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A Case of Polyarteritis Nodosa with Positive Anti-phospholipid Antibodies Presenting with Multifocal Myositis

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Abstract

Background: Poly Arteritis Nodosa (PAN) is a rarely necrotizing vasculitis in childhood and is characterized by the inflammation of small and medium vessels affecting multiple organs. Although the presence of Anti Phosphor Lipid (APL) antibodies in PAN has been documented, there is limited data on the prevalence and understanding of pathogenesis and management for such co-incidence.

Case study: We herein reported that a 7-year-old boy without significant past medical history presented initially with high-grade fever, painful subcutaneous nodules, and ankle arthritis for 14 days prior to hospitalization. Biological findings revealed increased acute inflammatory biomarkers and no evidence of infection. The deep skin biopsy taken from the subcutaneous nodule proved leukocytoclastic medium-sized vasculitis suggestive of the PAN diagnosis. He eventually achieved a complete response with the use of intravenous corticosteroids (2 mg/kg/day) and subcutaneous methotrexate (15 mg/m^2 body surface area (BSA)/week). However, the patient developed painfulness of bilateral proximal muscles and new-onset subcutaneous nodules of lower limbs after 1-week maintenance. Though the Creatine Phospho Kinase (CPK) level was normal, the Magnetic Resonant Imaging (MRI) showed multiple foci myositis of bilateral gluteal and femur regions. The serum level of D-dimer was remarkably elevated, and the lupus anticoagulant was positive. A combination of subcutaneous enoxaparin and pulsed cyclophosphamide (500 mg/m^2 BSA) and three-day methylprednisolone (30 mg/kg/day) have contributed to a favourable outcome in this case. She further sustained remission on maintenance of gradually tapering doses of oral prednisolone and methotrexate.

Conclusions: We describe a case of PAN with the presence of APL antibody manifesting with multifocal myositis. It is recommended that general testing for APL antibodies should be undergone in patients with PAN, as well as other systemic vasculitis. Despite unusual co-incidence, APL antibodies might worsen systemic vasculitis through thrombotic events, which clinicians should consider adequate coagulant therapeutics besides immunosuppressors.

Keywords: Antiphospholipid • Polyarteritis nodosa • Vasculitis • Myositis

Abbreviations: PAN: Polyarteritis Nodosa; APL: Antiphospholipid; CPK: Creatine Phosphokinase; BSA: Body Surface Area; MRI: Magnetic Resonant Imaging; DADA2: Deficiency of Adenosine Deaminase 2; ANCA: Antineutrophilic Cytoplasmic Antibody; APS: Antiphospholipid Syndrome; ESR: Erythrocyte Sedimentation Rate; WBC: White Blood Cells; NEU: Neutrophil Cells; LYM: Lymphocyte; CRP: C-Reactive Protein; LDH: Lactate Dehydrogenase; PT/INR: Prothrombin Time/International Normalized Ratio; ANA: Anti-Nuclear Antibody; dsDNA: double-stranded Deoxyribonucleic Acid; RF: Rheumatoid Factor; C3: Complement 3; C4: Complement 4; CTA: Computed Tomography Angiography; cPAN: cutaneous Polyarteritis Nodosa; aPTT: activated Partial Thromboplastin Time; dRVV: dilute Russell Venom Viper Test

Introduction

Polyarteritis Nodosa (PAN) is a necrotizing, focal segmental vasculitis predominantly affecting medium-sized arteries without glomerulonephritis or vasculitis in arterioles, venules, or capillaries [1]. The incidence of PAN is around 0.9-8.0 cases per million in European countries and peaks at the ages of 40 to 50, with a male-to-female ratio of 2:1 [2-4]. Although epidemiological data on childhood-onset PAN is scarce, the disease is known to be comparatively rare and related to a newly discovered monogenic disease, Deficiency of Adenosine Deaminase 2 (DADA2) in children [5,6].

