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Vascular malformations in children: a rare case of vascular nevus and its clinical features

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Vascular malformations in children encompass a diverse spectrum of congenital anomalies, often presenting at birth or in early childhood. Among these, vascular nevi (port-wine stains, or nevus flammeus) represent congenital capillary malformations that may lead to significant cosmetic and functional impairments.

Aim: to identify the main clinical and genetic features of a rare case of vascular nevus in a child and to determine the diagnostic criteria that allow timely differentiation of this pathology from other vascular malformations.

Clinical case. This study presents a retrospective analysis of a clinical case of vascular nevus in a 6-year-old girl. The patient exhibited congenital vascular pigmentation and progressive limb asymmetry. The patient presented with an extensive capillary malformation affecting the lumbar region, thigh, lower leg, and foot, associated with hypertrophy of the affected limb. X-ray examination confirmed a 2 cm anatomical lengthening of the right tibia with preserved growth plate function. Clinical features raised suspicion of phakomatosis, necessitating differential diagnosis with such conditions as Klippel–Trenaunay–Weber syndrome, Sturge–Weber syndrome, neurofibromatosis, and tuberous sclerosis. Genetic testing did not reveal pathogenic mutations commonly associated with phakomatoses, supporting the final diagnosis of an isolated vascular nevus with musculoskeletal involvement. The patient was prescribed a multidisciplinary treatment plan, including orthotic correction, physical therapy, and postural monitoring to prevent scoliosis progression.

Conclusions. Vascular malformations, particularly large vascular nevi, can induce disproportionate musculoskeletal growth, mimicking syndromic phakomatoses. The case highlights the necessity of integrating clinical, radiological, and genetic evaluations to ensure accurate diagnosis and tailored management. The findings emphasize the importance of long-term monitoring in children with complex vascular anomalies to optimize functional outcomes and prevent secondary orthopedic complications.

The study was carried out in accordance with the principles of the Declaration of Helsinki. The informed consent of the children's parents was obtained for the research.

No conflict of interests was declared by the authors.

Keywords: vascular malformations, vascular nevus, capillary malformations, phakomatoses, genetic analysis, limb asymmetry.

Судинні мальформації у дітей: рідкісний випадок судинного невусу та його клінічні особливості

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Судинні мальформації в дітей охоплюють різноманітний спектр вроджених аномалій, які часто виявляють при народженні або в ранньому дитинстві. Серед них судинні невуси, що є вродженими капілярними мальформаціями, які можуть призводити до значних косметичних та функціональних порушень.

Мета: визначити основні клінічні та генетичні особливості рідкісного випадку судинного невуса в дитини та встановити діагностичні критерії, що дають змогу своєчасно диференціювати цю патологію від інших судинних мальформацій.

Клінічний випадок. Представлено ретроспективний аналіз клінічного випадку судинного невуса у 6-річної дівчинки. Пацієнтка мала вроджену судинну пігментацію та прогресуючу асиметрію кінцівок. У неї також була обширна капілярна мальформація, що вражала поперекову ділянку, стегно, гомілку та стопу, асоційовану з гіпертрофією ураженої кінцівки. Рентгенологічне дослідження підтвердило анатомічне подовження правої великогомілкової кістки на 2 см зі збереженою функцією росткової пластинки. Клінічні ознаки викликали підозру на факоматоз, що вимагало проведення диференційної діагностики з такими станами, як синдром Кліппеля–Треноне–Вебера, синдром Штурге–Вебера, нейрофіброматоз та інші. Генетичне тестування не виявило патогенних мутацій, які зазвичай асоціюються з факоматозами, що підтвердило остаточний діагноз ізольованого судинного невуса з ураженням опорно-рухового апарату. Пацієнту було призначено мультидисциплінарний план лікування, який охоплював ортопедичну корекцію, лікувальну фізкультуру та постуральний моніторинг для запобігання прогресуванню сколіозу.

