

FURTHER VASCULAR, BONE AND AUTONOMIC INVESTIGATIONS IN ALGODYSTROPHY

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Direct clinical observation is the most common means of diagnosing algodystrophy. Further investigations may be helpful to rule out other pathological conditions, such as occult or stress fractures or avascular osteonecrosis and to obtain a better understanding of algodystrophy. Transient vascular hyperpermeability in the affected part is well demonstrated by the clinical findings, the MRI signs, and the three-bone scan features. 99m Technetium EHDP bone scan provides an evaluation of the vascular abnormalities and of the osteoblastic activity. Dermal microcirculation and its reactions to sympathetic stimuli are investigated by laser doppler fluximetry and videophotometric capillaroscopy. Perhaps the sweat test does unveil what might be specific about algodystrophy. The amount of bone loss in algodystrophy in a few weeks or months is what might be expected over 10 years during the natural history of uncomplicated osteoporosis. An initial fracture is undoubtedly an initiating event in the appearance of algodystrophy, but patients suffering from algodystrophy may still have significant osteoporosis for a long period and hence be at risk for fracture. Densitometry could be an aid to the diagnosis and probably to monitoring treatment as well. The local colonization of fibroblasts following the transient stage of hyperpermeability must be kept in mind to explain the results of joint, bone, muscles or neurological investigations in late algodystrophy.

Keywords : algodystrophy ; investigations ; fractures.

Mots-clés : algodystrophie ; investigations ; fractures.

For a clinician, reflex sympathetic dystrophy or algodystrophy is easy to diagnose, but difficult to define. The occurrence of an initiating noxious event is important. The pain is continuous and is accentuated with the use of the joints. Direct

clinical observation remains the most common means of diagnosing algodystrophy. There is a limited active range of motion in the joints of the affected area. Patients may have allodynia (pain following non-noxious stimulation since all stimuli are perceived as painful), hyperalgesia (exaggerated pain following noxious stimulation, owing to a lower pain threshold and increased sensitivity), hyperpathia (the threshold to pain is increased, but once exceeded, the sensation increases in intensity more rapidly and to a greater degree than expected ; the pain progresses explosively and is still perceived after the stimulus is removed). The pain is disproportionate to the initiating event. There is evidence at some time of edema, changes in blood flow or abnormal sudomotor activity in the region of the pain, changes in skin temperature, skin color and sweating, relative to the contralateral limb. The above signs and symptoms are present in an area larger than the area of primary injury or operation including the area distal to the primary injury.

Further investigations may be helpful in ruling out certain pathological conditions and obtaining a better understanding of algodystrophy.

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GENERAL INVESTIGATIONS IN ALGODYSTROPHY

The Question of Inflammation in Algodystrophy

There is no acute phase response in algodystrophy: the erythrocyte sedimentation rate and the concentrations of C-reactive protein and other acute phase reactants remain normal. The patients do not have fever, but local signs in the affected area suggest inflammation: swelling, marked edema, increased temperature and redness. In fact, patients with algodystrophy actually present major vascular abnormalities. A local increase in microvascular permeability in primarily warm algodystrophy has been demonstrated in the affected extremity for high molecular weight proteins (IgG labelled with ¹¹¹Indium) as well as for red blood cells (28). Dynamic gamma-angiography shows an 80% increase of ^{99m}Tc-pertechnate labelled red blood cells in the local vascular and extravascular compartments. The extended pattern observed in bone scans is attributed to regional hyperemia. We believe that microvascular hyperpermeability plays a key role in the pathogenesis of algodystrophy.

Laboratory Investigations (13, 23)

Routine biochemical assays, hematology, bacteriology and serology are not relevant in algodystrophy. Serum alkaline phosphatase and parathyroid hormone levels are usually normal. Hydroxyprolinuria, related to creatininuria, in the urine collected in the early morning is slightly enhanced or within normal limits. However, it may be useful to study cellular bone remodelling by means of biological markers such as osteocalcin (bone Gla protein) for bone formation, as well as pyridinoline (found in bone and cartilage) and deoxypyridinoline (found almost exclusively in bone) for bone resorption.

Some cases of algodystrophy have been observed in osteomalacia with renal tubulopathy and/or phosphorus and dicarbonated diabetes or hyperparathyroidism, with important therapeutic implications. It is interesting to note the high prevalence of diabetes mellitus in algodystrophy (6% in a French cooperative study, versus 0.2-2%

in the general population). The transient precocious hypertriglyceridemia observed in algodystrophy may be a consequence of immobility.