PAN can present a broad spectrum of manifestations with multiple organ systems, including the skin, gastrointestinal tract, kidney, nervous system, and muscle [7,8]. With spare lesions of the lung, the disease most commonly targets the peripheral nervous system and the skin and is distinctive to the Antineutrophilic Cytoplasmic Antibody (ANCA)-associated [9,10]. Musculoskeletal involvement in PAN may present in a limited form or a component of a systemic entity, including myalgia, arthralgia, polymyositis-like syndrome, asymmetric nondeforming polyarthritis, acute leg ischemia, or myopathy [11]. The pathophysiology of muscular manifestations in PAN might be ischemia due to compromised intramuscular arteries, neuropathy of the peripheral nervous system, or, more rarely, myositis [12,13]. Myositis in PAN has been described as a diffuse or focal form, which might suggest the underlying mechanisms of the lesion [7,8,14].

Antiphospholipid Syndrome (APS) is an autoimmune disease characterized by the presence of antiphospholipid antibodies, including lupus anticoagulant, anticardiolipin antibodies, and anti-β2-glycoprotein-1 antibodies [15]. The hallmark pathophysiology of APS is thrombosis, inducing a variety of clinical phenotypes, including occlusion in the veins, arteries, and microvasculature, as well as obstetrical complications [15,16]. The rare coexistence of the antiphospholipid syndrome and systemic vasculitis, such

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as Polyarteritis Nodosa (PAN), giant cell arteritis, Takayasu's arteritis, and Behcet's syndrome, has been documented in the literature [17]. The presence of APL antibodies in vasculitis might be hypothesized as the endothelial cell disruption occurring in vasculitis, which presents immunologically hidden antigens and stimulates anti-endothelial antibodies [17]. The coincidence of PAN and APS has also been described mainly just case by case. The severity of illness with both conditions might be more acute and organ-threatening, suggesting the resonant risk of occlusive vasculopathy associated with APL antibodies [17,18]. Besides the mainstay of immunosuppressors, therapeutic dilemmas with prophylactic anticoagulation for preventing thrombosis should be considered in such cases.

Here, we describe a case of PAN with the presence of APL antibody manifesting with multifocal myositis. This report contributes to the current pediatric literature on the existence of APL antibodies in PAN and highlights the need for further investigations into this issue. It is recommended that general testing for APL antibodies should be undergone in patients with PAN, as well as other systemic vasculitis. Despite unusual co-incidence, APL antibodies might worsen systemic vasculitis through thrombotic events, which clinicians should consider adequate coagulant therapeutics besides immunosuppressors.

Case Presentation

A previously healthy 7-year-old boy suffered from a high-grade fever (39 °C), painful subcutaneous nodules, and bilateral ankle arthritis for 14 days prior to hospitalization. His parents denied any history of purpura, Raynaud's phenomenon, oral ulcers, and hair loss. With suspected cutaneous infection, he was put on a seven-day-course oral oxacillin, but the symptoms were not resolved.

On initial examination at admission, the patient appeared to have normal vital signs, with a blood pressure of 115/65 mmHg and a heart rate of 95 beats per minute. He had good air entry to both lungs and no crackle on respiratory examination. Multiple palpable subcutaneous nodules are predominantly positioned over both the lower limbs. Hepatosplenomegaly sign or lymph node enlargement and livedo reticularis were absent.

Initial blood tests revealed a remarkable elevation in inflammatory markers, including Erythrocyte Sedimentation Rate (ESR), 93 mm/1st hour and 101 mm/2nd hour (ref. 3-13); White Blood Cells (WBC), 29,000 cells/μl (ref. 4-10); Neutrophil Cells (NEU), 24,000 cells/μl (ref. 4-10); Lymphocyte (LYM) 3260 cells/μl (ref. 4-10); C-Reactive Protein (CRP), 120 mg/L (ref. <6); Lactate Dehydrogenase (LDH), 300 U/L (ref. <266 U/L); and ferritin, 214 ng/mL (ref. <140).

The cogulation profile presented as following

Prothrombin Time/International Normalized Ratio (PT/INR), 1.19 (ref. 0.87-1.2); fibrinogen, 4.79 g/L (ref. 1.89-4.75); Activated Partial Thromboplastin Time (aPTT), 38 seconds, (ref. 25.1-36.5); D-dimer, 788 ng/mL (ref. <500); protein C, 100.5 UI/mL (ref. 45.9-153.5); protein S, 110 UI/mL (ref. 66.5-161.5); and antithrombin III, 97 UI/mL (ref. 64.2-136.4). Other laboratory investigations were insignificant, as described in (Table 1).

