

Electro-Clinical Profile of POEMS Cases at Montpellier University Hospital

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Abstract

Introduction: POEMS syndrome is a rare, multisystemic B lymphoid hematological disease, classified among paraneoplastic disorders. **Objective:** The main objective of our study was to investigate the electro-clinical aspects of POEMS cases. **Methodology:** This descriptive study evaluated five POEMS patients seen from 2014 to 2024 at the Gui de Chauliac functional explorations unit of Montpellier University Hospital. For diagnosis, the presence of two mandatory major criteria including polyneuropathie and monoclonal gammopathy, plus one of the other major criterion (elevated VEGF) were required. **Results:** The average age at diagnosis was 54.6 years, with a predominance of male patients (80%). Most patients had a follow-up duration between 8 and 10 years. The most frequently observed clinical signs included distal-predominant paresthesias of the lower limbs, sock-like hypoesthesia, and bilateral foot drop. The mean disability score (ONLS) was 4. The mean interval before performing the electroneuromyogram was 18 months. Elevated VEGF levels could reach up to 7000 pg/ml. All patients exhibited demyelinating neuropathy, which was generally intermediate and homogeneous in the upper limbs, with early, length-dependent axonal loss (4/5). **Conclusion:** POEMS manifests as an aggressive neuropathy in a young adult. Early axonal loss is found in the lower limbs and secondary demyelination in the upper limbs without conduction block.

Keywords

POEMS, Montpellier, Gammopathy, Neuropathy

1. Introduction

POEMS is a paraneoplastic syndrome with multisystem involvement, the acro-

nym referring to polyneuropathy, organomegaly, endocrinopathy, monoclonal gammopathy, and *skin changes*) [1]. The initial cases were described by Crow in 1956 and subsequently by Fukase in 1968, which gave rise to the term Crow-Fukase syndrome. The condition is also referred to as Takatsuki syndrome. Initially, the disease appeared more prevalent in Japan due to the early description of cases from this region. Nevertheless, subsequent large case series have been reported in France, the USA, China, and India. A Japanese epidemiological survey estimated its prevalence at 0.3 per 100,000 Inhabitants [2]-[4]. This is a rare but potentially severe disease, where clinical heterogeneity often contributes to delayed diagnosis [3]. There is a male predominance, with a mean age of onset between 40 and 50 years.

The pathophysiology remains poorly understood. Recent progress has been made in elucidating the mechanisms underlying POEMS. The most probable cause appears to be an increase in proinflammatory cytokines, particularly interleukins (IL6 and IL12) [1] and Tumor Necrosis Factor (TNF), rather than clonal plasma cell infiltration.

Vascular endothelial growth factor (VEGF) is regarded as a cytokine produced by osteoclasts and marrow-derived cells. VEGF plays a central role in regulating angiogenesis and microvascular permeability by targeting various endothelial cell receptors. Its production is modulated by several factors, including hypoxia-inducible transcription factor 1 [5]. Nerve biopsy is seldom valuable for diagnosing POEMS; however, it can assist in distinguishing POEMS from AL amyloidosis. In such cases, the biopsy demonstrates significant demyelination and uncompactation of myelin lamellae. Ovoid bodies, indicative of secondary axonal degeneration, may also be observed. The underlying mechanism of peripheral neuropathy in POEMS is thought to involve endothelial injury, either directly or indirectly caused by the activation of endothelial cells by VEGF, which is overexpressed in the nerves of patients with POEMS. These changes result in secondary ischemic microangiopathy, characterized by vascular lesions and increased vascular permeability.

The main criteria of the syndrome remain polyradiculoneuropathy, gammopathy Monoclonal, bone lesions, or elevated blood VEGF, as well as Castleman Disease. The minor criteria include organomegaly, endocrinopathy, skin changes, papilledema, thrombocytosis or polycythemia, and edema [6] [7].

Neuropathy is constant and found in 100% of cases in the series by Nakanishi *et al.* [4].

The diagnosis is made in the presence of at least three major criteria including polyradiculoneuropathy and at least one minor criterion [3].

