CASE REPORT

BONE AMYLOIDOMA IN A DIABETIC PATIENT WITH MORBID OBESITY

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SUMMARY - Bone localisations of amyloidosis are rare, usually diffuse and associated with myeloma. We report the case of a patient with massive obesity complicated by diabetes, hypertension, sleep apnea and liver steatosis, who complained of rapidly worsening bilateral polyradiculalgia of the lower limbs. After sufficient weight loss made nuclear magnetic resonance imaging feasible, a spinal tumour was visualised on the 5th lumbar vertebra, extending to soft tissues. Total excision was performed, and pathological studies revealed an amyloid bone tumour with no evidence of myeloma.

Key-words: amyloidoma, diabetes, neuropathy, morbid obesity.

RÉSUMÉ - Amylose osseuse chez un patient diabétique avec obésité morbide. Les localisations osseuses de l’amylose sont rares, le plus souvent diffuses et associées au myélome. Nous rapportons le cas d’un patient présentant une obésité massive compliquée d’un diabète, d’une hypertension artérielle, d’un syndrome d’apnée du sommeil et d’une stéatose hépatique, se plaignant de polyradiculalgies bilatérales des membres inférieurs s’aggravant rapidement. Après un amaigrissement suffisant l’imagerie par résonance magnétique devenue possible a montré une tumeur osseuse lombo-sacrée en sablier, centrée sur la cinquième vertèbre lombaire et s’étendant aux parties molles. Après exérèse totale l’examen anatomopathologique a permis le diagnostic de pseudo-tumeur amyloïde localisée à l’os, chez un patient ne présentant aucun signe de myélome.

Mots-clés : pseudo-tumeur amyloïde, diabète, neuropathie, obésité morbide.

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Amyloidosis is defined as the extracellular deposit of a fibrous protein which constitutes the amyloid substance in one or several tissues [1]. Various types have been described: primary amyloidosis and amyloidosis associated with multiple myeloma (both of the AL subtype); amyloidosis secondary to infectious or chronic inflammatory diseases (AA subtype); and isolated amyloidosis in an organ without systemic disease. Amyloidosis in bone is infrequent, and usually diffuse in patients with multiple myeloma and subjects undergoing chronic dialysis [2]. We report the extremely rare case of a Type 2 diabetic patient with morbid obesity and polyradiculalgia of the lower limbs, who presented pseudotumoural amyloidosis in bone without any clear etiology.

### Case report

A 45-year-old man was referred to our department in January 1993 for morbid obesity. His medical history included rheumatic fever in childhood and an abdominal lipectomy six years before, complicated by eventrations. He weighed 190 kg and was 1 m 74 cm tall (body mass index: 62.8 kg/m²). Obesity was of android type (waist-to-hip ratio: 1.2), mainly complicated by Type 2 diabetes which was diagnosed in 1991 and poorly controlled by glibenclamide 15 mg and metformin 1,700 mg in the absence of a suitable diet. His hypertension was poorly controlled by calcium-channel blockers, and he suffered from joint pain in the lower limbs. He also complained of recent sexual impotence. Neurological examination was normal, and neurophysiological investigation showed early signs of right carpal tunnel syndrome and sensorial neuropathy of the lower limbs. Biological investigations revealed a moderate inflammatory syndrome associated with multiple myeloma (both of the AL subtype); amyloidosis secondary to infectious or chronic inflammatory diseases (AA subtype); and isolated amyloidosis in an organ without systemic disease. Amyloidosis in bone is infrequent, and usually diffuse in patients with multiple myeloma and subjects undergoing chronic dialysis [2]. We report the extremely rare case of a Type 2 diabetic patient with morbid obesity and polyradiculalgia of the lower limbs, who presented pseudotumoural amyloidosis in bone without any clear etiology.

**Discussion**

Although all organs and joints (in particular) may be affected by amyloidosis, focused bone localisations have been reported only rarely [2–4]. Bone amyloidosis is usually diffuse, producing various types of clinical and radiological images: osteoporosis, destruc-
tive lesions, pathological fractures, osteonecrosis, nodes and inflation of soft tissues, cysts and subchon-
dral erosions, and joint subdislocations with contrac-
ture and neuropathic osteoarthropathy [5]. Amyloid
deposits in the skeleton are often associated with mul-
tiple myeloma related to diffuse amyloidosis with plasmacytic infiltration [2]. Amyloid bone tumour is
very rare, corresponding to the accumulation of amy-
loid material in an amount sufficient to destroy bone
and become visible on radiography, mainly in the
form of destructive lesions, often with fractures [1].
All bones may be affected, especially long ones, the
spine and the base of the skull [6, 7]. Symptoms are
various and non-specific, ranging from pain to com-
plete paraplegia [8]. Other diagnoses may be consid-
red, mainly primary bone tumour, metastasis or meta-

dolic diseases such as gout. Macroscopically, a solid
homogeneous yellow or brown mass is apparent. Mi-

croscopic examination shows lifeless, vitreous eosino-

philic material, sprinkled with giant multinuclear
cells, histiocytes and plasma cells [9]. Several stains
have been used to distinguish between amyloidosis and
other kinds of proteins, but the most common one is
the Congo red preparation which shows red-green

dacent phenomenon in polarising microscopy. The plasma
cells found in these amyloid tumours suggest a possi-
ble link with solitary myeloma [10].

Multiple myeloma is by far the main etiology for
these localised lesions, but in a few cases large bone
amyloid deposits have been found without plasma cell

filtration [1]. In some cases, no cause has been
determined, despite Bence-Jones proteinuria or extra-

bone localisations (heart, kidney, pancreas, nodes) [9].
Subjects under chronic dialysis are at great risk for
amyloid tumours due to the deposit of β2-

microglobulin in the skeleton, which usually causes
large destructive lesions leading to fractures [11]. In
our patient, apart from a moderate inflammatory syn-
drome, there was no evidence of myeloma. He never
exhibited clinical or radiological symptoms suggestive
of another bone disease, nor had significant proteinu-

ria. Moreover, histological examination failed to show
amyloid localisation in the liver.

This case report illustrates another effect of mas-
vative obesity. In addition to the classical metabolic,
cardiovascular and rheumatological complications, the
patient’s massive overweight condition made it im-
possible to use the most up-to-date imaging techni-

ques, such as CT-scan or MRI. This may seem anec-
dotal, but the consequences can be crucial in life-

threatening circumstances when these diagnostic
means are essential. In our patient, if it had been
possible to make the diagnosis earlier, many months
of suffering and disability would have been avoided
and surgery would have been safer and less traumatic.
Moreover, the patient’s morphology, in addition to
presenting a marked anaesthetic risk and obvious
 technically difficult related to the impressive thick-


ness of fat tissues, had a definite influence on the
surgical procedure itself since arthrodesis could not be
considered as the first indication. Arthrodesis is per-
formed to avoid secondary spondylolisthesis, but the

genupectoral position must be maintained for at least
five hours, which greatly increases the risk of postop-
ierative rhabdomyolysis.

Polyradiculalgias are a rare manifestation of diabe-
tic neuropathy. This case report indicates the need to
exclude causes of compression by making an appro-

priate assessment.

REFERENCES