ORIGINAL ARTICLE

Osteoporosis in Persons With Spinal Cord Injury: The Need for a Targeted Therapeutic Education

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ABSTRACT. Fattal C, Mariano-Goulart D, Thomas E, Rouays-Mabit H, Verollet C, Maimoun L. Osteoporosis in persons with spinal cord injury: the need for a targeted therapeutic education. Arch Phys Med Rehabil 2011;92:59-67.

Objectives: To identify circumstances surrounding the onset of fracture and common risk factors in persons with spinal cord injury (SCI) and to suggest an alternative or complement to the pharmacologic approach by evaluating the need for a prospective study based on the impact of a targeted therapeutic education on risk management of fractures in this population.

Design: Retrospective study.

Setting: Hospital and Rehabilitation Center Setting.

Participants: Women (n=7) and men (n=25; N=32; with ≥1 fracture after the initial SCI that occurred at home or in a hospital setting; mean ± SD age, 53±12y at the time of clinical review) with bone mineral density (BMD) measurements.

Interventions: Not applicable.

Main Outcome Measures: Demographics, main circumstances of onset, and complications of fractures, as well as transversal bone mineral density evaluation.

Results: Nine patients had more than 1 fracture and 23 patients had only 1 fracture (total, 43 fractures; mean age at onset of fracture, 49±12y; median time since injury, 13.9y; mean delay in diagnosis, 6.5±15d). Fractures occurred mostly in the lower limbs. The circumstances of onset of these fractures were different and very stereotyped. In 3 cases, no trauma was reported. The most frequent mechanisms identified were forced maneuvers by the patient or a third party and falls. In 10 cases, the fracture occurred during a wheelchair transfer with forced maneuver or a fall from the wheelchair. Twenty-five patients were confined to bed after the fracture (mean duration of bed confinement, 18±28d; range, 0–120d). Postfracture follow-up showed that for 43 cases of fractures, 19 had at least 1 orthopedic complication, 15 had local complications, and 23 had general complications. Patients (23 of 32) benefited from dual-energy X-ray absorptiometry to assess BMD a few months or years after the fracture (mean femoral neck BMD, $0.574\pm0.197g/cm^2$; mean femoral neck T score, −3.8±1.5).

Conclusion: With this retrospective analysis of common risk factors and circumstances of onset of secondary fractures, there is a clear future for a prospective study to evaluate the impact of targeted therapeutic education on risk factors for secondary fractures in patients with SCI.

Key Words: Fracture; Osteoporosis; Prevention; Rehabilitation; Spinal cord injuries; Therapeutic education.

FOR PATIENTS WITH SCI, fractures and their consolidation cause several medical challenges (eg, general, functional, psychological, orthopedic, neurologic, infectious). Vestergaard et al[1] and Frisbie[2] conducted studies with a 21-year follow-up on their cohorts and reported incidences of fracture of 31% and 33% for at least 1 fracture below the level of injury, respectively. Furthermore, as shown in the literature, this risk seemed to increase according to the age of the initial injury (the older the injury, the higher the risk).[3,4] These data unveiled the need to identify and define risk factors for the onset of fracture in a context of patients with SCI and associated osteoporosis. For these patients, the cause of osteoporosis is different from that in menopausal women. Numerous studies focused on BMD, as well as the biochemical and clinical reality of osteoporosis and its consequences.[1,5-9] Data showed that the bone loss process started from the first day postinjury and peaked at 3 to 6 months postinjury.[10,11] Afterward, bone loss reached a plateau at 16 months postinjury, to amount to a 50% BMD decrease compared with preinjury values in these patients.[7,9] Furthermore, pharmacologic approaches and several publications highlighted some SCI-specific mechanisms and labeled this bone loss neurologic osteoporosis.[5] The efficacy of bone-protector drugs has not been validated for the chronic phase[12] and has been suggested only during the first year post-SCI. However, physicians are still very reluctant to prescribe drug treatments for bone protection.[13]

Currently, no physical rehabilitation program, including standing exercises, validates a significant impact on slowing down bone loss in persons with SCI at higher risk for substantial and early bone loss.[5] The great advances in therapeutic education during the past few years did not focus enough on osteoporosis in the general population and rarely in persons with SCI. The literature reported the various mechanisms that

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List of Abbreviations

ASIA	American Spinal Injury Association
BMD	bone mineral density
BMI	body mass index
DXA	dual-energy x-ray absorptiometry
FES	functional electrical stimulation
HT	hormone therapy
PM&R	physical medicine and rehabilitation
SCI	spinal cord injury

correlated with the onset of fractures, yet very few publications tried to classify these mechanisms to set the basis for defining major prevention areas for a future therapeutic educational program. In consequence, the objectives of this study were to evaluate the circumstances surrounding fractures in subjects with chronic SCI and establish a therapeutic educational strategy targeted on prevention.

METHODS

The study focused on the most recent 32 cases of upper-and/or lower-limb fractures below the level of injury in patients with chronic SCI admitted to the Centre Mutualiste Neurologique Propara (within the SCI Unit).

The study was conducted on a retrospective (review of patients' medical charts completed by an interview) and a transversal level (DXA) to evaluate BMD and osteoporosis at a distance from the fracture.

For this study, the following inclusion criteria were defined: adult patients; persons with chronic SCI; ASIA grades A, B, or C; fracture below the level of injury; and documented therapeutic care of the fracture. Patients received detailed oral and written information and then signed a consent form, based on authorization from the local ethics committee.

Exclusion criteria were ASIA grade D, fractures above the level of injury, impossibility to interview the patients (not available, no more contacts, etc), or lack of relevant documents in their medical charts.