Synovial Fluid and Synovium Investigations (11, 21, 22)

The synovial fluid is clear or straw-colored. Its volume is minimal. The leukocyte count is usually between 500 to 2000 cells/ml. Cultures yield no organisms. The synovial biopsy — justified only for ruling out other pathological conditions — shows mild synovial changes: proliferation of small blood vessels or hypervascularity, synovial edema, mild or absent proliferation of synovial lining cells, little or absent inflammatory cell infiltrate, and fibrosis in the deeper synovial layers.

Magnetic Resonance Imaging (MRI) in Algodystrophy

In the early phase of algodystrophy, MRI may reveal a mild articular effusion (T2), a reduced T1 and increased T2 signal of the affected bone area without precise limits, reflecting transient bone marrow hyperemia (Fig. 1) (27). These modifications of the medullary signals precede maximum rarefaction and regress later on. Later, MRI is normal or in a few cases a "swiss-cheese" signal is present (Fig. 2). MRI is useful to rule out other diagnoses: radiographically occult avascular osteonecrosis or trabeculae microfractures, infections

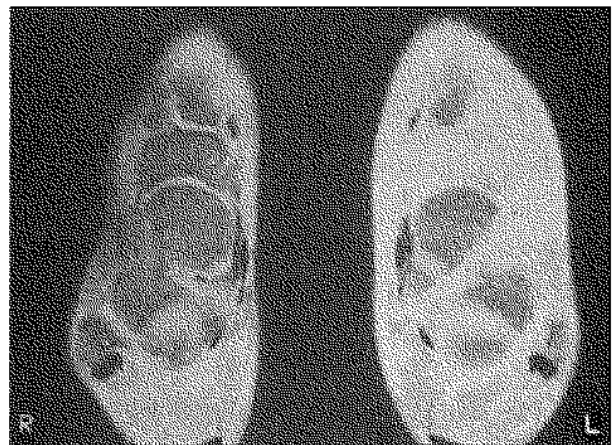


Fig. 1. — MRI (T1 + gadolinium) shows areas of hyperemia in a case of algodystrophy of the midfoot.

(tuberculosis, osteitis), malignant or nonmalignant neoplasms or other conditions. Bone marrow edema may occur in various conditions other than algodystrophy, such as trabecular fractures (Fig. 3), avascular osteonecrosis (Fig. 3) (venous stasis or medullary edema), nonmalignant neoplasm (osteoid osteoma) and so on. In fact, MRI is a modern way of demonstrating the presence of edema in superficial and deep tissues in cases of algodystrophy (Fig. 1). Edema and hyperemia

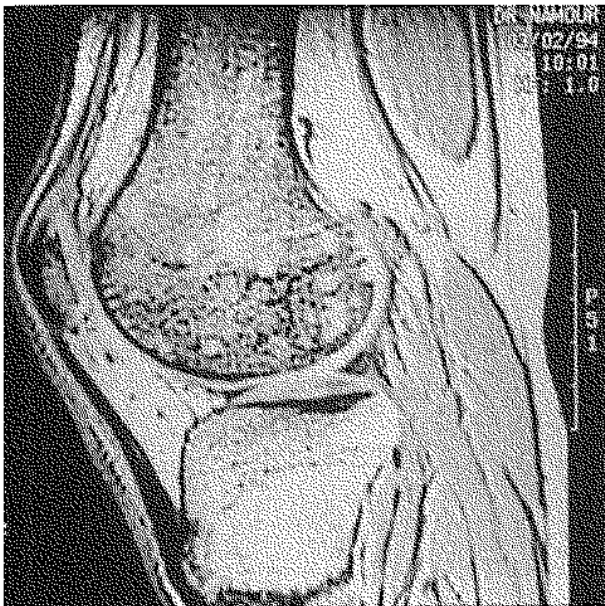
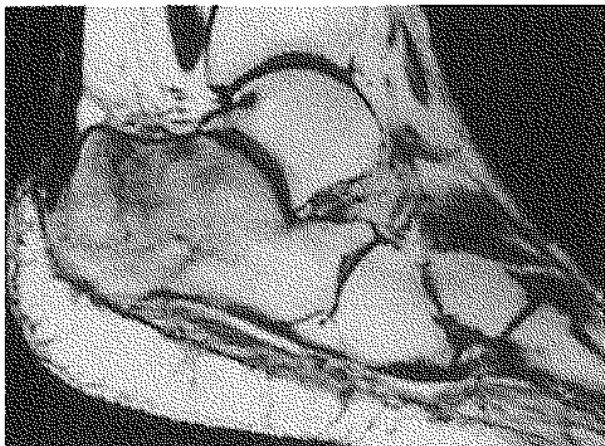