Висновки. Судинні мальформації, особливо великі судинні невуси, можуть індукувати непропорційний ріст опорно-рухового апарату, імітуючи синдромні факоматози. Цей випадок вказує на необхідність інтеграції клінічних, рентгенологічних і генетичних досліджень для забезпечення точної діагностики та індивідуалізованого лікування. Отримані дані вказують на важливість довготривалого моніторингу в дітей зі складними судинними аномаліями для оптимізації функціональних результатів та запобігання вторинним ортопедичним ускладненням.

Дослідження виконано відповідно до принципів Гельсінської декларації. На проведення досліджень отримано інформовану згоду батьків дитини.

Автори заявляють про відсутність конфлікту інтересів.

Ключові слова: судинні мальформації, судинний невус, капілярні мальформації, факоматози, генетичний аналіз, асиметрія кінцівок.

Introduction

Vascular anomalies of the skin represent a diverse group of congenital or early-onset lesions that frequently prompt medical evaluation in pediatric populations [9,6]. Although these lesions can appear at any age, they are often detected at birth or within the first weeks to months of life [7]. According to the contemporary classification by the International Society for the Study of Vascular Anomalies (ISSVA), vascular anomalies are broadly subdivided into two principal categories: vascular tumors (predominantly infantile hemangiomas) and vascular malformations (capil-

lary, venous, lymphatic, arteriovenous, or combined) [13,19].

Infantile hemangiomas (the most common vascular tumors in infancy) are characterized by a proliferative phase followed by partial or complete involution [11,12]. In contrast, vascular malformations, including vascular nevi (port-wine stains, or nevus flammeus), are structurally stable lesions that do not undergo the rapid growth or regression phases seen in hemangiomas [16,26]. Vascular nevi, in particular, are congenital capillary malformations that manifest as pink to reddish-purple macular lesions on the skin; they may progressively darken and thicken over time, depending on location and depth [3,10]. Although often benign in clinical course, these lesions can be associated with significant psychosocial and, in certain cases, functional complications – especially when affecting cosmetically or functionally sensitive areas such as the face or the periocular region.

The complexity of vascular anomalies necessitates a thorough diagnostic approach, which may include physical examination, imaging studies (e.g., Doppler ultrasonography, MRI), and, if indicated, histopathological assessment [15]. Early and accurate diagnosis is critical to differentiate potentially self-limiting lesions (such as infantile hemangiomas) from those requiring active intervention (e.g., large or symptomatic vascular malformations). Moreover, certain vascular anomalies can be linked to syndromic presentations – Sturge-Weber syndrome and PHACE syndrome being prime examples – warranting interdisciplinary evaluation by pediatricians, dermatologists, ophthalmologists, neurologists, and, if needed, geneticists [27].

The aim of the study is to identify the main clinical and genetic features of a rare case of vascular nevus in a child and to determine the diagnostic criteria that allow timely differentiation of this pathology from other vascular malformations. The study is aimed at analysing phenotypic manifestations, genetic markers, and their association with growth disorders of the musculoskeletal system, which will optimise the approach to diagnosis, treatment, and prevention of possible complications.

The present study is a retrospective analysis of a clinical case of a rare vascular nevus in a child, performed at the Ternopil Regional Children's Clinical Hospital, Ternopil, Ukraine. The patient, a 6-year-old girl, presented with complaints of gait disturbance and congenital skin pigmentation abnormalities. Clinical follow-up was conducted for a year to assess the progression of the vascular malformation and its impact on the musculoskeletal system.

The clinical examination included a general examination, assessment of skin pigmentation, limb asymmetry, and gait analysis. Orthopaedic examination included assessment of pelvic alignment, detection of scoliosis, and the Adams test. X-ray examination of both lower extremities in anterior-posterior projection was performed to detect bone lengthening and structural abnormalities.