For the retrospective approach, data collected were:

- Demographics.
- Clinical and functional data at the time of fracture.
- Fracture characteristics.
- Mechanisms and circumstances of onset.
- Fracture-related complications (eg, orthopedic, local, or general complications).
- Duration of hospital stay needed for treating the fracture.

Data were collected from patients' medical charts and completed with an individual interview with each patient. The collection grid was tested previously on 5 medical charts to make sure that the questions asked during the interview were clearly understandable and relevant. The statistical approach was mainly descriptive, and percentiles were computed to clarify results.

The transversal approach was based on full-body BMD measurements. To evaluate BDM (in grams per centimeter squared) at the anterior-posterior lumbar spine, the distal extremity of the radius, and femoral neck, DXA techniques were used with the same material[a] and acquisition software. T scores ± SD were calculated for each site. For BMD, the laboratory precision error is 1% at the lumbar spine, less than 1% at the femoral neck, and less than 1% at the forearm.

RESULTS

Demographics and Clinical Data

Patients (N=32) were selected in the early or late follow-up stage of a fracture below the level of injury (table 1). There were 7 women and 25 men (mean ± SD age, 53±12y at the time of clinical review and BMD measurements).

No patient had previous risk factors for bone demineralization (eg, steroid therapy, seizures, such aggravating pathologic states as parathyroid or thyroid disease, excessive alcohol intake). Five patients had a family history of osteoporosis. Of the 32 patients, 24 were heavy smokers or former heavy smokers (>5pack-years). No patient had ever benefited from osteoporosis prevention treatment. For the group of 7 women, mean age at fracture onset was 54 years (range, 37–61y). Median BMI for these patients was 25.4kg/m^2.

Table 1: Demographics and Clinical Data

Variable	Value
N	32
Sex (men/women)	25/7
Age at SCI onset (y)	34±13 (14–57) 33 (47)*
SCI origin (trauma/vascular)	31/1
Neurologic profile (paraplegia/tetraplegia)	23/9
ASIA grade (A/C)	29/3
FIM score	93±20.6 (50–121) 97 (110)*
SCIM score	51±15.5 (26–73) 55 (65)*
Gait	
Walking patients	0
Exclusive wheelchair dependent	28
Electric wheelchair	3
Manual + electric wheelchair	1
Manual wheelchair	24
Mixed ambulation mode (walking/wheelchair)	4
BMI (kg/m^2)	25±6 (14–39) 25 (29)*
No. of patients/no. of fractures (P/T)	23/1 (16/7) 7/2 (6/1) 2/3 (1/1)
Age at onset of fracture (y)	49±12 (20–77) 49.5 (55)*
Time from SCI to fracture onset (y)	16.3±12.5 (0.5–41) 13.9 (27.5)*
Delay in fracture diagnosis (d)	6.5±15 (0–60) 1 (3)*

NOTE. Values expressed as number of patients or mean ± SD (range) unless noted otherwise.
Abbreviations: P, patient with paraplegia; SCIM, Spinal Cord Independence Measure; T, patient with tetraplegia.
*Median (75th percentile).

At the time of BMD measurements and DXA, none of the women was using a contraceptive method. One woman was on HT, 1 woman previously had been on HT but stopped, and 5 women never were on HT. Two women were using bone-protection drugs.

At the time of fracture, 6 of 7 women were menopausal, 1 woman was on HT that she later discontinued, 1 was still on HT, and 1 had an early menopause onset at the age of 28 years.

Fracture-Related Data

Seven patients had 2 successive fractures, 2 patients had 3 successive fractures, and the other 23 patients only had 1 fracture, for a total of 43 fractures (see table 1). The 2 main locations reported for these 43 fractures were femur (27 cases) and tibia (16 cases) (fig 1). Median age at onset of fracture was 49.5 years. Median interval since SCI was 13.9 years. Mean delay in diagnosis was 6.5±15 days. In 3 cases, the fracture was discovered fortuitously.

The circumstances of onset of the fractures (table 2) were linked to various mechanisms. In 3 cases, no trauma was reported. However, for the other cases, it appeared that patient-elicited forced maneuvers and third-party mobilizations were, with falls, the most frequent circumstances surrounding the

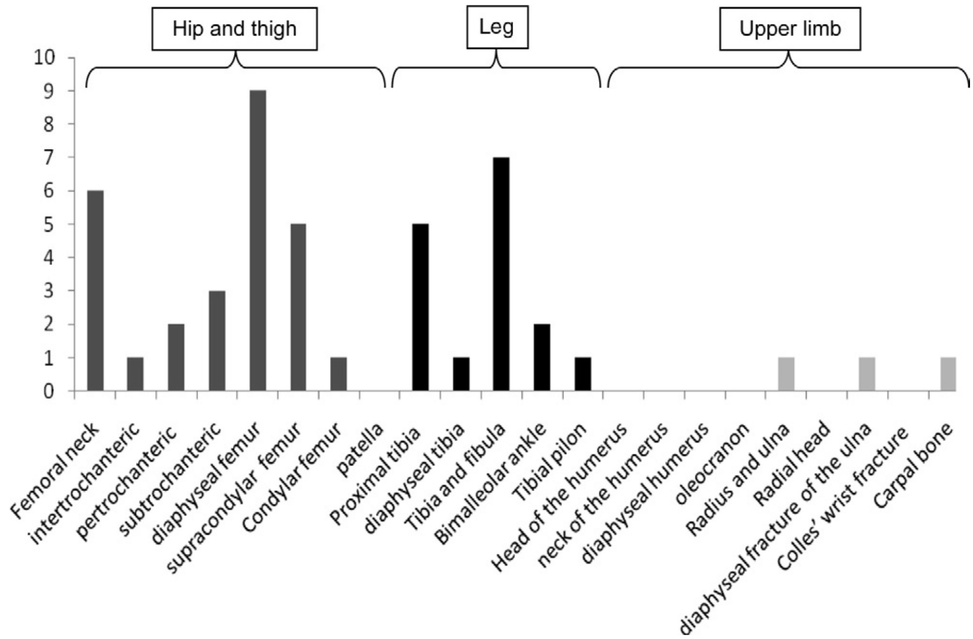

Fig 1. Fracture locations.

fracture. In 10 cases, the fracture occurred during wheelchair transfers with either forced maneuvers, forced mobilizations, falls, and sometimes a combination of these.

