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Physical Activity, Rehabilitation and Health Education in High-Risk Patients: Lessons from Vascular Ehlers–Danlos Syndrome

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Abstract

Background. Physical activity is a fundamental component of health, prevention, and education; however, in selected high-risk populations, safe exercise prescription remains a major challenge. Vascular Ehlers–Danlos syndrome (vEDS) represents an extreme model of exercise-related risk due to marked vascular fragility.

Aim. The aim of this study was to analyse the safety of physical activity in vEDS and to provide practical implications for sport participation, rehabilitation, and health education.

Material and methods. A narrative review of available literature was performed, focusing on exercise- and sport-related complications, rehabilitation practices, and current recommendations for physical activity in patients with vEDS.

Results. Analysis of reported cases shows that both competitive sports and low-intensity activities, including standard rehabilitation exercises, may trigger severe and potentially fatal vascular events. At the same time, appropriate low-load, supervised physical activity may support functional capacity and quality of life. Current recommendations are inconsistent and largely based on expert opinion, leading to uncertainty among patients and professionals responsible for exercise guidance.

Conclusions. The findings highlight the need for a structured, education-based approach to physical activity in high-risk populations. In vEDS, exercise prescription should prioritise safety, individualisation, and professional supervision. The development of clear guidelines and improved education of clinicians, physiotherapists, and sport practitioners is essential to ensure safe participation in physical activity while minimising the risk of serious complications.

Keywords: physical activity; exercise safety; rehabilitation; patient education, vascular ehlers–danlos syndrome

Content

1. Overview of Ehlers–Danlos Syndrome

Ehlers–Danlos syndromes (EDS) consist of a diverse group of inherited connective tissue disorders, with a combined prevalence estimated at roughly 1 in 5,000; however, recent evidence suggests that milder and hypermobile forms are likely underdiagnosed in the general population [1,2]. EDS is linked to pathogenic variants in at least 19 causal genes that primarily influence the synthesis and organization of collagen fibrils and other extracellular matrix components [1]. These molecular defects weaken connective tissue integrity across multiple organ systems, contributing to the clinical heterogeneity observed in EDS. Clinical manifestations span from mild joint and skin manifestations to severe, life-threatening complications affecting vital organs.

According to the 2017 international classification, EDS comprise 13 distinct heritable connective tissue disorder subtypes, each defined by characteristic clinical features and, in most cases, a causative genetic defect; these include: classical, classical-like, cardiac-valvular,

vascular, hypermobile, arthrochalasia, dermatosparaxis, kyphoscoliotic, brittle cornea syndrome, spondylodysplastic, musculocontractural, myopathic, and periodontal EDS [3]

Vascular Ehlers–Danlos syndrome (vEDS), that accounts for roughly 5% of all EDS cases, is the most severe subtype and is associated with a markedly increased risk of spontaneous and exercise-related vascular injury [4]. Unlike traumatic vascular events that typically occur in the setting of high-energy sports or major accidents, vascular complications in vEDS frequently arise after minimal physical stress or in the absence of identifiable trauma [3]. vEDS is caused by heterozygous pathogenic variants in the COL3A1 gene, which encodes the alpha 1 chain of type III collagen, a critical extra cellural matrix component of hollow organs and arterial walls [5]. Mutations in COL3A1 reduce the tensile strength of the vessel wall, leading to a high risk of arterial rupture, dissection, and aneurysm formation, and also contribute to fragility of other collagen-rich tissues, increasing the risk of spontaneous bowel and uterine rupture.

Epidemiological data underscore the substantial vascular burden and early onset of complications in vEDS. Its prognosis is poor because affected individuals are prone to complications early in life, sometimes presenting before the age of 20. Overall, the median life expectancy for patients with vEDS ranges between 40 and 50 years [4]. Approximately half of individuals with vEDS develop a major arterial complication during their lifetime, most commonly presenting in the fourth or fifth decade of life. Arterial aneurysms or dissections account for a substantial proportion of these events. Notably, more than 40% of patients experience recurrent vascular complications, emphasizing the progressive and cumulative nature of vascular fragility in this disorder. [6] Vascular injuries most commonly involve the abdominal, pelvic, and cerebrovascular arterial beds. Mortality remains high, with arterial rupture representing the leading cause of death, often occurring suddenly and without warning. Cerebrovascular events, including carotid artery dissection and intracranial aneurysm rupture, are also disproportionately frequent, contributing significantly to morbidity and fatal outcomes [6].