The immunology profile taken on admission was unremarkable, with Anti-Nuclear Antibody (ANA), double-stranded Deoxyribo Nucleic Acid (dsDNA), Rheumatoid Factor (RF), anti-streptolysin O, and Anti-Neutrophil Cytoplasmic Antibody (ANCA) were all negative. Complement C3 (1.7 g/L; ref. 0.9-1.8) and C4 (0.4 g/L; ref. 0.1-0.4) and IgG-4 (83 mg/dL; ref. 0.4-99.2) were in the normal range, while serum gammaglobulinemia (19 g/L; ref. 5.5-11.5) was increased. His microbiology investigations failed to detect mycoplasma pneumonia, tuberculosis, B hepatitis virus, Epstein-Barr virus, cytomegalovirus, human herpesvirus-6, human immunodeficiency virus, adenovirus, influenza virus, and coronavirus. The nasopharyngeal and blood cultures showed no growth of bacteria. Because of persistent leukocytosis, the bone marrow aspiration was obtained to exclude the malignant condition.

With the clinical suspicion of PAN, the deep incisional skin biopsy taken from the subcutaneous nodule revealed leukocytoclastic and necrotizing vasculitis of the medium-sized arteries, particularly at the branch points. No malignant characteristics were seen (Figure 1).

The Doppler ultrasonography and Computed Tomography Angiography (CTA) performed on general vessels showed normal structure and no thrombosis. The urinary analysis and peripheral electromyography of both upper and lower limbs confirmed spare lesions of kidney and nervous system. The child was diagnosed with cutaneous Polyarteritis Nodosa (cPAN) and was initially managed by intravenous corticosteroids (2 mg/kg/day) and subcutaneous methotrexate (15 mg/m^2 BSA/week). He eventually achieved a complete response regarding clinical signs and inflammatory markers and was switched to oral corticosteroid and methotrexate on day 7 for discharge.

However, the patient presented to us again after 1-week maintenance with complaints of bilateral muscular painfulness and new-onset subcutaneous nodules of lower limbs. No other organ was involved by clinical examination, but the laboratory findings showed elevated inflammatory markers (see Table 1). Though CPK level and repeated electromyography were normal, MRI with the contrast agent showed multiple foci myositis of bilateral gluteal and femur regions (Figure 2).

Table 1. Laboratory results.

Parameters	Admission 1	Day 3	Day 7	Admission 2	Day 5	Discharge
ESR (mm/h)	93/101	54/82	61/101	88/102	40/70	45/78
WBC (cells/μ)	29,000	13,600	12,000	20,500	21,000	15,410
NEU (cells/μ)	24,000	8,900	7,774	15,700	16,400	11,600
LYM (cells/μ)	3,260	3,200	3,600	3,400	3,800	3,450
PLT (× 103 cells/μ)	565	651	455	468	600	556
Ferritin (ng/mL)	214	194	205	245	213	196
LDH (UI/mL)	300	302	312	280	272	267
IL-6 (pg/mL)	5.34	14.5	N.A	N.A	N.A	N.A
D-dimer (ngFEU/mL)	788	650	675	2450	902	602
CRP (mg/L)	120	22.8	34.7	92	38	2.8
CK (UI/mL)	15	N.A	N.A	31.2	9.4	13.3

ESR: Erythrocyte Sedimentation Rate, WBC: White Blood Cell, NEU: Neutrophils, LYM: Lymphocyte, PLT: Platelets, LDH: Lactate Dehydrogenase, IL-6: Interleukin-6, CRP: C-Reactive Protein, CK: Creatine Kinase and N.A: Not Applicable

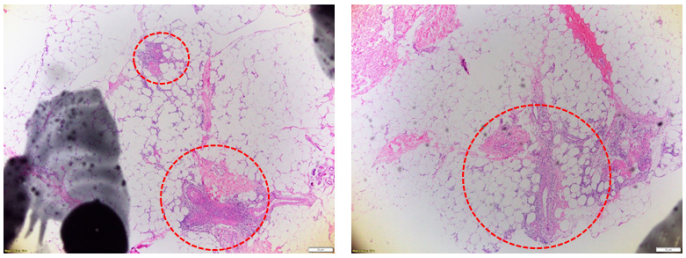

Figure 1. The deep dermis and subcutaneous tissue showed neutrophilic infiltration with prominent debris around medium-sized vessels and branch points of vessels. Fibrinoid necrosis within vessel walls, endothelial swelling, and thrombi are present. Granulomatous inflammation and neoplastic lesions were absent (40X).

