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Case Report

# Systemic Lupus Erythematosus Presenting as Mononeuritis Multiplex and Coronary Artery Vasculitis: A Rare Case Report

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**Abstract:**

Systemic Lupus Erythematosus (SLE) is a autoimmune disease characterized by multisystem involvement and heterogeneous clinical manifestations. Neurological, cardiovascular and Renal complications such as mononeuritis multiple, coronary artery vasculitis and Lupus nephritis are rare but severe presentation of SLE. This case report describes a patient with SLE who presented with mononeuritis multiplex and coronary artery vasculitis. Emphasizing the complexities of diagnosis, Treatment approaches and Patients outcome.

**Key words:** systemic lupus erythematosus; mononeuritis multiplex; coronary vasculitis; peripheral neuropathy; cardiac involvement; immunosuppressive therapy

## Introduction

Systemic lupus erythematosus (SLE) is a complex autoimmune disease characterized by multisystem involvement and wide range of clinical manifestations. It primarily affects young women, mean age is 28 years but it can occur in individuals of any age and gender. The clinical spectrum of SLE includes dermatological, musculoskeletal, renal, and neurological symptoms. Among its various presentations, neurological manifestations such as mononeuritis multiplex (MM) and vasculitis, particularly coronary artery vasculitis, are rare but significant. This report presents a rare case of SLE that manifested as mononeuritis multiplex and coronary artery vasculitis, highlighting the difficulties encountered in diagnosis and treatment.

## Case Presentation

A 26-year-old female previously healthy presented with progressive weakness of Right lower limb over 1 week duration, accompanied by sensory loss and paresthesia. The patient also reported a history of ischemic type of chest pain associated with autonomic disturbances and peri orbital edema. she did not have any symptoms of Connective tissue disorder Other than the history of Past First trimester Miscarriage. On General Examination, Patient has peri-orbital edema and mild pallor. Neurological examination revealed decreased muscle strength (3/5) in dorsiflexion and eversion of Right lower limb, diminished sensation in a "glove and stocking" distribution in Right Lower limb, and reduced deep tendon reflexes. Cardiovascular, Respiratory and Abdominal examination were normal.

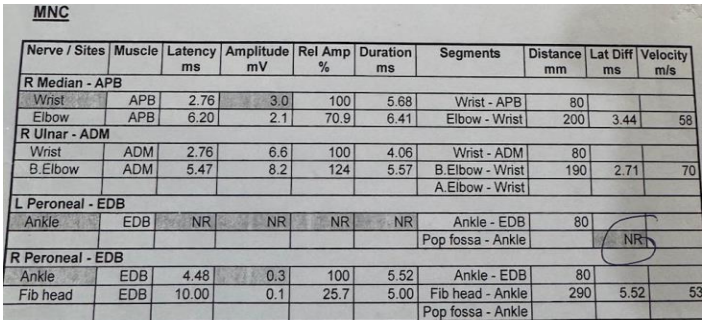
| Investigation | value | Reference range |
|---|---|---|
| WBC -10^3/uL | 8.38 | 4.00-11.00 |
| HGB- g/dl | 9.1 | 11.0-15.0 |
| PLT- 10^3 uL | 126 | 150-450 |
| Sodium- mmol/L | 128 | 136-145 |
| Potassium- mmol/L | 3.9 | 3.5-5.1 |
| AST- U/L | 23 | 15-37 |
| ALT – U/L | 29 | 12-78 |
| Alkaline Phosphatase- U/L | 61 | 46-116 |
| Total protein – g/L | 69 | 64-82 |
| Albumin- g/L | 23 | 34-50 |
| Total Bilirubin -umol/L | 3.5 | 3.4-17.1 |
| Blood Urea – mmol/L | 2.6 | 1.8-6.3 |