Fig. 2. — Rare "swiss cheese" pattern in MRI in stage III algodystrophy of the knee.



a



b



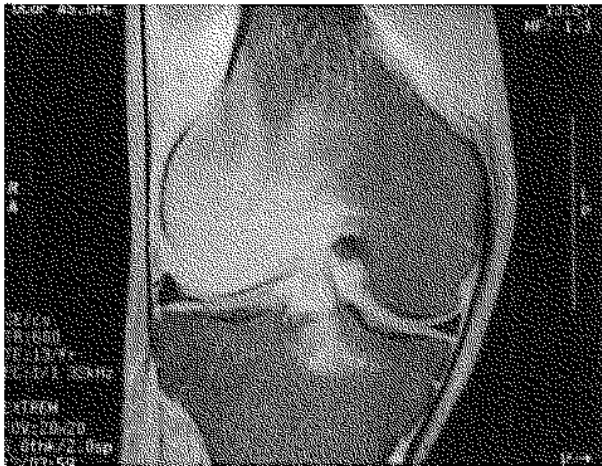
c



d

Fig. 3. — Bone marrow edema in occult trabecular fractures (a : calcaneus ; b : medial tibial plateau ; c : femoral head) and in ischemic osteonecrosis (d : femoral head).

are enhanced by the infusion of gadolinium. In the later stages, there is no edema, and MRI findings in the dystrophic stage are rather disappointing (Fig. 4). However, it should be noted that even in the early phase, some patients may have no bone edema in the affected extremity. In these cases, bone scans reveal no increase in radionuclide uptake, and even a low uptake in comparison with the other side, especially in adolescent or young adult female patients.



a

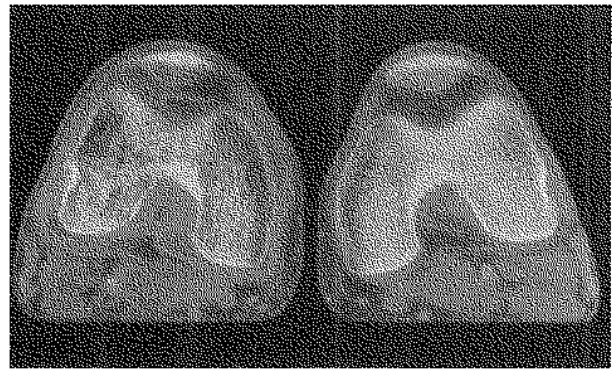


b

Fig. 4. — MRI shows transient bone-marrow edema in a case of knee algodystrophy limited to the lateral femoral condyle (a : July 8 ; 1996, b : November 13, 1996).

Computed Tomography in Algodystrophy (26, 27)

Computed tomography is not indicated in algodystrophy. However, when it has been used to rule out some other condition and when, retrospectively, the patient has been found to be suffering from algodystrophy it can show (i) local demineralization (metaphyseal or subchondral band-like osteopenia, homogeneous or patchy epiphyseal osteoporosis) (Fig. 5) ; (ii) thickened para-articular structures (capsule, tendons, ligaments) ; and (iii) integrity of the joint space (absence of joint space narrowing, absence of significant intra-articular erosion).



a



b

Fig. 5. — Tomodensitometric aspects. Patchy osteoporosis (a) versus homogeneous demineralization (b). a : A case of algodystrophy of the knee ; b : A case of algodystrophy of the hip.