Genetic analysis was performed using the next generation sequencing (NGS) technique with target enrichment based on hybridisation processed using Illumina technology [28]. DNA sequencing was performed at the laboratory of Labcorp Genetics, Inc. (1400 16th Street, San Francisco, CA 94103, No. 05D2040778). The analysis was focused on specific genomic regions associated with phacomatosis and vascular malformations, including the Invitae Hereditary Solid Tumour Panel, additional pediatric solid tumour pre-diagnostic genes, the Invitae Hereditary Paraganglioma-Pheochromocytoma Panel, and additional paraganglioma and pheochromocytoma pre-diagnostic genes. The obtained sequences were aligned to the GRCh37 reference genome, and exonic deletions and duplications were detected using the internal copy number algorithm. For quality control and confirmation of the expected genetic sex, markers on the X- and Y-chromosomes were analyzed.

The study was carried out in accordance with the principles of the Declaration of Helsinki. The informed consent of the children's parents was obtained for the research.

Clinical case

The clinical case of a 6-year-old patient born from a full-term pregnancy with an uncomplicated perinatal history was analyzed. At birth, extensive hyperpigmentation of the skin in the form of large pale red spots of various sizes and shapes was observed on the right lower extremity. The child had normal growth and developmental milestones. A year before the visit, the mother reported progressive lameness of the left leg, with no history of trauma.

Clinical examination revealed left-sided lameness, pelvic displacement with a leftward tilt, asymmetrical location of the shoulder and pelvic triangles. There was a deviation of the spinal axis to the left. There was a discrepancy in the length of the limbs: the right leg was 2 cm longer than the left. In addition, the circumference of the right thigh and lower