Initially misdiagnosed as chronic inflammatory demyelinating polyneuropathy, the diagnosis is frequently delayed [3]. Neurophysiologically, conduction blocks are less common in POEMS than in CIDP. Predominantly length-dependent axonal involvement is observed, accompanied by more frequent fibrillations than in CIDP. The clinical significance of monoclonal gammopathy of the IgG or IgA

Lambda type remains limited [5]. In contrast to multiple myeloma, there is no hypercalcemia, bone fracture, or light chain-related renal failure, nor is there a marked increase in monoclonal immunoglobulin during disease progression. Bone marrow plasma cells are rare and resemble those observed in monoclonal gammopathies of undetermined significance [1].

The discovery of vascular endothelial growth factor (VEGF) in association with pathology has been of considerable benefit to clinical diagnosis and monitoring of therapeutic response [8].

Patient management continues to involve a multidisciplinary team consisting of hematologists, neurologists, neurophysiologists, and physiotherapists. The selection of therapy depends on the timeliness of intervention, the severity of the disease, and the degree of medullary involvement.

Thus, therapeutic algorithms have been proposed by Angela Dispenzieri *et al.* [9]. Radiotherapy remains the first-line treatment in focal forms (for patients with two or fewer plasmacytoma lesions).

In cases of more extensive involvement, characterized by diffuse sclerotic lesions or widespread bone marrow infiltration, patients are managed with plasma exchange and intravenous immunoglobulins. In systemic forms, the standard treatment for POEMS consists of chemotherapy with thalidomide (a VEGF inhibitor) and lenalidomide followed by stem cell transplantation, resulting in favorable hematological and neurological outcomes. Lenalidomide is effective, achieving approximately 90% favorable responses in cases with neuropathy, except among patients at advanced stages of the disease. The most favorable outcomes have been observed with chemotherapy followed by high-dose melphalan and subsequent stem cell transplantation in eligible patients. For patients ineligible for transplantation, a regimen of lenalidomide and dexamethasone alone is preferred.

Furthermore, functional rehabilitation is required in the presence of motor deficits. Additionally, to prevent falls, orthopedic footwear is sometimes indicated. In cases of neuropathic pain, treatment with Gabapentin or a tricyclic antidepressant should be considered [5]. Management should also address endocrinopathy, thrombotic risk, and include anti-infective therapy [7] [10] [11].

Aside from corticosteroids and the surgical or radiotherapeutic management of a solitary bone lesion, available treatments yield disappointing results. The five-year survival rate is 60%. Disease progression is marked by worsening polyneuropathy and general symptoms, most notably progressive cachexia, which account for the majority of deaths [7] [11].

The primary aim of this study was to describe the electroclinical characteristics of POEMS cases followed during functional assessments at Gui de Chauliac in Montpellier and to compare these findings with those reported in the literature.

2. Methodology

This descriptive study evaluated patients with POEMS syndrome who were seen between 2014 and 2024 at the functional evaluation unit (GUI) of CHAULIAC,

Montpellier University Hospital (CHU). POEMS was diagnosed in the hematology department according to the Dispenzieri 2017 criteria. Patient recruitment was conducted through a comprehensive review of electronic medical records available in the Dxcare information system. Data collected included socio-demographic characteristics, clinical features, ONLS disability score assessment, ENMG diagnostic criteria, and paraclinical findings.

The electrodiagnostic criteria used to define axonal and demyelinating neuropathy as follows:

Axonal neuropathy:

- Reduction or abolition of the amplitude of motor potentials in the motor conduction study;
- Reduction or abolition of the amplitude of sensory potentials in the sensory conduction study;
- Possibly, presence of spontaneous resting activities (Fibs, PLD) and reduction in the number of motor unit potentials detected (voluntary contraction trace), sometimes polyphasic MU.

Demyelinating neuropathy:

- Prolongation of distal latency;
- Prolongation of distal latency;
- Reduction of segmental or proximal conduction velocity;
- Conduction block;
- Segmental temporal dispersion.

A primary motor conduction velocity (MCV) slowing was noted, with values below 35 m/s in the upper limbs and below 30 m/s in the lower limbs.