BONE TISSUE INVESTIGATIONS

Histologic Microscopic Features of Bone (1, 2, 4, 18, 23, 25)

Medullary Space

Bone medullary tissues may present abnormalities such as: erythrocyte accumulation in the medullary spaces, plasma exsudation dissociating the blood-forming bone marrow, capillary and venous dilatation, arteriolar intima and media thickening and/or vasospasm, and fibroblastic colonization of medullary space. In our opinion, plasma exsudation and erythrocyte accumulation correspond to an important stage (Fig. 6), followed by fibroblastic colonization (Fig. 6). The accumulation of erythrocytes along the trabeculae in the medullary space is frequently seen in bone biopsies irrespective of other histologic features. This accumulation is not linked to venous compression or thrombosis. The histological observation of this phenomenon in bone tissue has been confirmed by dynamic gamma-angiography. The accumulation of erythrocytes, together with the hyperpermeability, must be kept in mind to allow a satisfactory interpretation of the different types of bone scans in algodystrophy.

Trabecular and Cortical Bone

Zonal osteocyte degeneration is frequent. There may be considerable loss of trabecular bone in the affected area with a decrease in trabecular width, or even a complete disappearance of trabeculae, leaving some thin, widely spaced, poorly anastomosed large lacunae. Increased osteoblastic activity has been reported by several authors. Osteoclasts are present at irregular intervals on (necrotic) "dead" lamellar bone tissue as well as on newly formed irregular bone. The histologic images of algodystrophy bone rarely show osteoclasts, whereas osteoblastic activity is always well demonstrated. During or after recovery, there is reparative bony regeneration, and the bone is of the woven bone type with persistence of abnormal trabeculae and trabecular hypertrophy. Unfortunately, there are no data available for the cortical sites. It would be interesting to investigate the links

between the cortical site and the microvascular network.

With electron microscopy,

some trabeculae appear normally calcified. Other trabeculae show distinct disturbances in mineralization. In some zones of lamellar bone, the hydroxyapatite crystals form spiky clusters of various sizes irregularly dispersed among the collagen fibrils. At places, the mineralization totally disappears, and large areas of lamellar fibrillar collagen matrix are devoid of hydroxyapatite crystals. Osteocytes are either normal or degenerating. Osteoblasts are in the resting or the active state.



a



b

Fig. 6. — Histological bone features: plasmostasis with accumulation of erythrocytes (a), colonization by fibroblasts, osteoblastic hyperactivity (b).

Is Hyperosteoclastic Activity Really Prominent in Algodystrophy ?

Bone loss in algodystrophy (as high as 30% or more for some patients in only a few weeks) is far more rapid than in known hyperosteoclastic pathologies. Bone biopsies in the second or third month do not show the hyperosteoclastic activity which might explain the rapid bone loss. Electron microscopic studies show irregular mineralization of bone with patchy clear zones of collagen fibers (caused by apatite dissolution) never seen in active osteoclasts. The decrease in hematopoietic marrow and in the number of osteocytes is not in favor of durable hyperosteoclastic activity, nor of a multiplication of osteoclasts. Other mechanisms have to be investigated, e.g. the influence of the microvascular abnormalities *per se*. The localization of the area of demineralization and the distribution of the bone microcirculation are very likely two important related features of algodystrophy.

Local Evaluation of Bone Mineral Density and Content in Algodystrophy (Fig. 7)

Bone demineralization in the affected limb is clearly demonstrated by bone mineral density (BMD, g/cm²) and bone mineral content (BMC, g) measurements. BMD and BMC measured by dual x ray absorptiometry were significantly lower in the involved side of 12 patients with phase I algodystrophy following trauma less than 60 days after the appearance of symptoms (28.4 and 45.1%, respectively) compared with the contralateral normal limb and with 18 controls (3). A pathological demineralization threshold of 10% has been demonstrated by biphotonic absorptiometry for the involvement of the forepart of the tarsus (16). Bickerstaff *et al.* (5) evaluated 250 patients with Colles' fracture, of whom 44 had post-traumatic algodystrophy ; 33 of these patients were considered as controls. BMC and BMD were evaluated by single-photon absorptiometry at the distal 4th and 5th metacarpals. The loss of bone 7 weeks after fracture was significantly greater in algodystrophy than in controls both at the cortical and the trabecular sites. The decrease in cortical

bone density was greater than 10% and that of trabecular bone greater than 25%. Bone loss after Colles' fracture had already been well demonstrated by single-photon absorptiometry in previous studies : 18% at 1 year in the distal radius, 18% at 1 year in the midshaft radius and the ulna, 36% at 2.5 years in the distal radius. The bone