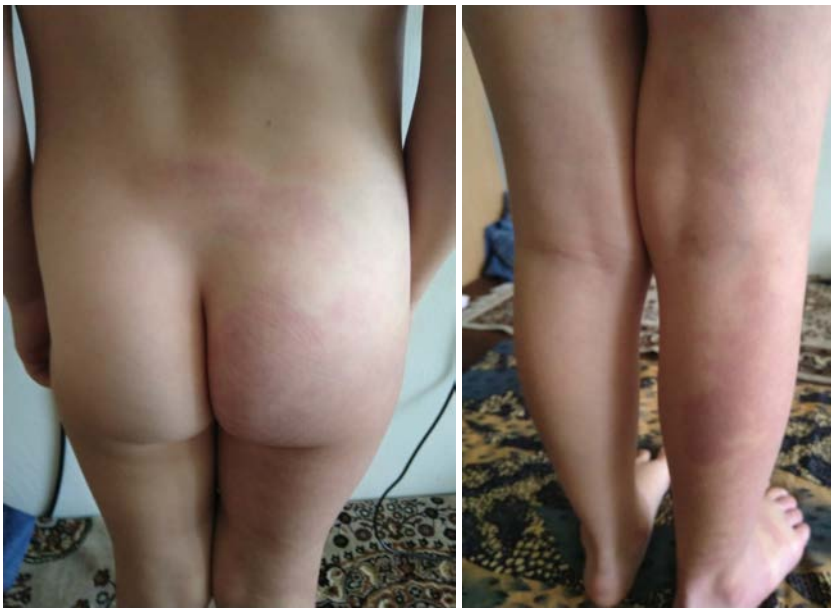

Fig. 1. Vascular nevus of the lumbar region and lower limb

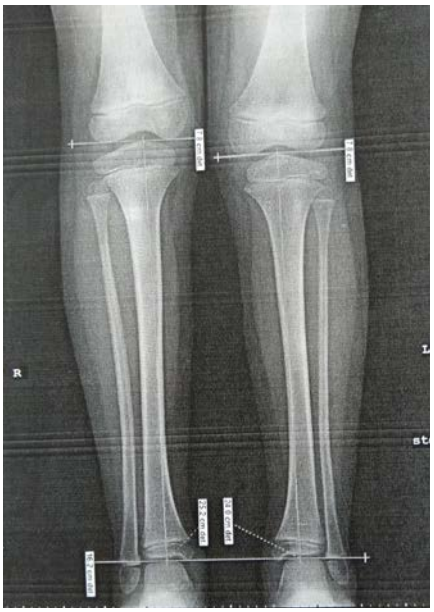

Fig. 2. Comparative radiograph of both tibiae

leg at the level of the middle third exceeded the same indicator of the opposite limb by 1.5 cm and 1 cm, respectively. The right foot was lengthened by 1 cm compared to the left, and hypertrophy of the right gluteal region was observed. The Adams test with a forward tilt was negative. After compensatory elevation of the left limb by 2 cm, the spinal curvature was eliminated, and the pelvic alignment was normalized. Dermatological examination of the right lumbar region revealed extensive hyperpigmentation, characterized by multiple well-defined, irregularly shaped, pale red spots that did not rise and were painless to palpation. Compression of the lesion did not lead to complete pallor, although a slight decrease in colour intensity was noted. The right thigh, lower leg, and dorsum of the foot were densely covered with multiple port-coloured spots of varying sizes with clearly defined edges (Fig. 1). In this case, it is indicative of a complex vascular malformation involving both skin and musculoskeletal structures, which required further radiological and genetic testing.

.Comparative anteroposterior radiography of both tibiae revealed an anatomical lengthening of the right tibia, which was approximately 2 cm longer than the contralateral limb. The epiphyseal growth plates of the tibia and distal femur metaphyses remained open, indicating ongoing skeletal maturation (Fig. 2). These findings confirmed the clinical picture of asymmetric limb growth and indicate the presence of a vascular malformation affecting bone development.

Accelerated growth of bones and muscles of the right leg, extensive vascular malformation in the form of a large port-coloured spot, and localized fever of the affected limb raised suspicion of a phacomatosis spectrum disorder. Phacomatoses are characterised by characteristic skin manifestations, often accompanied by neurological and systemic lesions. In early childhood, dermatological signs usually precede the onset of neurological and other systemic symptoms, requiring a thorough examination and long-term follow-up to establish a definitive diagnosis.

Preliminary clinical diagnosis: Phacomatosis? Unequal length of the lower extremities due to anatomical elongation of the right leg. Unstructured left-sided scoliosis.

Differential diagnosis was carried out within the spectrum of vascular malformations classified as phacomatoses, which cover a number of neurocutaneous syndromes with vascular, neurological, and systemic manifestations. The main conditions considered were:

- neurofibromatosis (NF) type 1 (Recklinghausen's disease);
- tuberous sclerosis complex (Borneville–Pringle syndrome);
- pigmented urinary incontinence (Bloch–Sulzberger syndrome);
- Klippel–Trenaunay–Weber syndrome;
- Sturge–Weber syndrome.

Neurofibromatosis types 1 and 2, also known as Recklinghausen disease, represent the most common phakomatoses [8]. These conditions are multisystemic, autosomal dominant neurocutaneous disorders characterized by the involvement of the skin, central and peripheral nervous systems, skeletal

structures, endocrine organs, gastrointestinal tract, and vascular walls [22]. The underlying genetic pathology involves mutations in the NF1 gene located on the long arm of chromosome 17 (17q11.2), leading to dysregulation of cell proliferation [21]. The intact NF1 allele, found on the homologous chromosome, encodes neurofibromin, a tumor suppressor protein. The loss of function in NF1 results in uncontrolled cell growth, leading to the development of both benign and malignant neoplasms, including neurofibromas, schwannomas, and malignant peripheral nerve sheath tumors (neurofibrosarcomas) [20].

Neurofibromatosis is classified into three major subtypes: NF1, accounting for approximately 96% of cases; NF2, representing 3%; and schwannomatosis (SWN), which comprises less than 1% of cases. According to the international consensus guidelines, NF2 and schwannomatosis have been reclassified under the term Schwannomatosis (SWN). The clinical phenotype of NF is primarily characterized by the presence of multiple neurofibromas along peripheral nerves, though genotype-phenotype correlations remain weak, making predictive diagnosis based on genetic mutations challenging. Approximately 50% of NF1 cases arise from de novo mutations, further complicating early genetic screening. The clinical diagnostic criteria for NF1 include the presence of six or more café-au-lait macules, at least two neurofibromas, a minimum of two Lisch nodules on the iris, and skeletal abnormalities such as scoliosis. In the case under study, these key diagnostic features were absent. The patient exhibited no multiple neurofibromas, Lisch nodules, or café-au-lait macules, effectively ruling out NF as the primary diagnosis. However, given the significant limb asymmetry and extensive vascular malformation, alternative differential diagnoses within the phakomatosis spectrum were considered, with a particular focus on Klippel–Trenaunay–Weber syndrome [25].