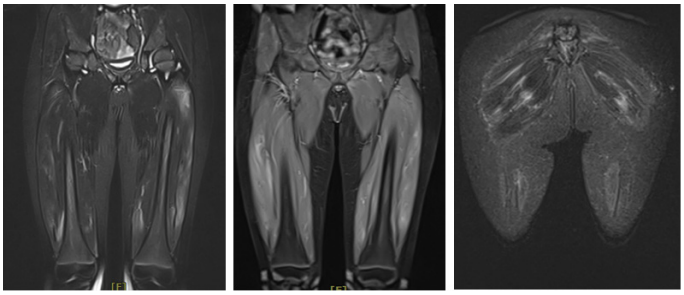

Figure 2. MRI with the contrast agent showed multiple foci myositis of bilateral gluteal and femur regions.

Histological image of muscular biopsy presented neutrophil infiltration, debris, and endothelial swelling within a muscular artery wall. Occlusion and fibrinoid necrosis within damaged small-sized blood vessels were noted (Figure 3).

We checked the panel of the 18 myositis-specific antibodies that showed negative. Along with focal lesions of myositis and blocking of blood vessels as described, an increase in fibrinogen (6.79 g/L; ref. 1.89-4.75), D-dimer (2450 ng/mL; ref. <500), and aPTT (42 seconds, ref. 25.1-36.5) were found. Assessing the APL antibodies profile exposed that the lupus anticoagulant was positive. This test was performed by an activated partial thromboplastin time assay, a dilute Russell Venom Viper Test (dRVVT), and confirmatory testing.

In conjunction with pulsed cyclophosphamide (500 mg/m^2 BSA) and intravenous corticosteroid of 30 mg/kg/day for 3-consecutive days, the patient received subcutaneous enoxaparin doses of 1 mg/kg every 12 hours. His symptoms and laboratory findings completely resolved after the end of the therapy course (Table 1). Switching to aspirin, gradually tapering oral corticosteroid doses, and maintenance on methotrexate were given based on the above result. After 15 days of admission, he was discharged home and required regular follow-up in our outpatient clinic.

Results

Poly Arteritis Nodosa (PAN) is a rarely necrotizing vasculitis in childhood and is marked by the predominant inflammation of medium vessels affecting multiple organs. Antiphos Pholipid Syndrome (APS) is an autoimmune disease characterized by the presence of APL antibodies in the bloodstream, showing the risk of multi-organ involvement-induced thrombosis [15,16]. The previous papers have described the co-incidence of APL antibodies and PAN with worse outcomes; however, there is only a limited number of case reports [18,19]. Here, we describe a case of PAN with the presence of APL antibody manifesting with multifocal myositis. This report contributes to the current pediatric literature on the existence of APL antibodies in PAN. It highlights the need for further investigations into pathophysiology insights and disease management.

Any organ containing small or medium arteries can be involved in PAN [20]. According to the literature, the most commonly affected organs are the skin, gastrointestinal tract, kidneys, peripheral nerves, and muscles [5,9,12]. Skeletal muscle manifestations are not uncommon in PAN, with a rate varying from 30% to 70% [20]. Musculoskeletal involvement in PAN may present in a limited form or a component of a systemic entity, including myalgia, arthralgia, polymyositis-like syndrome, asymmetric nondeforming polyarthritis, acute leg ischemia, or myopathy [11]. Among them, myalgia has been well documented in about 51% of PAN cases, while myositis is a rare form of scenario [12]. The high incidence of myalgia is in line with the common involvement of intramuscular arteries and the peripheral nervous system in PAN [7,12]. Patients with myositis-associated PAN usually present with symmetrical, proximal muscle weakness prominently in the lower extremities [7,13,14,20,21].

The probable underlying pathophysiology of myositis in PAN can be explained by an overwhelming immune-inflammatory infiltration and ischemia due to the occlusion of blood vessels supplying the muscles [13,14]. In the presenting case, muscular fiber necrosis, mixed leuko-infiltration, and occlusion of small-sized blood vessels were presented histologically, in line with the suspected theory. The normal CKP level presents one peculiarity in our report. Elevated CPK levels account for only 2% to 5% of cases, while elevated LDH and aldolase have been reported in some cases [13,22]. The popularity of a normal CPK level can overshadow a suspicion of myositis, for which we suggested the pivotal role of muscular MRI or biopsy in evaluating the lesion. Myositis in PAN has been described as a diffuse or focal form, which might suggest the underlying mechanisms of the lesion [7,8,14].