3. Results

A total of 5 patients were included in the study. The mean age at diagnosis was 54.6 years. Males were predominant (80%), with females representing 20% of cases. Most patients were between 39 and 46 years old. The majority had a follow-up duration between 8 and 10 years. The most common clinical manifestations were predominantly distal paresthesia of the lower limbs, decreased tactile sensation, and impaired vibration sense in a stocking distribution, accompanied by bilateral foot drop. The mean disability (ONLS) score was 4, ranging from 0 to 10. Additionally, Castleman's disease was reported in a single patient, a young woman with a history of MGUS, presenting mainly with sensory symptoms and no motor impairment. The majority of patients exhibited lower limb edema (3/5), endocrinopathy—including one case of dysthyroidism and one of hyperprolactinemia (3/5), skin changes (2/5), and organomegaly (2/5) (**Table 1**).

The average time to perform the ENMG from the first symptoms was 18 months, with extremes ranging from 2 months to 36 months.

A demyelinating neuropathy was identified in all patients. In the upper limbs, it presented as relatively intermediate and homogeneous, while an early length-dependent axonal loss (4/5), predominantly affecting the lower limbs, was also ob-

served (**Figure 1**).

Table 1. Distribution by age, gender, and clinical study.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Age at diagnosis	60	39	39	89	46
Sex	M	F	M	M	M
Specific history	Left MCA stroke	MGUS	Without related history	Atrial fibrillation	Infantile right hemiplegia
Follow-up period	8 years	8 years	10 years	2 months	10 years
Early symptoms	Burning of the feet	Paresthesia of the legs	Distal limb cramps	Sock and glove paresthesias	Calf cramps
Motor deficit	Bilateral steppage	No motor deficit	Bilateral steppage	Bilateral steppage	Motor deficit of the left upper limb
Subjective sensory deficit	Paresthesia of the feet	Paresthesias of the legs	Cramps in the lower limbs and hands	None	Paresthesias of the plantar arch
Objective sensory deficit	Hypoesthesia in a sock distribution Hypopallesthesia ascending to the level of the anterior tibial tuberosity	None	Hypopallesthesia of the ankles	Hypoesthesia to all modalities up to the knees, apallesthesia	Hypopallesthesia of the halluces and ankles
Castleman disease	No	Yes	No	No	No
Edema (LEO, pleurisy, ascites, pericarditis)	Yes (IMO)	Yes (IMO)	No	Yes (IMO)	No
Bone lesion	No	Yes	Yes	Yes	Yes
Papilledema	No	No	No	No	No
Endocrinopathy	Hypothyroidism, hypogonadism, hyperprolactinemia	Hyperprolactinemia at 493 microIU/L; Hashimoto's thyroiditis	hypothyroidism, hypogonadism, hyperprolactinemia (32ng/ml)	Yes (low cortisol levels)	No
Skin modifications	Periorbital xanthomas	No	Cherry angioma	No	No
Organomegaly	No	No	Hepatosplenomegaly CMD + HTAP.FE 35%	Hepatosplenomegaly	No

Most patients presented with a mixed demyelinating and axonal polyneuropathy.

Immunofluorescence most often identified a monoclonal IgA Lambda gammopathy (4/5 cases). All patients exhibited elevated VEGF levels, with initial values sometimes exceeding 7000, followed by substantial reductions following treatment.

Bone lesions of the spine, ribs, and pelvis, identified on PET and/or CT scan, were observed in most patients (3/5) (**Table 2**).

On the therapeutic level, chemotherapy appears to stop the progression but sig-

nificant aftereffects are observed, with, moreover, resistance to intravenous immunoglobulins.

All patients received at least two cycles of Revlimid-Dexamethasone, administered monthly, with an initial average of 3.6 cycles. In one patient, the regimen was used for consolidation following autologous transplantation. Autologous bone marrow transplantation was performed in two patients, while radiotherapy was given to the oldest patient in our cohort.

Neuropathy management primarily consisted of prescribing Laroxyl, most frequently administered as drops at an average dose of 9 drops (2/5). In one patient, treatment included Tegeline, followed by plasma exchanges combined with corticosteroid therapy after secondary disease progression.