Despite increasing recognition of spontaneous vascular events in vEDS, the role of physical activity, sports participation, and exercise-related mechanical stress as potential triggers of vascular injury remains insufficiently characterized. While physical exercise is widely promoted for cardiovascular health in the general population, the abnormal

biomechanical properties of connective tissue in vEDS raise critical concerns regarding the safety of both recreational and competitive sports [3]. Even low-to-moderate intensity activities may impose repetitive shear stress or temporary elevations in blood pressure on structurally compromised vessels.

In this paper, we focus on sport and exercise-related injuries in vEDS. We present clinical cases in which vascular events were directly associated with low- to high-intensity physical activity and analyze current recommendations regarding bodily activity and training in this high-risk patient population, highlighting the ongoing challenge of balancing exercise benefits with injury prevention.

2. Materials and methods

A literature search was conducted to identify studies reporting sports- and exercise-related injuries in patients with Ehlers–Danlos syndrome. Searches were performed in PubMed and Google Scholar using combinations of the keywords: “Ehlers–Danlos syndrome,” “sport,” “exercise,” “physical activity,” “trauma,” and “case report.” Inclusion criteria were: original research articles, case reports, and case series, and publications available in English with full-text access.

After excluding papers based on irrelevant titles and abstracts, our searches identified a total of 27 potentially relevant articles from PubMed and Google Scholar (Fig.1). Each record was then screened using the full text to assess relevance to sports- or exercise-related injuries in vascular Ehlers-Danlos syndrome. Following previous classifications, we also included reports related to type IV EDS. Studies that did not directly address exercise-related events were excluded. We subsequently evaluated the remaining studies to confirm eligibility and extract relevant data, including patient demographics, EDS subtype, type and severity of injury, mechanism of injury, and clinical outcomes. The final seven studies were analyzed narratively to provide an overview of the clinical characteristics, injury mechanisms, and implications for exercise and activity recommendations in this high-risk population. Table 1 presents a summary of the considered cases.