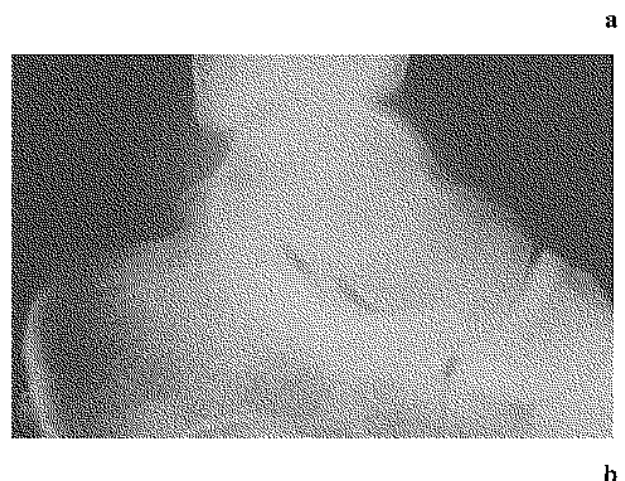
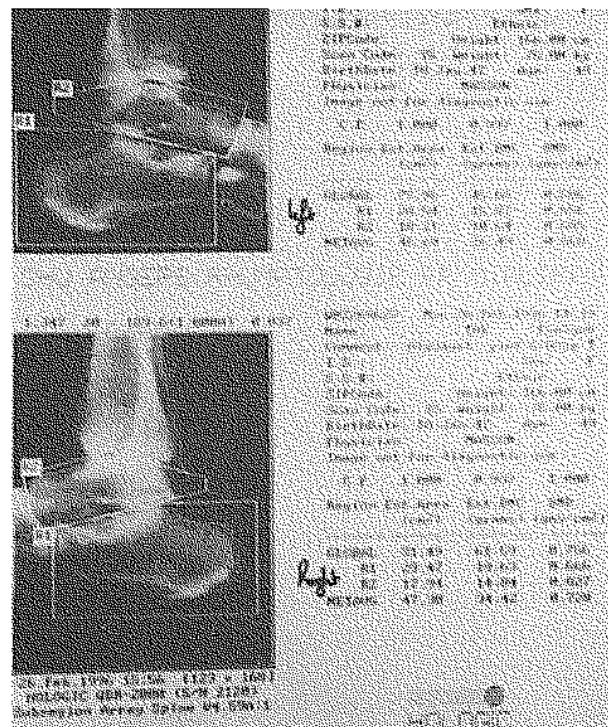


Fig. 7. — Dramatic decrease in bone mineral content in a case of algodystrophy of the foot (a) while x ray (at the same date) reveals slight homogeneous demineralization of the talus (b).

loss seen in patients with algodystrophy persisted for the 6-month duration of the follow-up and up to one year in all nine patients studied for a longer time. Normal bone mass was restored in the control patients by 19 weeks after fracture at cortical sites and by 31 weeks in trabecular bone. In 3 patients with transient osteoporosis of the hip, loss of whole BMD at the affected hip was similar, but the average loss was as high as 36% when Ward's triangle (the zone unaffected by weight-bearing) was considered (28). In 28 males and 11 females with a mean age of 37 years suffering from algodystrophy, BMC was decreased by 8.8%, BMD by 9.6% and fat-free mass by 6.2%, whereas body fat increased by 6% (17). These differences were largest in those patients with the longest disease duration. Serial bone-mineral density studies demonstrate the markedly delayed resolution of femoral head and neck osteoporosis despite apparent resolution from the symptomatic and the radiographic point of view. It took 25 to 31 months for the right and left femoral bone densities to equalize (7).

The Degree of Bone Loss in Algodystrophy

The degree of bone loss in algodystrophy over a few weeks or months is what might be expected in 10 years in the course of uncomplicated osteoporosis (5). Are there cases of algodystrophy with no loss of bone? This is still an open question. However, the discussion as to whether or not there are cases of algodystrophy without bone demineralization will soon be made obsolete by the new methods of evaluation of mineral content. If the demineralization is delayed, is it only related to disuse? Bone loss in algodystrophy occurs more markedly in trabecular bone, but increased endosteal resorption of cortical bone is also a feature of the disease. The decrease in bone density at the cortical site is associated with a decrease in cortical width. The loss in the cortical area is caused by endosteal resorption of bone, and the loss of apparent density results from intracortical resorption. The loss of trabecular bone results not only from a decrease in trabecular width but also from the disappearance of some trabeculae. Ac-

ording to Bickerstaff *et al.* (5), recovery of the BMD to normal in algodystrophy would probably only be achieved by hypertrophy of the remaining trabeculae with the persistence of abnormal trabecular architecture. This appears to be confirmed by the x ray appearance of the focal affected area in some cases of algodystrophy after recovery in which the bone resembles pagetic bone.