Fracture Complications

For 32 of 43 cases, the fracture required orthopedic treatment, alone or before surgical consolidation. In 18 cases, the fracture required surgical treatment. Twenty-five patients were confined to bed after their fracture (mean duration of bed confinement, 18±28d; range, 0–120d). In 34 cases, therapeutic care required a hospital stay in the SCI unit. Patients were forbidden to perform unassisted transfers in 33 cases for a mean of 65±39 days (range, 3–150d). During postfracture follow-up, of 43 cases of fractures, 19 led to at least 1 orthopedic complication, 15 had at least 1 local complication, and 23 had at least 1 general complication. Orthopedic complications (fig 2) were dominated by consolidation disorders (pseudoarthritis, late consolidation, heterotopic ossifications) and secondary joint stiffness. Local complications (see fig 2) essentially were due to skin damage caused by orthoses or fiberglass casts. General complications (see fig 2) consisted of pressure ulcers on the pelvis, anemia, and recrudescence of neurologic disorders (spasms, autonomic dysreflexia, neuropathic pain).

Bone Mineral Density

Twenty-four of 32 patients agreed to DXA. One DXA scan could not be interpreted. One patient died between the clinical check-up and the day of BMD measurements. The other 8 patients either declined the imaging examination or could not be reached. For the 23 patients who benefited from BMD measurements, median delay post-SCI was 19 years (range, 1–43y), and median age at examination was 51 years. Mean BMD and mean T score for each site are listed in table 3. These data highlighted a median BMD at the femoral neck of .561g/cm^2 and a median femoral neck T score of −3.9. Nine of 23 patients had a femoral neck T score estimated at −2 to −3.5 SD, and 13 had a femoral neck T score of −3.5 SD or less. One patient had normal results (patient 13). In patients with tetraplegia and paraplegia, a BMD decrease in the radius was reported with a mean T score of −1±1.5 (range, −4.9 to 1.4).

DISCUSSION

Characteristics of Fractures Below the Level of Injury in Persons With SCI

The onset of fracture below the level of injury often correlates with minor or low-impact trauma. By focusing patients' interviews on the circumstances surrounding the onset of fracture, the goal was to identify its mechanisms for defining and setting the basis for an educational therapeutic strategy targeted on prevention.

For this study, results matched those reported in the literature[4,14]; fractures were located mostly in the lower extremities. The predominant reported location was above the knee.[4,15,16] The relative fracture risk for the femur was deemed higher than for other locations, estimated at 23.4 versus 5.2 for legs and 2.4 for feet.[1] Two areas were identified. First, above the knee, fractures mostly occurred in the supracondylar region and diaphyseal femur, and second, below the knee, affecting the proximal tibia. Freehafer,[17] in a series of 99 fractures in 133 patients, reported 15 fractures located in the upper extremities. This element from the literature should correlate with the BMD decrease in the radius for some patients (see table 3).

To identify potential arguments and elements to design and implement a standardized and targeted therapeutic education strategy for fracture prevention, this study focused mainly on (1) mechanisms and circumstances of onset for each fracture, and (2) morbid complications of these fractures.

Two main mechanisms (forced maneuvers, forced mobilizations) and 2 predominant circumstances of onset (transfer, stretching exercises) were identified as fracture risk factors.

These results show the need to differentiate 3 at-risk situations.

1. Well-identified and diagnosed traumas: usually caused by direct involuntary impact (eg, falling or falling/sliding from the wheelchair or bed and/or traffic accidents). Several investigators[1,15,16,18] also agreed that