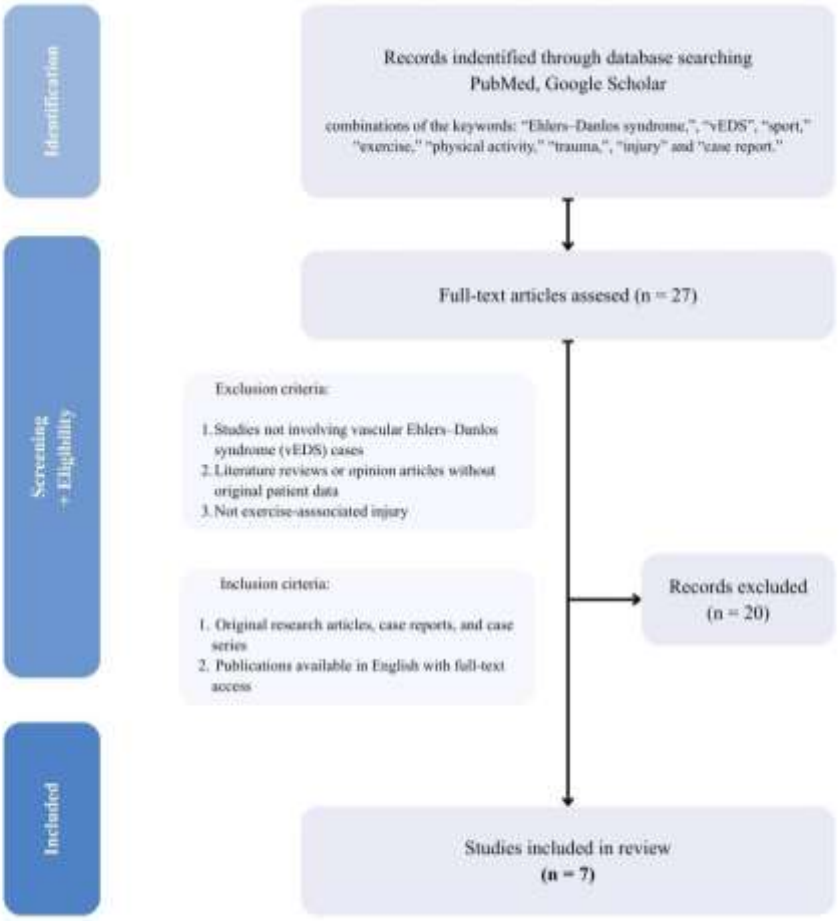

Figure 1. Flowchart of literature search and study selection. After screening titles, abstracts, and full texts, seven studies on exercise- or sport-related injuries in vascular Ehlers–Danlos syndrome were included in the final analysis.

	Principle Author	Type physical activity	Age/ Gender	Type of injury	Comorbidities	Handling of injury	Outcome	Complications	Influence on sport activity	Genetic testing	Hospitalization time	vEDS diagnose
[4]	Endo T. et al.	Playing	11 / F	Popliteal artery rupture	Tricuspid valve regurgitation (Kawasaki disease at age 1), gastric volvulus at age 2 without surgical intervention	Emergency hematoma evacuation; popliteal artery interposition with great saphenous vein graft, then polytetrafluoroethylene vascular graft	Recovered with mild intermittent claudication in left lower extremity	GSV graft rupture on postoperative day 7; early occlusion of synthetic graft	Advised to avoid sudden movements such as dashing and jumping; full recovery of knee function at 1 year follow-up	Missense variant in COL3A1 (c.3554 G>A, p.Gly1185Asp)	20 days	Not previously diagnosed with EDS, family history given as insignificant
[8]	Kalia R. et al.	High-intensity rowing exercise with energy drink consumption	19 / F	Exercise-induced myocarditis with ventricular tachycardia	Myocarditis episode	Beta-blocker therapy (metoprolol ER), bed rest, cardiac monitoring, restriction of physical activity	Clinical improvement; troponin normalization; no recurrent VT during hospitalization	Recurrent myocarditis episodes; ventricular tachycardia; myocardial edema and scarring on MRI	Restriction from high-intensity exercise; advised low-level physical activity only; indefinite beta-blocker prophylaxis	Confirmation of vEDS in history	8 days	Previously diagnosed, family history of vEDS (mother died of aortic dissection)
[9]	Maskad K. et al.	Arm stretching	12 / M	Rupture of distal right axillary artery with massive hemorrhage	No comorbidities reported	Emergency blood transfusion; CT angiography; endovascular covered stent placement; surgical evacuation of hematoma; failed bypass due to vessel fragility; above-elbow amputation	Survival with limb salvage failure	Neurological deficit due to limb ischemia; upper-limb amputation	Permanent exclusion from sports and activities involving upper-limb strain	Confirmation of vEDS in history	Not reported	Previously diagnosed, family history of EDS (mother)
[10]	Buettner D. et al.	Basketball-related fall	18 / M	Thoracic aortic dissection with massive hemothorax	No comorbidities reported	Emergency intubation, CPR, bilateral thoracotomy, attempted vessel ligation, massive transfusion	Fatal	Aortic rupture, bilateral hemothoraces, cardiac arrest	-	Confirmation of EDS in history	Not reported	Pre-existing EDS, with diagnosis disclosed after the incident; vEDS confirmed postmortem
[11]	Rossi P. et al.	Cervical hyperextension during wrestling	16 / M	Pseudoaneurysm of proximal right subclavian artery	Smoking; history of epistaxis; prior superficial femoral artery injury do associated with vEDS	Medical stabilization with atenolol and analgesia; surgical repair with aortocarotid bypass using Dacron grafts under continuous neurologic monitoring	Successful vascular reconstruction; patient discharged on postoperative day 5	Arterial friability, risk of recurrent vascular complications	Avoidance of contact sports and heavy exercise	De novo G52C COL3A1 mutation	Not reported	Not diagnosed previously, no family history
[12]	Wimmer P. et al.	Playing basketball	15 / M	Transected right subclavian artery, transected right vertebral artery, lacerated brachiocephalic vein, massive cervical mediastinal hematoma	History of multiple hip surgeries, club feet, easy bruising, joint instability; previously undiagnosed EDS	Emergency intubation, massive transfusion, median sternotomy with attempted vascular repair; ultimately operative site packed	Fatal	Extreme vascular friability, uncontrollable bleeding, retroperitoneal hematomas, dissecting aortic aneurysm	-	Histologic findings were strongly suggestive of EDS type IV	Not reported	Not diagnosed previously, no family history
[13]	Iriuna R. et al.	Passive hamstring stretching during rehabilitation	46 / M	Intramuscular muscle hematoma (biceps femoris & semitendinosus)	Prior stroke with spasticity; hypertension; arterial dissection; on antiplatelet therapy	Discontinue clopidogrel, rest, compression, hemostatic measures	Hematoma resolved within 5 days, discharged home after 1 month	Intramuscular muscle hemorrhage; progressive anemia (improved after stopping antiplatelet)	Rehabilitation and stretching protocols modified to avoid similar injury; caution advised in vEDS	COL3A1 mutation	1 month	Not diagnosed previously, no family history