Fractures and Algodystrophy

On the one hand, an initial fracture is undoubtedly an initiating event in the appearance of algodystrophy. This is well demonstrated for fractures of the upper limbs (shoulder, Colles' fractures). This is true also for cortical fractures as well as trabecular fractures of the lower limbs, with a pitfall clinical situation for stress fractures, which initially remain radiographically occult. These stress fractures caused by repetitive application of force to a bone, the magnitude of which is not sufficient to cause an acute fracture, include: (i) fatigue fractures following unusual, repetitive physical activity involving a bone of normal strength (normal elastic resistance) and (ii) insufficiency fractures which result from application of normal stress or torque to a bone with deficient elastic resistance (osteoporosis, rickets or osteomalacia, hyperparathyroidism, osteogenesis imperfecta). On the other hand, patients suffering from algodystrophy may still have significant regional osteoporosis for a long period (more than a year) and hence be at risk for insufficiency fracture despite the appearance of symptomatic resolution. Serial evaluation of bone-mineral content of the affected algodystrophic area would appear to be useful for the follow-up of these patients, since there is a well-known relationship between the risks of fracture and bone-mineral content.

The initial healing phase of fractures is frequently associated with significant local bone resorption. *When algodystrophy occurs, the signs and symptoms are present in an area larger than the fracture area, including the area distal to the fracture.*

AUTONOMIC TESTING IN ALGODYSTROPHY

Abnormal Thermoregulatory Responses

Thermographic abnormalities must be interpreted very cautiously. A standard thermal stress test consists of the immersion of a hand unaffected by algodystrophy in water at 15°C for one minute (14). The time lag between the end of the cold challenge and the onset of rewarming was significantly increased in the patients (median 5.67 min, range 0.50 to > 15 min) compared to the controls (median 0.50 min, range 0.50 to 12.92 min). The median maximum temperature recovery was lower in the patients than in the controls. These findings support an association between algodystrophy and a generalized abnormal response to cold challenge. These authors believe that early screening for thermoregulatory dysfunction in patients after fractures might allow for the identification of patients at risk of developing algodystrophy. In 11 patients with frozen shoulder and 17 controls, a 15°C cold pack was held against the skin for 60 sec (14). Both prior to and immediately following the cold challenge, the shoulder skin temperature tended to be lower in the patients. During a 10-min rewarming phase, the intergroup temperature differences increased significantly.

The Sweat Test

Chelimsky *et al.* (9) have proposed three autonomic tests in algodystrophy: (i) **Resting skin temperature** measured at standard points on the extremities (14 sites for the upper limbs, 18 sites for the lower limbs). (ii) **Resting sweat output** determined by a capsule attached to the skin, with nitrogen flowing in and out at 100 mL/min. Both incoming and outgoing nitrogen flows through an evaporative water-loss unit which compares the heat conductances. The difference between the two values at 50°C generates a voltage that is linearly related to the water content of the air. The resting sweat output is continuously monitored for 5 minutes. (iii) **Sweating elicited by axon reflex.**

The stimulation of a sweat gland by a cholinergic agent activates a distant sweat gland through an axon reflex. A 2-mA current is used to force acetylcholine into the skin. An increased resting sweat output was found to predict algodystrophy with 94% specificity in a group of 396 patients. However, in an editorial in the same issue of the Mayo Clinic Proceedings, Ochoa stated that the sweat test was unlikely to signal a specific disease process (20).