Table 2: Description of Mechanisms Involved in Bone Fracture

Patient No.	Mechanism of Bone Fracture	Circumstances of Onset
1	Forced maneuver	While skiing/mechanical constraint or overloading on part of the limb
2	Fall	WC tipped over (wheel stuck in a hole)/mechanical constraint or overloading on part of the limb
3	Fall-slide	During WC-bed transfer
4	Forced maneuver	While getting dressed
5	Fall-slide	While walking
6	Direct contact	Bump on knees
7	Forced maneuver	Leg stuck in elevator door
8	Forced maneuver	Transfer: ankle blockage and twisting
9	Forced maneuver	While skiing/direct bump on knee and forced movement in extreme valgus and external rotation and flexion of the knee
10	Slow-impact direct bump	NA
11	Forced maneuver	Transfer:
12	Forced maneuver	Right foot on the left knee to stop a muscle spasm
13	Fortuitous discovery	NA
14	Forced maneuver	Anteflexion of the trunk before transfer
15	Forced solicitation	Stretching exercises by physiotherapist
16	Forced solicitation	Stretching exercises by physiotherapist
17	Forced maneuver	Crossed-leg sitting position
18	Fall	From his/her own height (walking with canes)
19	Forced maneuver	Transfer: Blockage of leg and torsion
20	Fall during a transfer	Transfer: Mechanical constraint or overloading on part of the limb
21	Forced maneuver	Forearm stuck in car’s driving wheel when used as lever to self-lift from driver seat
22	Forced maneuver	During WC-floor transfer, foot blocked, axial rotation of the leg/sudden high-impact movement
23	Forced solicitation	Stretching exercises by third party/hyperextension of knee to fight a muscle spasm
24	Motor vehicle collision	Driver
25	Motor vehicle collision	Driver
26	Fall	On the floor from WC
27	Fall-slide	From WC/mechanical constraint or overloading on part of the limb
28	Forced maneuver	On the hip when getting dressed (flexed hip and half-extended knee)
29	Fortuitous discovery	NA
30	Forced maneuver	Hyperflexion of knee during transfer
31	Fortuitous discovery	NA
32	Forced maneuver	Stretching exercises by the patient
33	Motor vehicle collision	Hit by car
34	Forced solicitation	Stretching exercises by the patient
35	Forced maneuver	Blockage of the leg and torsion during bed-WC transfer
36	Fall	When trying to get back on the manual WC transfer
37	Fall-slide	From WC and landing on his/her knees
38	Fall	WC tipped over (wheel stopped by obstacle)
39	Forced maneuver	While getting dressed, taking socks off
40	Forced maneuver	While trying to get his/her stuck foot out during transfer from his/her car
41	Forced maneuver	While trying to correct hyperflexion of the knee after the fall on the floor from a stand-up device
42	Forced maneuver	Fall from a handbike, lying on side after falling on his/her thigh
43	Fall	WC tipped over (wheel stuck in hole)/mechanical constraint or overloading on part of the limb

NOTE. N=43 cases.
Abbreviations: NA, not available; WC, wheelchair.

low-impact falls were a predominant circumstance of onset for fractures.

2. Traumas unidentified at first, but incriminated later on: extreme stretching movements and bad posture applying mechanical constraint or overloading on 1 part of the limb. In 10 of 43 cases, traumas correlated with badly performed transfer tasks. This item then was singled out as a high-risk task for falls or other harmful accidents. Furthermore, Morse et al[19] reported that 20% of fractures requiring hospital admission were due to transfer and wheelchair techniques. In most cases, stretching exercises or bad postures aimed at improving joint stiffness and comfort. They also could have been used to ”break” a spasm in extension or flexion or simply because the patient needed to put on his/her shoes or socks, for example.
3. No reported traumas that led to fortuitous or delayed fracture discovery: most times such symptoms as swelling, wrongly positioned lower limb, and/or shortened and deformed lower limb guided the physician toward a diagnosis of fracture. This was a common occurrence, and even if it was estimated at only 7% in this series, it

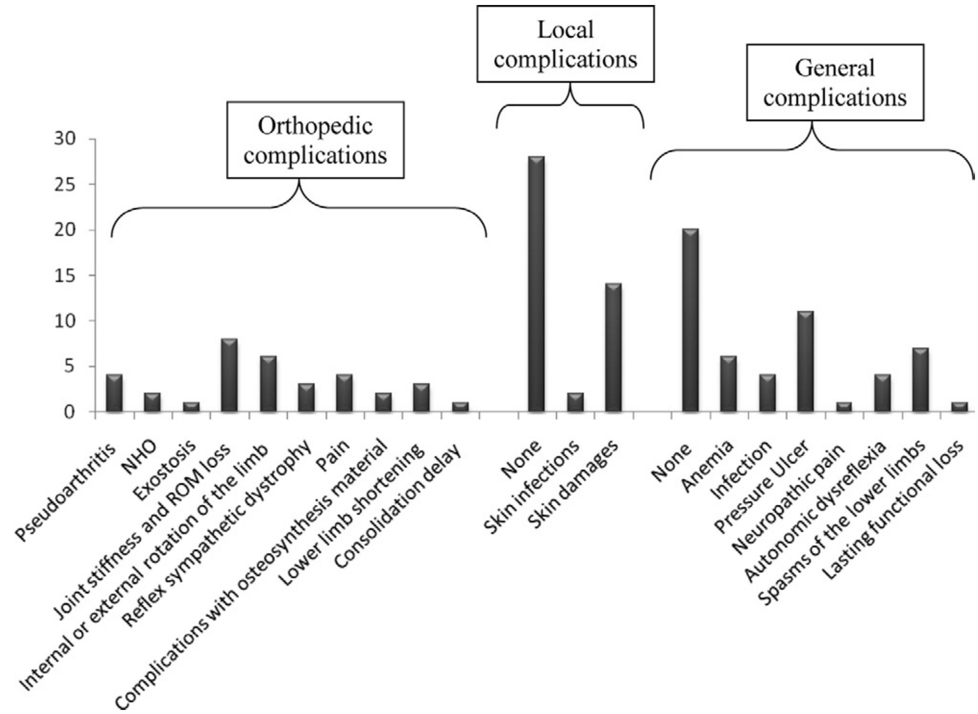

Fig 2. Orthopedic, local, and general complications. Abbreviations: NHO, neurogenic heterotopic ossification; ROM, range of motion.

has been widely reported throughout the literature at about 20%.[1,15,16,18] This element correlated with delayed diagnosis, with a mean estimate of 6 days. However, in a series reported by Ingram et al,[15] the rate varied from 1 to 4 weeks for 36% of patients in a population of 25. Several reasons were identified to explain this delay in diagnosis: misleading first-line symptoms, lack of pain, lack of identified trauma in some cases, and difficult imaging diagnosis for some poorly visible fractures.