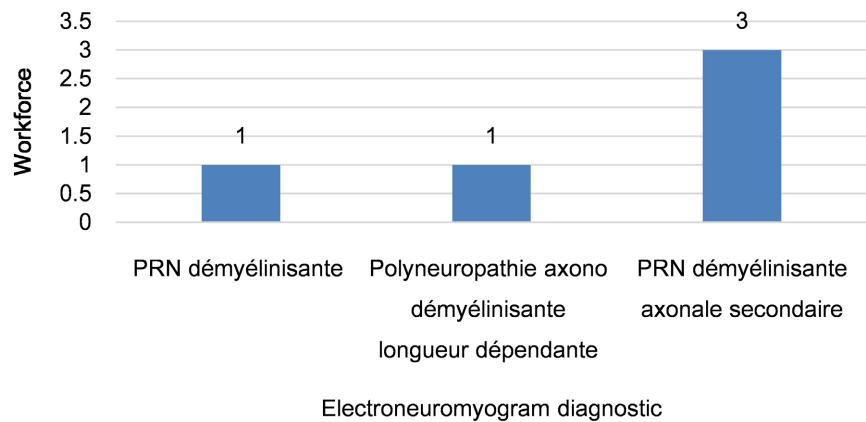


Figure 1. Distribution according to electroneuromyogram diagnosis of POEMS cases EAN/PNS criteria.

Table 2. Distribution according to the results of the additional assessment.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Immunology (IFE)	Without peak; Ig A Lambda (IFE normal)	IgG Lambda, Kappa light chains 16.8, L 18, ratio 0.9	Absence of measurable peak IFE gammopathy with monoclonal IgA Lambda with a K/L ratio at 2.05	EPS normal; IgG peak 4.7 g/l IFE IgG Lambda , light chains Kappa 48.33, Lambda 94.02 ratio 0.51;	Monoclonal IgA lambda and IgM lambda spike on immunofixation without a measurable spike on the SPE, free lambda light chains at 29.5 mg/l , free kappa at 21.1 mg/l, normal ratio.
VEGF (evolution of levels during follow-up)	425 - 1.12 (at 5 years)-343	7848-1125	1430 - 2704 -4630-118 -115-normalization in 2021	Not reported	Not reported
Platelets	158 000	Thrombocytosis	Thrombocytosis (490000)	Normal	255 000
Renal function	Creatinine 84 GFR 84	Creatinine 54	Creatinine 127, proteinuria 0.4 g/l	Creatinine 109	Creatinine 114 DFG 63 24h protein: 80

Continued

Blood sugar	5.16 mmol/l	Not reported	Not reported	Not reported	4.6 mmol/l
TSH	1.05	0.07	1.55	5.5	Not done (ND)
BGSA	Nonspecific lymphocytic sialadenitis with 1 lymphocyte aggregation focus	NF	NF	NF	NF
TEP scanner/TAP	PET: Absence of foyer hypermetabolic bone with pathological appearance in the explored regions.	PET: Several hyperfixing bone sites in the spine, ribs, pelvis with a maximum SUV of 8.5 at the left iliac level as well as hypermetabolic lymphadenopathies with a maximum SUV of 10.7 in the right inguinal region.	TAP: osteocondensing bone lesions of the spine	PET: Intense hypermetabolism in relation to the lytic lesion of the vertebral body and pedicles of T6 with para-vertebral and epidural infiltration. Two hypermetabolic foci aligned with the right costal grid.	PET: Absence of significant uptake. Persistence of an uptake at the cardia and of the ileocecal valve and a slightly heterogeneous hepatic fixation at segment IV. Stability of the left adrenal lesion.

4. Discussion

POEMS remains a rare disease. This study specifically examined the electroclinical profiles of POEMS cases seen in the functional exploration department at Gui de Chauliac from 2014 to 2024, comparing them with cases reported in the literature. Over the past decade, we identified only 5 patients with this pathology. During this interval, our cohort was larger than that reported by Bouchra, who found the same number of cases but over a longer time span [12]. In contrast, Souissi identified a greater number of cases within a shorter period [13]. The smaller size of our cohort may indicate a lower incidence of cases in our region compared to the aforementioned Tunisian study, or it may be attributable to patient recruitment bias. Specifically, patients were included in electrophysiological functional explorations only after hematology follow-up.

