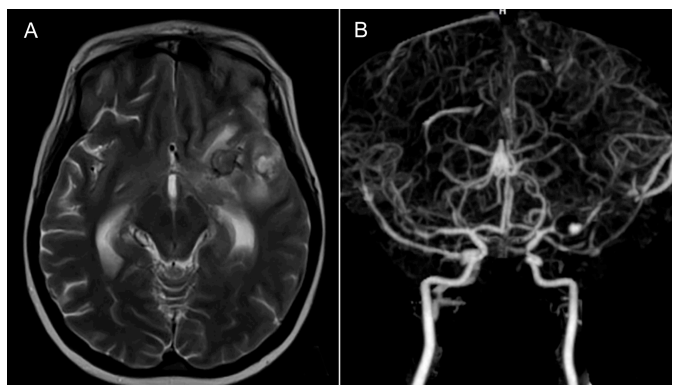

Figure 2: (A) T2 sequence showing the development of the left middle cerebral artery (MCA) aneurysm. (B) MR angiogram, showing it clearly.

DISCUSSION

Septic embolisation refers to the process by which infected materials, such as bacteria or fungi, travel through the bloodstream and lodge in distant organs, forming abscesses or mycotic aneurysms. Septic emboli can originate from a variety of sources, including endocarditis, septicemia, or infected intravascular devices. In the case of our patient, the septic embolisation was likely a complication of her immunocompromised status due to primary disease of LN or because of drugs like steroids or cyclosporine. According to a literature review, patients with lupus have a higher risk of infections because of several factors, such as immunosuppressive medications, kidney damage, and immune system changes brought on by the disease (such as complement deficiencies and reduced neutrophil and macrophage phagocytic activities).[1] In particular, LN has been associated with an increased risk of bacterial and fungal infections, including sepsis, endocarditis, and osteomyelitis.[2] In addition to impaired immune function, patients with lupus may exhibit endothelial dysfunction, which can lead to the development of thrombotic and embolic complications.[3,4]

CNS septic embolisation is uncommon but may be fatal. In the case of unexplained fever, neurological symptoms, or signs of systemic infection in an immunocompromised host, medical professionals should keep a high index of suspicion for this consequence. The pathogenesis of septic embolisation in lupus patients is complex and multifactorial. One potential mechanism includes immune complex formation, which can trigger the complement cascade and encourage vascular injury and thrombosis.[5] Another potential mechanism involves the dysregulation of cytokines and chemokines, which can lead to an abnormal immune response and impaired host defense. Interleukin (IL)-6 and tumour necrosis factor (TNF) levels are higher in SLE patients, whilst IL-10 levels are lower.[6] This cytokine imbalance may contribute to decreased pathogen clearance and encourage the development of septic emboli. Together with these immune-related processes, lupus patients may also have underlying cardiovascular risk factors such as hypertension, dyslipidemia, and obesity, which can raise the risk of atherosclerosis and thrombosis.[7] Endothelial dysfunction, which is commonly observed in SLE patients, may further exacerbate these cardiovascular risk factors and contribute to the formation of septic emboli.[8] Management of septic embolisation in lupus patients is challenging and requires a multidisciplinary approach.

In conclusion, clinicians need to understand the increased risk of septic embolisation in patients of LN and consider this diagnosis in the presence of unexplained fever, neurologic symptoms or evidence of systemic infection.

PATIENT'S CONSENT:
A written informed consent was obtained from the patient.

COMPETING INTEREST:
The authors declared no competing interest.

AUTHORS' CONTRIBUTION:
SZ, FM, EM: Substantial contributions to the conception, drafting the work, and revising it critically for important intellectual content.
All the authors have approved the final version of the manuscript to be published.

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