These results also highlighted the need to focus on the impact of associated factors. The study by Vestergaard et al[1] of a cohort of 438 individuals with SCI did not report associated factors. Conversely, a recent prospective study[19] of 351 veterans 1 year or longer post-SCI showed a significant linear trend for risk for fracture associated with excessive drinking and a significant correlation between risk for fracture and completeness of SCI (ASIA grade A or B). The responsibility of excessive drinking has not been clearly determined. Further studies are needed to identify whether it increases bone loss below the level of injury or generates accidents (wheelchair maneuvering or transfers). Finally, for some investigators, additional risk factors deserve to be considered:

- Patient age[2,20]
- Age of the original SCI[1,12]
- Time since SCI[21]
- BMI[20]

Physiologic and Biochemical Reality of Osteoporosis in Persons With SCI

In persons with SCI, results from full-body BMD measurement at a distance from the fracture emphasized the persistence of this fracture risk. Of 7 patients who had 2 successive fractures, 1 patient had another fracture within 3 months and 6 patients had another fracture after several years. At the femoral neck level, patients' BMD values were above the fracture threshold, and in some cases, the value was doubled. Only 3 of 7 women benefited from DXA. These 3 women were menopausal at the time of the imaging, which could explain in part a potential cumulative effect with postmenopausal osteoporosis.

The reality of osteoporosis in persons with SCI is well known in the literature. During the first year postinjury, investigators[4,22] reported a severe BMD decrease of about 25% to 50%, mainly in areas rich in trabecular bones, with a BMD decrease estimated at 4% a month (−2%/mo for cortical bone).[10] For Chantraine et al,[23] bone loss was maximal in the first 6 weeks postinjury, and for other investigators,[11] the peak occurred between months 3 and 6 postinjury regardless of the level of injury. However, this bone loss progressed after a year (months 12–16) to finally reach 60% to 70% of normative values for the femoral neck and 40% to 50% for the proximal tibia.[7,9,10,24] Most investigators reported no change in BMD for the upper extremities. However, regarding BMD at the lumbar spine, the present study reported that 10 of 23 patients who underwent DXA had T scores less than −2 at the lumbar spine, challenging the widely admitted idea that in patients with SCI, the lumbar spine is not affected by osteoporosis.[10,24-26] The postinjury delay separating the onset of fracture from the initial SCI can be very short (5.6mo for 1 patient).

Regarding the correlation between osteoporosis and fracture, the fracture threshold was reached at 1 to 5 years post-SCI in the study by Szollar et al[27] and 1 to 9 years in the study by Lazo et al.[4] By comparing 2 groups of subjects with SCI, 1 with and 1 without fractures, Lazo[4] reported a very significant difference between the 2 groups in terms of BMD decrease.[8,28] BMD was reported as a valid element for predicting risk for fractures in patients with SCI.[4]

Table 3: DXA Data

Patient No.	Sex	Neurologic Profile	No. of Fractures	Delay/SCI	Age at Time of DXA	Lumbar Spine L1–4 (g/cm^2)	T Score at Lumbar Spine (SD)	Forearms (Total) (g/cm^2)	T Score Total Foreams	Forearms (1/3 sup) (g/cm^2)	T Score (1/3 sup)	Forearms (Ultra-Distal) (g/cm^2)	T Score (UD)	Femoral Neck (g/cm^2)	T Score at Femoral Neck Level (SD)
1*															
2	M	P	2	25	75	0.737	−3.2	.519	−3.0	.721	−1.8	.383	−2.2	.351	−5.5
3*										NA					
4	M	P	1	4	36	0.768	−2.9	.602	−1.4	.790	−0.6	.428	−1.4	.433	−4.9
5*															
6*															
7	F	T	1	23	51	0.657	−3.5	.314	−4.9	.413	−4.7	.141	−5.3	.151	−6.9
8*															
9	M	P	2	38	60	1.006	−0.8	.611	−1.3	.790	−0.6	.509	0.0	.426	−5.0
10	M	P	1	10	51	1.285	1.5	.659	−0.4	.869	0.7	.460	−0.9	.720	−2.7
11*															
12	F	T	2	31	51	1.422	3.4	.613	1.0	.771	1.5	.383	−0.6	.375	−5.0
13	M	P	1	6	39	1.457	3.3	.578	−1.9	.677	−2.6	.443	−1.2	.990	−0.6
14	M	P	1	3	53	1.105	0.1	.738	1.1	.973	2.5	.497	−0.2	.689	−2.9
15	M	P	2	43	70	0.880	−1.9	.618	−1.1	.811	−0.3	.422	−1.6	.539	−4.1
16*															
17	M	T	3	30	46	0.992	−0.9	.539	−2.6	.738	−1.5	.339	−3.0	.450	−4.8
18	M	P	2	19	49	0.952	−1.3	.752	1.4	.934	1.8	.590	1.4	.646	−3.0
19	F	T	1	9	47	0.803	−2.2	.534	−0.6	.680	−0.1	.344	−1.3	.373	−5.0
20	M	P	1	26	61	1.219	−0.2	.637	−0.8	.825	0.0	.412	−1.7	.753	−2.5
21	M	T	1	24	43	0.857	−2.1	.622	−1.1	.782	−0.8	.443	−1.2	.409	−5.1
22	M	T	1	36	50	0.811	−2.5	.556	−2.3	.727	−1.7	.297	−3.8	.390	−5.2
23	M	P	2	1	51	0.698	−3.6	.556	−2.3	.718	−1.9	.407	−1.8	.792	−2.4
24	M	P	1	2	45	0.959	−1.0	.666	−0.2	.787	−0.7	.449	−1.1	.616	−3.5
25	M	P	1	42	64	1.192	0.9	.674	−0.1	.872	0.8	.452	−1.0	.741	−2.5
26*															
27	M	P	1	14	61	1.239	1.1	.655	−0.4	.759	−1.2	.545	0.6	.753	−2.5
28	M	P	2	12	58	1.208	1.1	.634	−0.8	.809	−0.3	.459	−0.9	.764	−2.4
29*															
30	M	P	1	3	53	1.207	1.1	.687	0.1	.858	0.5	.536	0.5	.748	−2.5
31	M	P	1	7	48	0.803	−3.1	.633	−0.9	.841	0.2	.404	−1.9	.561	−3.9
32	M	P	1	42	63	1.065	−0.2	.631	−0.9	.819	−0.1	.424	−1.5	.527	−4.2
		Mean				1.014	−0.7	.610	−1.0	.781	−0.5	.425	−1.3	.574	−3.8
		SD				0.233	2.1	.088	1.4	.109	1.5	.092	1.4	.197	1.5
		Minimum				0.657	−3.6	.314	−4.9	.413	−4.7	.141	−5.3	.151	−6.9
		Maximum				1.457	3.4	.752	1.4	.973	2.5	.590	1.4	.990	−0.6
		Median				0.992	−0.9	.622	−0.9	.790	−0.3	.428	−1.2	.561	−3.9
		75th Percentile				1.208	1.0	.657	−0.3	.833	0.4	.460	−0.8	.745	−2.5