VASCULAR INVESTIGATIONS

Evaluation of Changes in Dermal Microcirculation

Finger-skin microcirculation and its reactions to sympathetic stimuli can be investigated. Nail-skin capillary blood cell velocity is measured by videophotometric capillaroscopy. Laser doppler fluximetry is used to provide an index of skin circulation in vessels in addition to the superficial capillaries (i) at rest, with the patient in the supine position and the hand at the level of the sternal notch; (ii) following lowering of the hand to 40 cm below heart level (60 sec); (iii) during cooling of the contralateral hand in water at 4°C for 60 seconds. The temperature of the room is kept between 21 and 23°C. The patients rest in a supine position for 20 minutes before investigations. When a normal extremity is lowered, local blood flow is reduced by a peripheral sympathetic axon reflex, leading to local arteriolar constriction. This reflex is probably also enhanced by a local myogenic response to the increased arteriolar transmural pressure. Capillary blood cell velocity and laser doppler fluximetry measure the blood flow in the microcirculation of the outermost layer of the skin. The other measurements, including skin temperature, reflect what happens in the blood vessels below this layer. Rosen *et al.* (24) found significant decreases in capillary blood cell velocity and laser doppler flux values in 12 patients with algodystrophy, compared with 11 healthy controls even though the skin temperature was the same in both groups. During cooling of the contralateral hand, capillary blood velocity and laser doppler fluximetry decreased markedly (22

to 60%) in the controls but not in the patients (0 to 13%). The decrease in skin perfusion normally seen upon lowering of the hand was also impaired in the patients (7%) compared with controls (42%). In this study we should take into account the long delay between the appearance of the symptoms and the investigations (mean duration: 37 months). In earlier stages of algodystrophy some investigators have reported increased subcutaneous and regional blood flow in the affected extremity compared with the control (8, 10, 19). Cutaneous resistance vessels, AV shunts and veins are richly innervated with sympathetic vasoconstrictor nerves. The vasoconstrictor response to an increase in venous pressure corresponds to a mechanism for maintaining capillary pressure and filtration within normal limits. Edema in patients suffering from algodystrophy may result partly from a defect in this protective mechanism. The venoarteriolar reflex thus seems to be closely involved and deserves further study.

Arteriography in Algodystrophy

Some patients with algodystrophy in one distal lower limb seem to have acute ischemia. A vascular disease, such as a vasculitis, might be evoked in this pitfall situation, especially in adolescent and young female patients, with no increase in radionuclide uptake on bone scan. Arteriography in one such case revealed a regular distal arterial narrowing (12).

MUSCLE AND NEUROLOGICAL INVESTIGATIONS

Muscle Investigations (29)

Muscle atrophy in the affected part of the limb is a well-known sign of algodystrophy. An impairment of high-energy phosphate metabolism is demonstrated by ³¹P NMR spectroscopy. Electron microscopic studies show reduced mitochondrial enzyme activity, vesiculation of mitochondria, disintegration of myofibrils, abnormal deposition of lipofuscin, swelling of endothelial layers and thickening of the basal membrane. It has been

observed that patients with algodystrophy are unable rather than unwilling to exercise.

Current electromyographic studies show minimal nonspecific neuropathic and myopathic changes. In a case of regional migratory osteoporosis, a denervation pattern coincident in time and location of each acute attack has been documented (11).

Neurological Investigations

Most cases of algodystrophy follow minor injuries to those regions that are particularly rich in nerve endings. These areas, known as "watershed" zones, are the hand, wrist, dorsum of the foot, knee, and so on (6).

From the neurological point of view, algodystrophy raises questions concerning the role of injury to the smaller nerves and/or to nerve terminals, the degree of increased sensitivity to substances released by local nerves, the sensitivity to pressure and to the sympathetic amines of nerve growth sprouts from injured nerves, the evaluation of increased orthodromic impulses on both A and C fibers, and the evaluation of constant (?) bursts of activity particularly in response to touch or light pressure on new free nerve terminals. The difference between algodystrophy and causalgia is linked to the diameter of the involved nerves: injury to the larger nerves produces causalgia whereas injury to the smaller nerves and nerve terminals may be implicated in algodystrophy.