Tuberous sclerosis complex (TSC), also known as Borneville–Pringle disease, is a rare genetic disorder characterized by the formation of benign tumors in many organs and tissues, including the brain, heart, skin, eyes, kidneys, and lungs [5]. The disease is inherited in an autosomal dominant manner and is caused by mutations in the TSC1 and TSC2 genes located on chromosomes 9q34 and 16p13.3, respectively. These genes encode hamartin and tuberin, tumour suppressor proteins that regulate cell proliferation and tissue growth. Mutations in these genes lead to dysregulation of the activation of the mTOR pathway (a mechanical target of rapamycin), which promotes excessive cell growth and leads to the widespread development of hamartomas [2,29]. The tuberous sclerosis complex is characterized by a wide range of clinical manifestations, mainly neurological, dermatological, and systemic signs. The classical triad of Vogt's TSC includes epileptic seizures, which are observed in 70-80% of affected children and often manifest during the first year of life progressive intellectual disability, cognitive impairment of varying degrees, often associated with autism spectrum disorders and behavioural abnormalities and facial angiofibromas (sebaceous gland adenoma) – pink or reddish-brown papular lesions, mainly located on the central part of the face, in particular on the nasolabial folds, chin and forehead.

Cutaneous manifestations often include hypomelanotic macules (ash spots), shagreen patches, and periradial fibromas, none of which were observed in this clinical case. Due to the genetic heterogeneity and variable expression of TSC, the International Consensus Conference on Tuberous Sclerosis (2012) revised the diagnostic criteria, emphasising the need for genetic testing for early and accurate identification of the disease [14,30]. Pathogenetic variants of TSC1 or TSC2 confirm the diagnosis, especially in cases with an ambiguous clinical picture.

The patient in this study had localized vascular pigmentation on the lower extremity without the presence of multiple fibrous skin nodules, angiofibromas, or other dermatological features characteristic of TSC. Moreover, neurological symptoms such as seizures or developmental delay were absent, which also excluded the diagnosis of tuberous sclerosis. In view of these findings, TSC was excluded as a potential underlying pathology in favour of vascular nevus with clear clinical and genetic characteristics.

Encephalotrigeminal syndrome (Sturge–Weber–Krabbe syndrome) is a rare congenital developmental disorder that causes multiple angiomas, skin and eye angiomas [4]. Many patients develop neurological and psychiatric health problems, such as hemiparesis, as well as intellectual disability and epilepsy, due to damage to the second or first branch of the nerve, which leads to multiple port-coloured spots on the face. The disease has three identifiable clinical types, including cases with leptomeningeal angioma together with facial angiomas that lead to glaucoma, as well as patients who develop facial angiomas alone and patients with leptomeningeal angioma that does not lead to glaucoma. Glaucoma and central nervous

system lesions accompany the main clinical picture of this condition, along with port-coloured spots on the face that form multiple fibrous growths. The patient did not have facial involvement, as the dark purple port wine stains were only on her lower legs. The results of eye pressure measurements were normal, and the child demonstrated typical developmental signs without neurological abnormalities, which allowed to exclude Sturge–Weber–Krabbe syndrome. Numerous clinical markers excluded the diagnosis of encephalotrigeminal angioma, which confirmed that this vascular disease corresponds to the characteristics of a vascular nevus, rather than a lesion of the central nervous system and facial neurocutaneous syndrome.

Bloch–Sulzberger syndrome (degenerative melanosis) is a rare hereditary disease manifested by mottled pigmentation of the skin of a child's body. The first signs of the disease are detected in the first 2 weeks of a newborn's life [23,31]. The course of the disease is characterized by a clear sequence. Initially, a vesicular-bullous rash occurs, which transforms into keratotic plaques. At the age of 12–40 weeks, pigment spots appear and gradually transform into linear atrophic depigmented areas of the skin, which does not coincide with our clinical observation.