NOTE. DXA data (in grams per centimeter squared and T scores) were collected for each patient included in the study. Each patient is described according to 4 parameters: sex, neurologic profile, number of fractures below the level of injury, and age of injury.
Abbreviations: F, female; M, male; NA, not available; P, patient with paraplegia; sup, superior; T, patient with tetraplegia; UD, ultra-distal.
*Data were not available.

Primary Prevention of Fractures and Secondary Prevention of Complications: Arguments for a Targeted Therapeutic Education

Data from the literature and results from this study unveiled some essential data and elements specific for persons with SCI:

- Osteoporosis is a reality, occurring early and lasting for the long term.
- There is an early and constant risk for fracture regardless of the patient's age, age of the initial SCI, and additional risk factors.

Patients rarely are aware that osteoporosis is a reality, and health care professionals tend to overlook it. The life expectancy of a patient with paraplegia has increased greatly over the years, and although the risk for fracture is greater in this population, patients take more risks daily and thus cumulate various risk factors.[1,3,14,16] Several investigators also reported that consequences of multiple complications after a fracture should not be underestimated. Morse et al[19] estimated the rate of fracture-related complications at 54%. More than 80% of patients had to be admitted for subsequent hospital stays (mean duration of stay, 66d).[29] Thirty-four of 43 fracture cases required admission to an SCI unit, with a mean duration of 65 days. Circumstances surrounding the onset of fracture are easily identifiable and similar from one patient to the next. This element should be significant enough to open the way for preventive recommendations and setting the basis for a targeted therapeutic education aimed at persons with SCI. Finally, for a long time, this problem was poorly and inadequately addressed. Some studies[8,30,31] have reported the positive impact of standing exercises on preventing bone loss in persons with SCI. However, this impact was contradicted in 2005 in a controlled randomized study[6] with level A scientific evidence that compared a group benefiting from passive standing exercises (30min/d for 12wk) and a group that did not do standing exercises. The study reported that passive standing exercises had no impact on femoral BMD (mean gain, .005g/cm^2; 95% confidence interval, −.015 to .025). This element had already been suggested in prior studies[7,32,33] with less rigorous methods and lower levels of scientific evidence.

The confusion might arise from linking osteoporosis to bed confinement (the correlation with duration of the initial bed confinement period clearly is significant; $P<.009$),[7] yet with no real validated significant impact of standing exercises on preventing BMD decrease. This lack of effect was reported for 2 types of standing exercises: passive standing[6] and FES proposed to patients with paraplegia.[34] Furthermore, it commonly is accepted that when used improperly, FES also correlates with a high risk for fracture.[35] Nevertheless, these 2 types of standing exercises are not the only ones available, and some studies[36] recently conducted in different populations brought up other stimulation techniques (vibrations mainly). Finally, the study by Goemaere et al,[8] which did not have the method reliability of the study by Ben et al,[6] focused on a population of patients with paraplegia walking with a technical aid (orthosis), and the reported result was very different from the previous conclusions, with significantly higher BMD data for these patients.

Thus, trying to slow down the osteoporosis process with only 1 type of prevention method, standing exercises, has been confronted by its lack of validated and lasting efficacy on BMD decrease and fracture risk.[37] The literature review also showed that no controlled randomized study had validated the relevance and harmlessness of treatment with bisphosphonates or other bone-protection drugs during the early stages of SCI.

Because BMD loss is a long-lasting health issue for patients with SCI, there is a real need to implement a therapeutic educational strategy from the acute stage of the SCI.

The main principles of this strategy are outlined next.

Theoretical. Inform persons with SCI of the clinical and physiologic nature of their pathologic state, make them aware of the early and constant risk for fractures, and demystify wrong solutions to real problems.

Functional. Inform patients of at-risk situations and provide cautionary advices for exercises and rehabilitation care as follows.