Table 1. Physical activity-related injuries reported in patients with vascular Ehlers–Danlos syndrome (vEDS). The table summarizes patient characteristics, type of physical activity, injury type, management, outcomes, complications, impact on sport participation, genetic findings, hospitalization time, and timing of vEDS diagnosis. Severe vascular events occurred across a wide range of activities, frequently in patients without a prior vEDS diagnosis.
Abbreviations: vEDS, vascular Ehlers–Danlos syndrome; EDS, Ehlers–Danlos syndrome; GSV, great saphenous vein; VT, ventricular tachycardia; CPR, cardiopulmonary resuscitation; MRI, magnetic resonance imaging; CT, computed tomography.

3. Clinical Case Overview

Endo et al. [4] reported the first case of a ruptured popliteal artery aneurysm in a pediatric patient. An 11-year-old girl experienced the rupture while playing, initially requiring emergency repair with a great saphenous vein graft, which failed on postoperative day 7 due to graft fragility. A second procedure used a synthetic graft, which occluded early, but the patient ultimately recovered with only mild intermittent claudication and full knee mobility at one year. The patient was instructed to avoid sudden or forceful movements, such as sprinting or jumping. The patient was not diagnosed with vEDS at the moment of the accident. Later genetic testing confirmed a COL3A1 mutation. Popliteal artery complications are rare in vEDS, reported in only ~3% of cases [7]

Kalia et al. [8] described a presentation of vEDS in a 19-year-old female student-athlete who developed exercise-induced myocarditis. Following intense physical exercises on the rowing machine, she presented with chest pain, elevated cardiac biomarkers, and ventricular tachycardia. Cardiac MRI demonstrated myocardial edema and non-ischemic late gadolinium enhancement consistent with myocarditis, without evidence of arterial dissection. Management consisted of beta-blockers pharmacotherapy, bed rest, and restriction of high level of physical activity for the next six weeks. After a recurrent episode of exercise-associated myocarditis, the patient was advised to permanently avoid high-intensity physical exertion. Notably, the patient had experienced a similar exercise-associated episode previously and had a strong family history of vEDS-related vascular complications. This case highlights myocarditis as an uncommon and atypical manifestation of exercise-related cardiovascular involvement in vEDS.

Mankad et al. [9] reported a severe vascular complication in a 12-year-old boy with vEDS following minimal physical activity. The patient developed progressive pain and swelling of the right upper limb and chest wall after casual arm stretching. Imaging revealed active hemorrhage from a tear in the distal right axillary artery, resulting in hemodynamic instability, neurologic deficits, and loss of distal pulses. Emergency endovascular stenting was performed, followed by surgical evacuation of a large hematoma; however, due to extreme vessel fragility and nonviable forearm musculature, revascularization was unsuccessful and an above-elbow amputation was required. Permanent exclusion from sports and activities involving the upper-limb was advised.