The clinical syndrome is known as Cavernous Cutaneous Angiomatosis and Osteohypertrrophy (Klippel–Trénauney–Weber Syndrome). This very uncommon and interesting syndrome shows various malformations of skin and vascular structures in organs and tissue [17,18]. The complex vascular disorder of the PIK3CA gene somatic mutation causes excessive growth throughout the body. The condition leads to increased Fibroadipose overgrowth together with Hemihypertrophy-multiple lipomatosis while causing developmental blood vessel anomalies and epidermal nevi and affects skeletal bone formation (skeletal-spinal syndrome) in addition to macrodigitism and megalencephaly, which follows the genotypic-phenotypic correlation. The main clinical features of the condition include a combination of cutaneous birthmarks and nevi, together with vascular tumors that manifest on the patient's limb as well as soft tissue and bone enlargement and venous and lymphatic duct dilation [30]. Signs include an enlarged surface area affected by the hamartoma, together with tissue hypertrophy. Doctors can see the hemangioma of a baby at birth, along with its size and shape, which may differ from one another, while its vascular nevi are typically faint pink. The excessive blood flow at the affected area expands and lengthens

GENE	TRANSCRIPT
AIP	NM_003977.3
ALK	NM_004304.4
APC*	NM_000038.5
BAP1	NM_004656.3
BLM	NM_000057.3
BMPR1A	NM_004329.2
BUB1	NM_004336.5
BUB1B	NM_001211.5
CDC73	NM_024529.4
CDK4	NM_000075.3
CDKN1B	NM_004064.4
CDKN1C	NM_000076.2
CDKN2A (p14ARF)	NM_058195.3
CDKN2A (p16INK4a)	NM_000077.4
CEP57*	NM_014679.4
CTR9	NM_014633.4
DICER1*	NM_177438.2
DIS3L2*	NM_152383.4
DLST	NM_001933.5
EGLN1*	NM_022051.2
ELP1	NM_003640.3
EPAS1*	NM_001430.4
EPCAM*	NM_002354.2
EXT1	NM_000127.2
EXT2	NM_207122.1
EZH2*	NM_004456.4
FBXW7	NM_033632.3
FH*	NM_000143.3
GPC3*	NM_004484.3
GPR161	NM_001267609.1
HRAS	NM_005343.2
KIF1B*	NM_015074.3
LZTR1	NM_006767.3
MAX*	NM_002382.4
MDH2	NM_005918.3
TRIM28	NM_005762.3
TRIP13	NM_004237.4
TSC1*	NM_000368.4

GENE	TRANSCRIPT
MEN1*	NM_130799.2
MITF	NM_000248.3
MLH1*	NM_000249.3
MSH2*	NM_000251.2
MSH6*	NM_000179.2
NBN	NM_002485.4
NF1*	NM_000267.3
NF2	NM_000268.3
NYNRIN	NM_025081.3
PHOX2B*	NM_003924.3
PMS2*	NM_000535.5
POT1	NM_015450.2
PRKAR1A	NM_002734.4
PTCH1	NM_000264.3
PTCH2	NM_003738.4
PTEN*	NM_000314.4
RB1*	NM_000321.2
RECQL4*	NM_004260.3
REST	NM_005612.4
RET	NM_020975.4
SDHA*	NM_004168.3
SDHAF2	NM_017841.2
SDHB	NM_003000.2
SDHC*	NM_003001.3
SDHD	NM_003002.3
SLC25A11	NM_003562.5
SMAD4	NM_005359.5
SMARCA4	NM_001128849.1
SMARCB1	NM_003073.3
SMARCE1	NM_003079.4
STK11	NM_000455.4
SUCLG2	NM_001177599.1
SUFU	NM_016169.3
TMEM127	NM_017849.3
TP53	NM_000546.5
TSC2	NM_000548.3
VHL	NM_000551.3
WRN*	NM_000553.4
WT1	NM_024426.4

Fig. 3. List of analyzed genes

the limb, while potential arteriovenous connections may also develop [1]. Radiological investigations reveal elongation and density increase in bone structures of the limb, which causes the limb to grow disproportionally and distorts spinal formation. The phenotype of our clinical observation matched this syndrome the most, which led us to conduct DNA genomic testing for differential diagnosis.