The median age of our patients was 54 years, with extremes of 39 years and 89 years at the time of diagnosis. This result is similar to that of the study by Souissi in 2019, which reported a mean age of 56.1 years [13]. However, Bouchra reported a lower age [12].

The clinico-electrical profile of our POEMS patients compared to those of other patients in the literature (respectively in Morocco and Tunisia) is summarized in **Table 3**.

An earlier retrospective study, spanning 15 years and comprising two series for a total of 25 American and Japanese patients, as well as an additional series of non-Asian patients identified in the literature, reported a mean patient age of 49 years. These latter series also noted a predominance of older and male patients, a finding

that was similarly observed in our study. Furthermore, these pathologies are generally identified in a paraneoplastic context [14], and within this age group, tumor-related diseases remain common overall, which may account for the higher number of reported cases. However, in our study, no neoplastic context was identified.

In most patients, symptom onset was characterized by paresthesia of the lower limbs, frequently accompanied by a steppage gait disturbance. Furthermore, sensory-motor conduction studies confirmed predominant involvement of the lower limbs, consistent with the initial impairment of long fibers [15].

A study investigating the cellular origin of this pathology reported a case of a 65-year-old patient with POEMS syndrome complicated by B-cell lymphoma, who exhibited a favorable therapeutic response; in this case, lower limb weakness also constituted one of the initial presenting symptoms [14].

Furthermore, only a single case of Castleman Disease was observed in our cohort, accounting for 20% of our study. Notably, the literature indicates that 11 to 30% of patients with Castleman Disease, or with histology similar to this pathology, are associated with cases of POEMS, which is consistent with our data [16].

Regarding disability, while some patients were asymptomatic, a generally moderately severe level of disability was observed, reflected by a mean ONLS score of 4—corresponding to an inability to walk and perform daily tasks without assistance—with scores ranging from 0 to 10 during patient follow-up. This indicates a significant impact of neuropathy on activities of daily living. However, in a study examining the association between POEMS syndrome and ischemic stroke (AVCI), 250 cases of POEMS were collected, with 8% presenting with an ischemic stroke. The ONLS score in this context was high, exceeding 4, suggesting that comorbidity may exacerbate disability. Additionally, a higher frequency of ischemic stroke has been reported in untreated patients with POEMS syndrome. In our cohort of 5 patients, only a single case of ischemic stroke was identified, which occurred concurrently with the onset of POEMS and prior to the initiation of treatment [17].

From a paraclinical perspective, an elevation of the Gamma peak in the Lambda light chain, as well as increased VEGF levels, was observed in most of our patients, with some cases reaching very high values (up to 7848), as reported in the literature [18]. Treatment generally yielded a favorable response in VEGF follow-up, occasionally resulting in normalization of the levels. This cytokine is thus a valuable diagnostic and monitoring marker for the disease.

Bone lesions were present in nearly all of our patients. Endocrinopathies were observed in the majority of cases, consistent with previous literature [13]. In Zhou's study, this was manifested as diabetes, whereas in our cohort, 2 patients presented with dysthyroidism [14]. Soubrier's study identified gynecomastia and amenorrhea as predominant endocrine features. Regarding organomegaly, splenomegaly and, more notably, hepatomegaly were observed in over one-third of our patients, consistent with existing reports, although some cohorts have documented a higher prevalence [15].

The study notes 18-month interval between symptom onset and electroneuromyogram (ENMG). This delay in diagnosis could be observed due to the multifaceted symptoms with multi-organ involvement of the disease, which can cause patients to seek multiple medical opinions before receiving a definitive diagnosis, but it could also be explained by the aggressive nature of the disease.

Electroneuromyography (ENMG) findings in all our patients indicated features consistent with demyelinating polyradiculoneuropathy; among these, four patients exhibited a significant reduction in amplitudes, pointing to secondary axonal involvement. Similarly, in the series by Soubrier, axonal involvement was predominant [15].