Buettner et. al [10] reported a fatal vascular complication in an 18-year-old man following minor trauma during recreational basketball. The patient fell backward and struck his head, immediately losing consciousness and developing seizures, followed by rapid hemodynamic deterioration. Despite the absence of external hemorrhage, imaging and emergent surgical exploration revealed massive intrathoracic bleeding, which resulted in the patient's death. Autopsy confirmed a thoracic aortic dissection with bilateral hemothoraces and a large retroperitoneal hematoma. Postmortem evaluation confirmed vEDS using physical assessment and family-provided history.

Rossi et al. [11] described a 16-year-old male who developed a large right subclavian artery pseudoaneurysm following a cervical hyperextension injury sustained during wrestling. He presented with progressive neck and upper back pain, ulnar-sided paresthesias, and Horner's syndrome. Imaging revealed a pseudoaneurysm arising from the origin of the right subclavian artery, which was surgically repaired using prosthetic bypass grafting. Notably, the patient had a prior history of traumatic femoral artery injury but no known connective tissue disorder at presentation. Subsequent analyses confirmed vEDS due to a de novo COL3A1 mutation. This case highlights trauma-associated arterial rupture as an atypical initial manifestation of vEDS, with diagnosis established only after recurrent vascular complications.

Wimmer et al. [12] reported a fatal vascular catastrophe in a 15-year-old male with previously undiagnosed vEDS. The patient initially presented with right shoulder pain after feeling a "pop" while playing basketball. Examination revealed a clavicular hematoma, dysmorphic facial features, along with a history of easy bruising and multiple orthopedic surgeries for hip and foot deformities. While undergoing radiologic evaluation, he acutely deteriorated with rapid expansion of the hematoma and loss of pulses in the right upper extremity. Emergency surgery revealed transection of the right subclavian and vertebral arteries, laceration of the brachiocephalic vein, and extensive hemorrhage. Despite aggressive resuscitation and operative interventions, the patient ultimately passed away from hypovolemic shock.

Izumi et al. [13] reported the first documented case of intramuscular hemorrhage in spastic muscles during post-stroke rehabilitation in a patient with vEDS. The patient, a 46-year-old male with a family history of subarachnoid hemorrhage and aortic dissection, developed hematomas in the biceps femoris and semitendinosus muscles during routine passive hamstring

stretching. Conservative management—including discontinuation of antiplatelet therapy, rest, compression, and hemostatic agents—led to resolution within five days, and he regained independent ambulation. Subsequent genetic testing confirmed a pathogenic COL3A1 variant. Conservative management with rest, compression, and hemostatic measures led to full recovery within five days, but activity restrictions and modified rehabilitation protocols were necessary to prevent recurrence.

4. Analysis of Reported Cases

In the majority of cases we reviewed, exercise-related complications in vEDS occurred in adolescents and young adults, with a median age of 16 years and six of seven patients under 20. Males predominated in our series (6/7, 86%), consistent with previous studies indicating that serious vascular complications in vEDS occur more frequently and at a younger age in men [14].

The spectrum of triggering activities ranged from high-intensity or contact sports such as basketball, wrestling, and rowing to seemingly minimal trauma including casual arm stretching, general play, and passive rehabilitation exercises, demonstrating that even low-impact movements can cause severe complications in vEDS.

Arterial rupture or dissection was the most frequent and severe manifestation, observed in five cases, involving major vessels including the subclavian, axillary, popliteal arteries, and the thoracic aorta. Other presentations included intramuscular hemorrhage in a spastic muscle following stroke and exercise-induced myocarditis. These events were highly dangerous: two patients died despite intensive resuscitation and surgical interventions, one survivor suffered permanent disability (above-elbow amputation), and four patients recovered, with any remaining deficits correlated to other incidents rather than the events described.

Emergency surgical intervention was required in the majority of cases (6/7, 86%), emphasizing the need for meticulous perioperative planning due to tissue fragility. In one adult case with intramuscular hemorrhage, conservative management sufficed, illustrating that severity and management strategies vary depending on the type of injury.

A family history or prior vEDS diagnosis was documented in three cases, yet four patients (57%) were previously undiagnosed, including one fatality. In one instance, the

diagnosis was disclosed to medical personnel only after the acute event, highlighting the importance of early recognition.