CONCLUSION

No single procedure or laboratory test can determine the diagnosis of algodystrophy. Even the "three-bone scan" is nonspecific for algodystrophy. Transient hyperpermeability is well demonstrated by the clinical findings, the MRI signs, and the three-bone scan features. A ^{99m}Tc EHDP bone scan affords an evaluation of the vascular abnormalities (on each of the three scans) and of the osteoblastic activity (on the third scan). Vascular abnormalities can also be investigated by the new methods, especially for dermal microcirculation. Perhaps the sweat test does

unveil what might be specific about algodystrophy, but the value of autonomic testing in algodystrophy has to be better evaluated. Bone involvement is an important feature of algodystrophy. However in some patients, osteoporosis occurs in a few weeks and in others only after several months, with perhaps different mechanisms of demineralization. Histologic bone demineralization deserves further study, especially in the distal localization of algodystrophy but bone biopsy can be performed only in special cases. Densitometry is certainly a very precise way of evaluating the degree of local or regional demineralization, and it could be an aid in diagnosis and probably help in monitoring treatment as well. Further investigations are still needed in algodystrophy for a better understanding of this condition so as to improve its management.

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SAMENVATTING

C. MASSON, M. AUDRAN, C. PASCARETTI, A. NAMOUR, J. L. SAUMET, M. F. BASLE, E. LEGRAND, C. BREGEON, J. Cl. RENIER. Verdere onderzoeken in algodystrofie.

Klinische observatie is de meest frequente manier om de diagnose algodystrofie te stellen. Verdere onderzoeken laten toe om andere pathologieën uit te sluiten zoals stress fracturen en avasculaire botnecrosen en om een beter inzicht in de algodystrofie te verkrijgen. Transiënte hyperpermeabiliteit in het getroffen deel, is duidelijk door het klinisch beeld, MRI and 3 fasen botscaan. 99m Technesium ENDP bot scan evalueert de vasculaire abnormaliteiten en de osteoblastische activiteit. Huidmicrocirculatie en de reactie op sympatische stimuli worden onderzocht door laser doppler fluxmetrie en videofotografische capillaroscopie. Waarschijnlijk is de zweetest in staat om de specifieke eigenheid van algodystrofie te ontsluiten. De hoeveelheid botverlies in algodystrofie in enkele weken of maanden is wat men verwacht na 10 jaar ongecompliceerde osteoporose.

Al is een fractuur vaak het uitlokkend gebeuren in algodystrofie, hebben patiënten gedurende jaren te maken met een duidelijk osteoporose met het gevaar voor fracturen. Bot densitometrie kan een hulp zijn in de diagnose, maar ook in de monitoring van de behandeling. De lokale kolonisatie door fibroblasten na de transiënte fase van hyperpermeabiliteit laat toe de effecten op gewrichts-, bot-, spier- en zenuwonderzoeken te verklaren.

RÉSUMÉ

C. MASSON, M. AUDRAN, C. PASCARETTI, A. NAMOUR, J.-L. SAUMET, M.-F. BASLE, E. LEGRAND, Ch. BREGEON, J. Cl. RENIER. Investigations complémentaires dans l'algodystrophie.

L'observation clinique directe est le moyen de diagnostic le plus habituel de l'algodystrophie. D'autres investigations peuvent être utiles pour exclure certaines pathologies comme des fractures de fatigue ou par insuffisance osseuse, une ostéonécrose aseptique et pour mieux comprendre l'algodystrophie. L'hyperperméabilité vasculaire transitoire dans la zone affectée est bien démontrée par l'examen clinique, les signes IRM et la scintigraphie osseuse en trois temps. La scintigraphie osseuse par l'EHDP 99m Technetium permet une évaluation des anomalies vasculaires et de l'activité ostéoblastique. Les flux de microcirculation dermique, leurs relations avec le système sympathique sont explorés par le laser doppler et la capillaroscopie vidéophotonique. Le test de la sueur pourrait objectiver ce qui est le plus spécifique de l'algodystrophie. L'importance de la perte osseuse survenant en quelques semaines ou quelques mois dans l'algodystrophie correspond à celle qui se produit en 10 ans d'évolution naturelle d'une ostéoporose post-ménopausique. Une fracture corticale et/ou trabéculaire initiale est un facteur déclenchant fréquent de l'algodystrophie. Mais les patients souffrant d'algodystrophie peuvent garder une ostéoporose significative pendant une longue période et ainsi être sujets à des fractures par insuffisance osseuse. L'ostéodensitométrie pourrait être une aide au diagnostic et aussi pour la surveillance thérapeutique. La colonisation locale par des fibroblastes suivant la phase initiale d'hyperperméabilité vasculaire doit être gardée à l'esprit pour expliquer les résultats des investigations portant sur les conséquences articulaires, osseuses, musculaires dans l'algodystrophie à sa phase tardive.