Table 3. Comparative study of the clinico-electrical profile of POEMS cases.

	Our study	Etude Bouchra F <i>et al.</i>, 2015	Etude Souissi W <i>et al.</i>, 2019
Type of study	Retrospective	Retrospective	Retrospective
Duration of the study (Period)	10 years (2014 - 2024)	17 years old	4 years (2014 - 2018)
Cohort size	5 cases	5 cases	7 cases
Gender predominance (Sex ratio)	Masculine (4)	Masculine (0)	Masculine (1,3)
Average age	54.6 years	40 years	56.1 years
Gammapathy	5/5	5/5	7/7
Peripheral neuropathy	5/5	5/5	7/7
Skin signs	2/5	Late hemangiomas	4/7
Endocrinopathy	4/5	Not reported?	4/7
Edema	3/5	Not reported?	7/7
Electroneuromyogram (ENMG)	Neuropathy -Secondary axonal demyelinating (PRN pattern) (3/5) -Axonal (1/5) -Demyelinating (1/5)		Axonal sensorimotor neuropathy (4/7) and demyelinating (3/7)

Furthermore, POEMS presents diagnostic challenges when distinguishing it from chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) [3]. According to the cases reported in the literature, axonal involvement is observed more frequently in POEMS than in CIDP. This axonal loss is more pronounced in the lower limbs, as was similarly observed in our study (severely affected PGAM). Demyelination, on the other hand, predominates in the nerve trunk rather than at the distal nerve ending. Alterations in conduction velocity are found to be more intermediate than distal when compared with CIDP. Conduction blocks are less common [18].

Axonal involvement as well as multisystem involvement probably account for the severity of POEMS cases observed in our context as well as those noted in other studies. Although studies in the literature have not shown large cohorts, the limi-

tation of our study remained the small sample size. In addition, this was a single-center study, which could also explain the sample size obtained. Furthermore, the retrospective collection of data may limit the generalizability of the findings, with some data being incomplete.

5. Conclusion

POEMS presents as an aggressive neuropathy in young adults. Electrophysiologically, it is characterized by early axonal loss in the lower limbs and secondary demyelination in the upper limbs, without conduction block. It is nevertheless useful to differentiate POEMS from CIDP, as treatment will depend on the underlying condition. Indeed, as in CIDP, the treatment of generalized lesions involves the prescription of intravenous immunoglobulins, but in POEMS, resistance to intravenous immunoglobulins is commonly observed, hence the importance of early diagnosis. This diagnosis also appears crucial for initiating appropriate therapies that target plasma cells.

Contributions

All authors contributed equally to this work. Diouf Mbourou Nelly and Dr. Taeib Guillaume drafted the manuscript. Dr. Saphou Damon Michel Arnaud, Dr. Klevo r Raymond, Dr. Attal Arthur, Dr. Esselin Florence, Dr. Durand A, Dr. Mambila Grass, and Dr. Malekou Doris conducted the literature review and revised the manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

References

- [1] Rose, C., Mahieu, M., Hachulla, E., Façon, T., Hatron, P., Bauters, F., *et al.* (1997) Le POEMS syndrome. *La Revue de Médecine Interne*, **18**, 553-562. [https://doi.org/10.1016/s0248-8663\(97\)80807-7](https://doi.org/10.1016/s0248-8663(97)80807-7)
- [2] Dispenzieri, A., Kyle, R.A., Lacy, M.Q., Rajkumar, S.V., Therneau, T.M., Larson, D.R., *et al.* (2003) POEMS Syndrome: Definitions and Long-Term Outcome. *Blood*, **101**, 2496-2506. <https://doi.org/10.1182/blood-2002-07-2299>
- [3] Arkhipov, I.E., Vergunova, I.Y., Malkova, N.A. and Korobko, D.S. (2023) POEMS-Syndrome. *S.S. Korsakov Journal of Neurology and Psychiatry*, **123**, 15-21. <https://doi.org/10.17116/jnevro202312307215>
- [4] Mahfoudhi, M., Turki, S. and Kheder, A. (2015) POEMS Syndrome: Un diagnostic à ne pas méconnaître. *Pan African Medical Journal*, **20**, Article 448. <https://doi.org/10.11604/pamj.2015.20.448.6353>
- [5] Cerri, F., Falzone, Y.M., Riva, N. and Quattrini, A. (2018) An Update on the Diagnosis and Management of the Polyneuropathy of POEMS Syndrome. *Journal of Neurology*, **266**, 258-267. <https://doi.org/10.1007/s00415-018-9068-4>
- [6] Dispenzieri, A., Kourelis, T. and Buadi, F. (2018) POEMS Syndrome: Diagnosis and Investigative Work-Up. *Hematology/Oncology Clinics of North America*, **32**, 119-