Given the patient's complex phenotypic presentation, which included a combination of an extensive vascular malformation, asymmetric limb growth, and orthopedic abnormalities, a more detailed approach to differential diagnosis was required. Since these clinical signs can be characteristic of various phakomatoses, molecular genetic analysis was necessary to determine the specific etiology of the pathology.

To refine the diagnosis, next-generation sequencing (NGS) of 76 genes was performed, covering ma-

jor genetic markers associated with phakomatoses and vascular malformations. The analysis included the following genetic panels:

- invitae hereditary pediatric solid tumors panel;
- add-on preliminary-evidence genes for pediatric solid tumors;
- invitae hereditary paraganglioma-pheochromocytoma panel;
- add-on preliminary-evidence genes for paraganglioma-pheochromocytoma.

The results of the genetic panel screening did not reveal any pathogenic variants associated with autosomal dominant disorders (phakomatoses), tumors, or tumor-like formations. However, the analysis does not exclude the possibility of other genetic conditions that were not covered by this specific method of examination (Fig. 3).

Final clinical diagnosis. Vascular nevus of the right half of the lumbar spine, right thigh, lower leg, and foot. Different lengths of the lower limbs due to anatomical elongation of the right lower limb. Hypertrophy of the muscles of the right buttock, thigh, and lower leg. Unstructured left-sided scoliosis without functional disorders.

Taking into account the combination of orthopaedic and dermatological manifestations, a multidisciplinary approach was applied with an emphasis on posture correction, orthopaedic support, and regular monitoring to prevent possible complications:

• To address the leg length discrepancy and its impact on spinal alignment, the patient was fitted with individual orthotics designed to compensate for the 2 cm difference in leg length. This intervention aims to restore pelvic balance, prevent further curvature of the spine, and reduce the biomechanical load on the lower extremities and spine.

• A structured physical therapy programme was developed to improve postural control, muscle symmetry, and joint function. The programme includes targeted physical therapy exercises to strengthen core muscles and promote spinal stability, stretching techniques to maintain flexibility and prevent muscle imbalances. Additionally, manual therapy and massage were recommended to help relax muscles and correct posture. However, lumbar massage was excluded to avoid excessive vascular stimulation in the affected area.

• To further improve spinal alignment and reduce the compensatory load, the patient was recommended to use a posture corrector. This device provides additional support, especially during prolonged sitting and standing, minimising the risk of scoliosis progression.

Given the possible progressive nature of vascular malformations, ongoing monitoring and preventive measures were emphasised, such as:

• Annual radiographic assessment of the lower extremities to monitor growth and skeletal maturation discrepancies.

• Regular orthopaedic examinations every 6-12 months to monitor spinal curvature and leg length discrepancies.

• Avoidance of excessive mechanical irritation, including the use of protective clothing and footwear to prevent friction and minor injuries to the affected skin.

• Sun protection measures, including the use of high SPF sunscreen and avoidance of prolonged exposure to direct sunlight, to prevent potential changes in pigmentation or vascular reactivity.

• Exclusion of thermal treatments such as paraffin applications, ozokerite therapy, ultrasound therapy, and hydrotherapy, including radon or sulphur baths, which can increase vascular proliferation.

• genetic counselling to further assess potential late onset or family predisposition.

Conclusion

The phenotype and genotype of vascular malformations do not always coincide, so the final diagnostic criterion is genetic DNA testing. A large vascular nevus that stimulates the outstripping growth of muscles and bones of the limb is very rare, which should be remembered when examining various vascular malformations and phacomatosis in particular.

Future research prospects include further study of various genetic and phenotypic manifestations of vascular malformations of the skin in children for the purpose of timely diagnosis, treatment, and prevention of possible complications.

The authors declare that the study was conducted in the absence of any commercial or financial relationship that could be interpreted as a potential conflict of interest.

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