| | | |
|---|---|---|
| Creatinine – ummol/L | 77 | 62-115 |
| C Reactive Protein – mg/L | 48 | 0-5 |
| Phosphorus – mmol/L | 0.6 | 0.8-1.6 |
| Calcium – mmol/L | 1.95 | 2.1-2.5 |
| ESR – mm/1st hr | 115 | |
| LDH -U/L | 304 | 81-234 |
| Urine Albumin | +++ | |
| Urine Red cells | Negative | |
| Urine Protein – g/dl | 1.85 | |
| Creatinine- umol/l | 11 027 | |
| PCR-mg/mmol | 168 | |
| Hemoglobin A1c | 6.5% | |
| Hepatitis B Surface Antigen | 0.15 (negative) | <0.89 |
| Anti – Hepatitis C Virus | 0.1 (negative) | <0.89 |
| ANA Levels – AU/ml | 389 | 0-40 |
| ANA Titers- Nuclear pattern | 1:1280 (Positive) | |
| ds DNA Antibody | Positive | |
| P-ANCA | Negative | |
| C -ANCA | Negative | |
| Complement C 3 – mg/dl | 49 | 83-177 |
| Complement C 4 -mg/dl | 5 | 12-36 |
| Troponin I- ng/ml | 1.51 | 0-0.12 |

**Table 1:** Hematological, biochemical, imagining investigations.

ECG- ST segment Elevation seen in Chest lead V2, V3, V4 and Lead 11,111, aVF with ST elevation seen in V4R

Nerve conduction test-There is evidence of asymmetrical patchy axonal sensory and motor neuropathy which could be due to MNM type pathology



MNC

| Nerve / Sites | Muscle | Latency ms | Amplitude mV | Rel Amp % | Duration ms | Segments | Distance mm | Lat Diff ms | Velocity m/s |
|---|---|---|---|---|---|---|---|---|---|
| R Median - APB | | | | | | | | | |
| Wrist | APB | 2.76 | 3.0 | 100 | 5.68 | Wrist - APB | 80 | | |
| Elbow | APB | 6.20 | 2.1 | 70.9 | 6.41 | Elbow - Wrist | 200 | 3.44 | 58 |
| R Ulnar - ADM | | | | | | | | | |
| Wrist | ADM | 2.76 | 6.6 | 100 | 4.06 | Wrist - ADM | 80 | | |
| B.Elbow | ADM | 5.47 | 8.2 | 124 | 5.57 | B.Elbow - Wrist | 190 | 2.71 | 70 |
| | | | | | | A.Elbow - Wrist | | | |
| L Peroneal - EDB | | | | | | | | | |
| Ankle | EDB | NR | NR | NR | NR | Ankle - EDB | 80 | | |
| | | | | | | Pop fossa - Ankle | | NR | |
| R Peroneal - EDB | | | | | | | | | |
| Ankle | EDB | 4.48 | 0.3 | 100 | 5.52 | Ankle - EDB | 80 | | |
| Fib head | EDB | 10.00 | 0.1 | 25.7 | 5.00 | Fib head - Ankle | 290 | 5.52 | 53 |
| | | | | | | Pop fossa - Ankle | | | |