1. Adjust transfer routine to ensure safer transfers
   By monitoring that feet are positioned properly on the wheelchair's foot rests.
   By using, if necessary, a transfer board or asking for third-party monitoring and/or assistance in case of fatigue and vulnerability or when transfer surfaces are uneven.
2. Reduce the objectives for third-party joint mobilization, self-mobilization, or antispasticity stretching by keeping in mind that the goal is to avoid extreme movements.
3. Avoid nonphysiologic postures, even if dictated by functional necessity, as with the typical example of a cross-legged sitting posture. This posture with 1 hip in flexion and internal rotation and loading on the internal side of the knee is a postural attitude chosen by some patients to decrease the intensity of spastic contractures. It must be excluded.
4. Wear proper shoes to correctly position the feet on the foot rests and remove them from heel loops or toe straps before transfers.
5. Avoid useless risky situations (eg, using the 2 rear wheels of the wheelchair to avoid obstacles when not absolutely necessary or propelling the wheelchair at excessive speed).
6. Promote adapted physical exercises while being aware that these activities can lead to major risks. Individuals playing sports should wear protection on the limbs at risk for fractures (shin guards, padding, etc).
7. Implement antitilting systems at the back of wheelchairs and in general make sure that all equipment is safe.
8. Make the home as accessible as possible to avoid negotiating difficult areas with the wheelchair.
9. Banish all muscular electrical stimulation (FES) without a specific medical recommendation of caution and avoid FES during or just after a muscle spasm.

Medical.

1. Control the aggravating risk factors for osteopenia. It was reported that about two-thirds of patients with SCI have coexisting and secondary causes of osteoporosis.[38,39]
2. Ensure that patients are given proper diet recommendations.
   Adapt their diets:
   - To include calcium. Given the calcium-intake–related risk for urinary tract stones, it is recommended to closely monitor calcium levels in urine, as suggested by Ott,[22] who stated that a supplement of 1000mg/d can be beneficial without adverse effects if calcium excretion is less than 250mg/d.
   - To limit caffeine intake because it is an aggravating factor for excessive calcium excretion in urine.
   - To limit alcohol consumption as an aggravating factor for osteopenia and risk for falls.
   - To lose weight, considering that being overweight and thus applying more loading forces on limbs increases the risk for fractures, even if, paradoxically, low BMI less than 19 kg/m^2 correlated with decreased BMD.[40]

   Limit smoking, a well-known factor implicated in decreasing calcium absorption.
3. Argue the relevance of vitamin-calcium supplement treatment and antiresorptive drugs in regard to risk factors for osteopenia and/or history of a first fracture episode.
4. Avoid drugs that interfere with vitamin D absorption.
5. Avoid drugs that interfere with awareness.
6. Consider traumas to be potentially responsible for fractures, even if low-impact traumas.

Study limitations

There were 2 limitations to this study: 1.) the retrospective nature of the data collected and 2.) the DXA imaging not reporting the BMD at knee level. In the literature, the knee is the location of choice for this imaging examination because it is correlated with the lowest error margin. However, the latter remains acceptable for the hip.

CONCLUSIONS

Fractures in persons with SCI can be costly in terms of functional outcomes. In light of this reality, it is essential to integrate this risk in the therapeutic educational effort and rehabilitation strategies of PM&R teams. This retrospective study emphasized 3 essential facts involved in the onset of fracture during the chronic SCI phase:

1. Mechanisms involving minor or low-energy traumas that could be prevented in daily life situations.
2. The risk is long lasting, and young or elderly patients are not exempted from the risk for fracture.
3. Consequences are orthopedic, local, and general complications.

In light of this reality, it is essential to integrate this risk in the therapeutic educational efforts and rehabilitation strategies of PM&R teams. Clinical research still has some work to do to validate whether fighting bone loss could correlate with decreasing the risk for fracture. Finally, if it is the case, what BMD improvement margin should be aimed for to limit the risk for fracture. This is the reason this study aimed at setting the basis for designing and defining an educational therapeutic strategy targeted at preventing fractures in a context of bone loss for persons with SCI. Although therapeutic education is part of PM&R rehabilitation programs, it seems essential to focus on this specific population of patients with SCI. These results also could open the door for a prospective study in this population evaluating the impact of such a therapeutic education program based on the person's age during the acute SCI phase, comparing 2 arms according to age. It also would be interesting to validate the impact of such a targeted therapeutic education on only an SCI population (persons with paraplegia or quadriplegia) to validate that contrary to popular beliefs, in persons with SCI, the lumbar spine is affected by osteoporosis, and to establish a profile of persons more at risk for fractures and thus refine the therapeutic educational program according to results.