Timely diagnosis and genetic confirmation of vEDS are essential to guide preventive counseling and inform individualized emergency planning. Recommendations for physical activity should be carefully tailored, emphasizing avoidance of maneuvers that impose stress on structurally fragile vessels. Multidisciplinary coordination and readiness for emergent interventions are crucial to optimize outcomes when injury occurs. Education of patients and families, particularly in pediatric and adolescent populations, is imperative, as even seemingly minor trauma can result in catastrophic events.

These findings underscore a critical clinical question: how can practitioners safely balance the benefits of physical activity with the heightened risk of vascular injury in vEDS, especially during rehabilitation or routine exercise in vEDS patients.

5. Management of Vascular Ehlers–Danlos Syndrome

Although most vascular complications in vascular Ehlers–Danlos syndrome occur spontaneously, the cases reviewed here highlight physical activity as a potential precipitating factor in a subset of events. These observations inform the following discussion on management strategies aimed at minimizing activity-related risk while preserving functional capacity and quality of life.

Management of vascular Ehlers–Danlos syndrome (vEDS) is multifaceted, addressing both the prevention of vascular complications and the mitigation of musculoskeletal, fatigue-related, and psychosocial burdens. Pharmacological therapy is primarily focused on controlling hemodynamic stress. Beta-blockers and angiotensin receptor blockers are commonly prescribed to reduce aortic wall stress and lower the risk of aneurysm formation or dissection. While precise treatment rates vary across cohorts, observational studies report that among vEDS patients treated with antihypertensive therapy, more than 80% receive beta-blockers, including celiprolol in a substantial proportion, reflecting their central role in current management strategies [14,15]. Some individuals also require additional agents, including calcium channel blockers or angiotensin-converting enzyme inhibitors, to achieve optimal blood pressure control, particularly in the context of progressive aortic dilation. However, the UK national

diagnostic service cohort highlighted that pharmacological management in vEDS is largely based on clinical experience, with limited evidence supporting the use of drugs to prevent vascular events [16].

Pain, fatigue, and psychosocial distress are major contributors to the disease burden in vEDS. High levels of fatigue, as measured by the Fatigue Severity Scale, are reported by 44% of patients with vEDS. Moreover, only about one-third of patients report no musculoskeletal pain, underscoring the substantial impact of these symptoms on daily functioning and quality of life [17].

Anxiety and depression are common contributors to disease burden in vEDS and related hereditary connective tissue disorders with vascular complications (HDCTv). In cohorts of patients with HDCTv, anxiety and depression scores measured using the Hospital Anxiety and Depression Scale (HADS) are frequently elevated, consistent with clinically relevant psychological distress. [17,18]. Patients adopt diverse coping strategies, including pacing of daily activities, prioritization of low-impact mobility, and avoidance of high-risk situations. Qualitative studies show that physical activity represents a major source of uncertainty and anxiety for patients with heritable thoracic aortic disease, including vEDS. Difficulty distinguishing safe from dangerous exercise often leads to excessive activity avoidance, highlighting the need for clearer guidance and targeted rehabilitation strategies to reduce fear and stress related to movement [18].

Surgical and interventional treatment in vEDS is reserved for selected cases. Many arterial dissections are self-limiting and managed conservatively with pain control and strict blood pressure regulation, whereas arterial rupture requires urgent intervention, most often embolization or targeted repair [19]. Planned aneurysm repair offers the safest surgical context, but open procedures are avoided when possible due to extreme vessel fragility. Acute management prioritizes permissive hypotension, cautious fluid use, non-invasive imaging, and multidisciplinary expertise to reduce secondary vascular injury [19]. Post-intervention care integrates pharmacological therapy, cautious regulation of physical activity, and ongoing cardiovascular surveillance, with multidisciplinary follow-up involving cardiology, genetics, and rehabilitation specialists to optimize outcomes and individualized counseling.