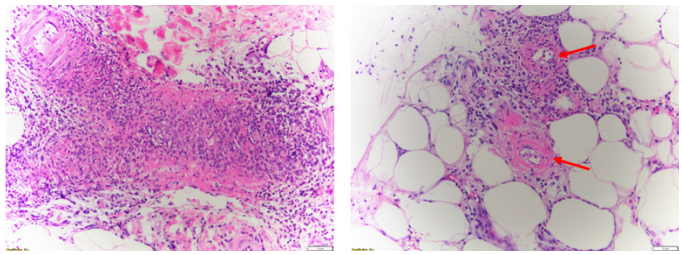

Figure 3. Histological image of muscular biopsy presented neutrophil infiltration, debris, and endothelial swelling within a muscular artery wall. Occlusion and fibrinoid necrosis within damaged small-sized blood vessels were noted (40X).

Discussion

Antiphos Pholipid Syndrome (APS) is an autoimmune disease characterized by the presence of antiphospholipid antibodies, including lupus anticoagulant, anticardiolipin antibodies, and anti-β2-glycoprotein-1 antibodies [15]. The literature mentions the rare coexistence of the antiphospholipid syndrome and systemic vasculitis, such as Poly Arteritis Nodosa (PAN), Giant Cell Arteritis (GCA), Takayasu's arteritis, and Behcet's syndrome [17]. The presence of APL antibodies in vasculitis might be hypothesized as the endothelial cell disruption occurring in vasculitis, which presents immunologically hidden antigens and stimulates anti-endothelial antibodies [17]. Therefore, we emphasize that general testing for APL antibodies should be undergone in patients with PAN, as well as other systemic vasculitis. The severity of illness with both conditions might be more acute and organ-threatening, suggesting the resonant risk of occlusive vasculopathy associated with the presence of APL antibodies [17,18]. In our case, the existence of APL may account for the occlusion of small-sized blood vessels in the muscular biopsy, in line with focal rather than diffuse myositis in the illustrated MRI. Nung-Hoang, et al. reported a PAN case with APL positivity that presented as acute deterioration of renal function and then responded well to immunosuppression and anticoagulation combination [23]. Similarly, some case reports described central nervous system damage in PAN with the high anticardiolipin antibody [24,25]. While many reports reinforce the critical role of APL in worsening thrombosis events [2,18,26], the intriguing question is whether clinical outcomes are favorable if anticoagulation therapy is administered. This point underlines the difficulties in clinical decision-making regarding the anticoagulation options and duration in managing this complex situation.

According to the pathophysiology, we accentuated the need for integrative therapy in PAN cases with APL antibodies coexistence, including immunosuppression to limit muscular damage in the inflammatory response and anticoagulation therapy to reduce further lesions due to thrombosis events. However, there remains not enough evidence-based data on treatment and outcome of this picture; further well-designed investigations on this issue are necessary.

Conclusion

We describe a case of PAN with the presence of APL antibody manifesting with multifocal myositis. In conclusion, it is recommended that general testing for APL antibodies should be undergone in patients with PAN, as well as other systemic vasculitis. Despite unusual co-incidence, APL antibodies might worsen systemic vasculitis through thrombotic events, which clinicians should consider adequate coagulant therapeutics besides immunosuppressors.

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Author Contributions

HTTN, AVTN, and CQL provided the concept for the study and were major contributors to the manuscript revision. GDN and HTTN were involved in the patient care and collected the data regarding the patient's history, clinical course, and the trends in vital parameters. TNH collected, prepared, and

researched the biopsy samples. HTTN reviewed the literature and drafted the manuscript. All authors read and approved the final manuscript.

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Availability of Data and Materials

The datasets used during the current study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

The study complied with the Declaration of Helsinki and was approved by the Institutional Review Board of Vietnam National Children's Hospital. Written informed consent to participate in this study was provided by the participant's legal guardian/next of kin.

Consent for Publication

Informed written consent for patient information and images to be published was obtained from the participant's legal guardian/next of kin.

Competing Interests

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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