**Figure 1:** Nerve conduction test

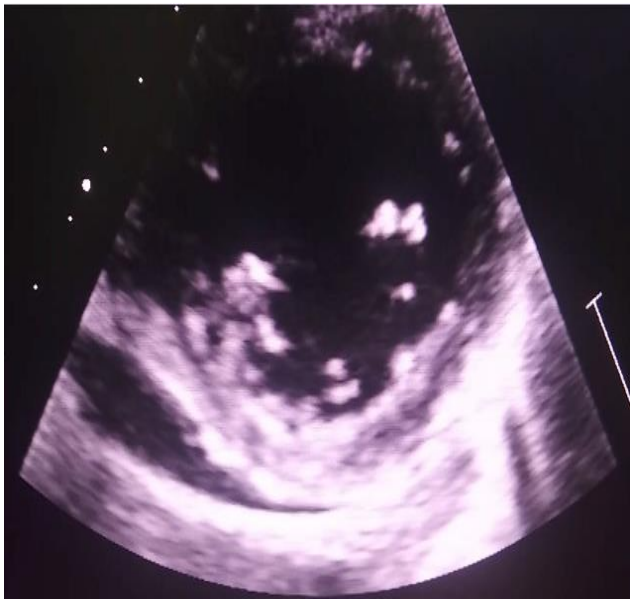


**Figure 2:** Echo picture of Right coronary Artery Dilation

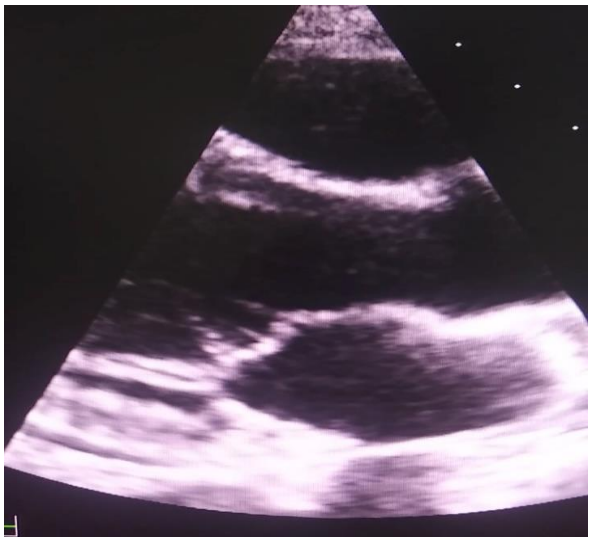


**Figure 3:** Right coronary Artery

### Diagnostic Challenges

The diagnostic process in this case was complex due to the overlapping symptoms of SLE and mononeuritis multiplex. Mononeuritis multiplex is characterized by asymmetric sensory and motor dysfunction due to ischemic or inflammatory lesions affecting individual nerves. In the context of SLE, it may result from vasculitis affecting vasa nervorum, leading to ischemia and nerve damage.

Coronary artery vasculitis in SLE can present as acute chest pain, myocardial infarction, or even sudden cardiac death. , In conjunction with her neurological symptoms, led to a suspicion of an underlying autoimmune process. The challenge was to differentiate between SLE-related complications and other possible neurological disorders such as multiple sclerosis or diabetic neuropathy.

### Diagnosis and Treatment

With the above Investigation it was concluded that this patient's presentation is due to SLE, complicated with mononeuritis multiplex and coronary artery vasculitis and renal involvement. The medical management of this case involved a multidisciplinary approach, including rheumatology, neurology, and cardiology. The patient was initiated on high-dose corticosteroids pulses with Methyl prednisone. Intravenous cyclophosphamide pulse therapy was administered to mitigate the neurological manifestations and renal manifestations, specifically targeting the mononeuritis multiplex and renal lupus. A rapid response to the treatment was noted both clinically and biochemically. Antiplatelet therapy was initiated, and the patient was started on hydroxychloroquine as a disease-modifying antirheumatic drug (DMARD) to control the underlying SLE. Follow-up evaluations revealed gradual improvement in motor and sensory functions, although some residual deficits persisted.

This case illustrates the complex interplay of SLE and its neurological manifestations, particularly mononeuritis multiplex and coronary artery vasculitis. The pathophysiology of MM in the context of SLE involves immune-mediated vasculitis affecting the vasa nervorum, resulting in ischemic injury to the nerves. Coronary artery vasculitis, although less common, poses substantial risks due to the potential for myocardial infarction.

Early identification and management of these symptoms are essential for enhancing patient results. This instance emphasizes the need to take into account SLE in individuals with unexplained neurological symptoms, particularly when they lack any systemic features of other connective tissue diseases.

## Conclusion

This case of systemic lupus erythematosus, characterized by mononeuritis multiplex and coronary artery vasculitis, underscores the importance of being aware of the varied symptoms of this autoimmune disease. An integrated management approach combining corticosteroids and immunomodulatory therapies can significantly enhance outcomes for patients with complex SLE-related vasculitis presentations. Future research should concentrate on the underlying mechanisms of SLE-related vasculitis and the development of targeted treatments.

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