6. Activity and Rehabilitation in vEDS

In addition to pharmacologic therapy, lifestyle modification is fundamental for vEDS management. Patients are advised to avoid activities associated with abrupt increases in intrathoracic pressure or transient blood pressure surges, such as heavy lifting, high-intensity sports, and isometric exercises [15,16,20]. Daily routines should prioritize low-stress, low-impact activities that maintain mobility and cardiovascular health without imposing significant mechanical or hemodynamic strain. Across studies, adherence to these recommendations appears variable. In one survey, 88% of patients reported receiving advice regarding physical activity restrictions, and 77% reported modifying their activity habits accordingly [21]. In the same cohort, only half of patients demonstrated physical activity levels considered appropriate, whereas 40% exhibited low activity levels and 10% engaged in excessively high-intensity activity despite medical advice. Notably, higher physical activity levels were associated with lower fatigue severity and lower anxiety scores, suggesting that some patients may intentionally exceed recommended limits to mitigate fatigue or psychological distress. At the same time, approximately one-third of participants reported unmet rehabilitation needs.

Acceptable aerobic activities include walking at a conversational pace, gentle cycling, and swimming in a safe, supervised environment [15,16,19]. In contrast, high-intensity interval training, competitive sports, sprinting, and endurance events are discouraged due to the associated risk of transient blood pressure surges, which can precipitate aneurysm expansion or dissection [15,16]. Strength training requires particular caution. In most cohorts, conventional resistance training is contraindicated, especially when involving heavy loads, high repetitions with strain, or isometric contractions. If muscle strengthening is indicated, programs should use very low resistance with high repetition counts under the supervision of clinicians familiar with connective tissue disorders [15,16]. Flexibility exercises should also be controlled, avoiding end-range loading or ballistic movements, and focusing on slow, pain-free, functional range-of-motion training [18,19].

Emerging evidence suggests that structured, low-load rehabilitation programs with supervised aerobic activity, posture and balance training, and gentle mobility exercises may provide benefits without inducing adverse vascular events [16,19]. However, standardized, evidence-based exercise and rehabilitation protocols for vEDS are largely absent, and research is urgently needed to evaluate the effects of structured, multidisciplinary programs on clinical

outcomes, including aortic integrity, cardiovascular events, fatigue, and quality of life [16,19]. This knowledge gap represents a critical area for future clinical research and the development of guideline-based patient care.

7. Future Directions

While pharmacological management, including beta-blockers and angiotensin receptor blockers, remains the mainstay for vascular Ehlers–Danlos syndrome (vEDS), evidence for non-medical interventions is limited. Research priorities include structured exercise and rehabilitation programs, as patients frequently report inconsistent guidance and unmet needs for individualized support. A registered trial is exploring biofeedback training in vEDS (ClinicalTrials.gov: NCT05994664), reflecting emerging interest in safe, physiological stress-modulating interventions, although results are not yet available. Future studies should combine controlled trials of tailored physical activity, psychosocial support, and multidisciplinary rehabilitation with ongoing advances in translational research into disease mechanisms. Together, these approaches aim to improve functional outcomes, quality of life, and long-term safety in vEDS, while addressing the current gaps in patient-centered guidance and evidence-based lifestyle recommendations.

8. Conclusions

There is currently no causal therapy for vascular Ehlers–Danlos syndrome, and symptom-focused treatment alone remains insufficient. Management should prioritize patient education, clear clinical protocols, and multidisciplinary collaboration, aiming to meet patient expectations while reducing pain, fatigue, and anxiety. Individualized emergency planning, including a “vEDS passport” with diagnosis and management information, is essential to ensure rapid and appropriate response during acute vascular events [15]. Blood pressure control and careful surveillance of the vascular system are important preventive measures. Physical activity should be adapted to minimize the risk of vessel or organ injury, favoring low- to moderate-intensity, non-contact, dynamic exercises while avoiding high-intensity, isometric, or contact sports. There is a clear need to develop specific, evidence-based exercise protocols for patients and physiotherapists to guide safe activity and rehabilitation. Comprehensive care that integrates medical, psychological, and rehabilitative guidance, as well as supervised exercise programs, is critical to improving quality of life in this high-risk population.

Disclosure

Author's Contributions:

Conceptualization and Methodology: KR, KK

Investigation: WB, UZ, AP, JG

Resources: Not applicable.

Writing - rough preparation: KR, KK, JPe, NP, JPo

Writing review and editing: WB, UZ, AP, JG

Visualization: KR, KK, JPe

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