139. <https://doi.org/10.1016/j.hoc.2017.09.010>
- [7] Warsame, R., Yanamandra, U. and Kapoor, P. (2017) POEMS Syndrome: an Enigma. *Current Hematologic Malignancy Reports*, **12**, 85-95. <https://doi.org/10.1007/s11899-017-0367-0>
- [8] Brown, R. and Ginsberg, L. (2018) POEMS Syndrome: Clinical Update. *Journal of Neurology*, **266**, 268-277. <https://doi.org/10.1007/s00415-018-9110-6>
- [9] Jaccard, A. (2018) POEMS Syndrome: Therapeutic Options. *Hematology/Oncology Clinics of North America*, **32**, 141-151. <https://doi.org/10.1016/j.hoc.2017.09.011>
- [10] Khwaja, J., D'Sa, S., Lunn, M.P. and Sive, J. (2022) Evidence-Based Medical Treatment of poems Syndrome. *British Journal of Haematology*, **200**, 128-136. <https://doi.org/10.1111/bjh.18400>
- [11] Keddie, S. and Lunn, M.P. (2018) POEMS Syndrome. *Current Opinion in Neurology*, **31**, 551-558. <https://doi.org/10.1097/wco.0000000000000610>
- [12] Ferdous, B., Habtany, Y., El Mautawakil, B., Otmani, H., Rafai, M. and Slassi, I. (2015) POEMS syndrome: Étude de 5 cas. *Revue Neurologique*, **171**, A153-A154. <https://doi.org/10.1016/j.neurol.2015.01.339>
- [13] Souissi, W., Riahi, A., Abuhassan, A., Bedoui, I., Mansour, M., Zaouali, J., *et al.* (2019) Le syndrome de POEMS: Étude de 7 cas et revue de la littérature. *Revue Neurologique*, **175**, S37-S38. <https://doi.org/10.1016/j.neurol.2019.01.121>
- [14] Zhou, L., Lu, J., Lin, Z., Wang, X., Luo, L., Wang, C., *et al.* (2023) POEMS Syndrome: Origination from Clonal Plasma Cells or B Cells? *Hematology*, **28**, Article ID: 2186044. <https://doi.org/10.1080/16078454.2023.2186044>
- [15] Soubrier, M.J., Dubost, J. and Sauvezie, B.J.M. (1994) POEMS Syndrome: A Study of 25 Cases and a Review of the Literature. *The American Journal of Medicine*, **97**, 543-553. [https://doi.org/10.1016/0002-9343\(94\)90350-6](https://doi.org/10.1016/0002-9343(94)90350-6)
- [16] Andhavarapu, S. and Jiang, L. (2013) POEMS Syndrome and Castleman Disease. *Blood*, **122**, 159-159. <https://doi.org/10.1182/blood-2013-01-477661>
- [17] Feng, J., Gao, X., Zhao, H., He, T., Zhang, C., Shen, K., *et al.* (2020) Ischemic Stroke in Patients with POEMS Syndrome. *Blood Advances*, **4**, 3427-3434. <https://doi.org/10.1182/bloodadvances.2020001865>
- [18] Dispenzieri, A. (2021) POEMS Syndrome: 2021 Update on Diagnosis, Risk-Stratification, and Management. *American Journal of Hematology*, **96**, 872-888. <https://doi.org/10.1002/ajh.26240>