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References

1. Vestergaard P, Krogh K, Rejnmark L, Mosekilde L. Fracture rates and risk factors for fractures in patients with spinal cord injury. Spinal Cord 1998;36:790-6.
2. Frisbie JH. Fractures after myelopathy: the risk quantified. J Spinal Cord Med 1997;20:66-9.
3. Krause JS. Aging after spinal cord injury: an exploratory study. Spinal Cord 2000;38:77-83.
4. Lazo MG, Shirazi P, Sam M, Giobbie-Hurder A, Blacconiere MJ, Muppidi M. Osteoporosis and risk of fracture in men with spinal cord injury. Spinal Cord 2001;39:208-14.
5. Maimoun L, Fattal C, Micallef JP, Peruchon E, Rabischong P. Bone loss in spinal cord-injured patients: from physiopathology to therapy. Spinal Cord 2006;44:203-10.
6. Ben M, Harvey L, Denis S, et al. Does 12 weeks of regular standing prevent loss of ankle mobility and bone mineral density in people with recent spinal cord injuries? Aust J Physiother 2005;51:251-6.
7. Dauty M, Perrouin Verbe B, Maugars Y, Dubois C, Mathe JF. Supralesional and sublesional bone mineral density in spinal cord-injured patients. Bone 2000;27:305-9.
8. Goemaere S, Van Laere M, De Neve P, Kaufman JM. Bone mineral status in paraplegic patients who do or do not perform standing. Osteoporos Int 1994;4:138-43.
9. Garland DE, Stewart CA, Adkins RH, et al. Osteoporosis after spinal cord injury. J Orthop Res 1992;10:371-8.
10. Wilmet E, Ismail AA, Heilporn A, Welraeds D, Bergmann P. Longitudinal study of the bone mineral content and of soft tissue composition after spinal cord section. Paraplegia 1995;33:674-7.
11. Maimoun L, Couret I, Micallef JP. Use of bone biochemical markers with dual-energy x-ray absorptiometry for early determination of bone loss in persons with spinal cord injury. Metabolism 2002;51:958-63.
12. Zehnder Y, Risi S, Michel D, et al. Prevention of bone loss in paraplegics over 2 years with alendronate. J Bone Miner Res 2004;19:1067-74.
13. Ashe MC, Craven BC, Eng JJ, Krassioukov A. Prevention and treatment of bone loss after a spinal cord injury: a systematic review. Top Spinal Cord Inj Rehabil 2007;13:123-45.
14. Ragnarsson KT, Sell GH. Lower extremity fractures after spinal cord injury: a retrospective study. Arch Phys Med Rehabil 1981; 62:418-23.
15. Ingram RR, Suman RK, Freeman PA. Lower limb fractures in the chronic spinal cord injured patient. Paraplegia 1989;27:133-9.
16. Martinez A, Cuenca J, Herrera A, Domingo J. Late lower extremity fractures in patients with paraplegia. Injury 2002;33:583-6.
17. Freehafer AA. Limb fractures in patients with spinal cord injury. Arch Phys Med Rehabil 1995;76:823-7.
18. Keating JF, Kerr M, Delargy M. Minimal trauma causing fractures in patients with spinal cord injury. Disabil Rehabil 1992;14:108-9.
19. Morse LR, Battaglino RA, Stolzmann KL, et al. Osteoporotic fractures and hospitalization risk in chronic spinal cord injury. Osteoporos Int 2009;20:385-92.
20. Garland DE, Adkins RH, Stuart CA. Fracture threshold and risk for osteoporosis and pathologic fractures in individuals with spinal cord injury. Top Spinal Cord Inj Rehabil 2005;11:61-9.
21. Jones LM, Legge M, Goulding A. Intensive exercise may preserve bone mass of the upper limbs in spinal cord injured males but does not retard demineralisation of the lower body. Spinal Cord 2002; 40:230-5.
22. Ott SM. Osteoporosis in women with spinal cord injuries. Phys Med Rehabil Clin N Am 2001;12:111-31.
23. Chantraine A, Nusgens B, Lapiere CM. Bone remodeling during the development of osteoporosis in paraplegia. Calcif Tissue Int 1986;38:323-7.
24. Biering-Sorensen F, Bohr HH, Schaadt OP. Longitudinal study of bone mineral content in the lumbar spine, the forearm and the lower extremities after spinal cord injury. Eur J Clin Invest 1990; 20:330-5.
25. Maimoun L, Couret I, Mariano-Goulart D, et al. Changes in osteoprotegerin/RANKL system, bone mineral density, and bone biochemicals markers in patients with recent spinal cord injury. Calcif Tissue Int 2005;76:404-11.
26. Garland DE, Adkins RH, Rah A, Stewart CA. Bone loss with aging and the impact of SCI. Top Spinal Cord Inj Rehabil 2001; 6:47-60.
27. Szollar SM, Martin EM, Parthemore JG, Sartoris DJ, Deftos LJ. Demineralization in tetraplegic and paraplegic man over time. Spinal Cord 1997;35:223-8.
28. Jaeger RJ, Yarkony GM, Roth EJ. Rehabilitation technology for standing and walking after spinal cord injury. Am J Phys Med Rehabil 1989;68:128-33.
29. Nelson A, Ahmed S, Harrow J, Fitzgerald S, Sanchez-Anguiano A, Gavin-Dreschnack D. Fall-related fractures in persons with spinal cord impairment: a descriptive analysis. SCI Nurs 2003;20: 30-7.
30. Dunn RB, Walter JS, Lucero Y, et al. Follow-up assessment of standing mobility device users. Assist Technol 1998;10:84-93.
31. de Bruin ED, Dietz V, Dambacher MA, Stussi E. Longitudinal changes in bone in men with spinal cord injury. Clin Rehabil 2000;14:145-52.
32. Kunkel CF, Scremin AM, Eisenberg B, Garcia JF, Roberts S, Martinez S. Effect of "standing" on spasticity, contracture, and osteoporosis in paralyzed males. Arch Phys Med Rehabil 1993; 74:73-8.
33. Frey-Rindova P, de Bruin ED, Stussi E, Dambacher MA, Dietz V. Bone mineral density in upper and lower extremities during

12 months after spinal cord injury measured by peripheral quantitative computed tomography. Spinal Cord 2000;38: 26-32.
34. Lai CH, Chang WH, Chan WP, et al. Effects of functional electrical stimulation cycling exercise on bone mineral density loss in the early stages of spinal cord injury. J Rehabil Med 2011;42: 150-4.
35. Hartkopp A, Murphy RJ, Mohr T, Kjaer M, Biering-Sorensen F. Bone fracture during electrical stimulation of the quadriceps in a spinal cord injured subject. Arch Phys Med Rehabil 1998;79: 1133-6.
36. Verschueren SM, Roelants M, Delecluse C, Swinnen S, Vanderschueren D, Boonen S. Effect of 6-month whole body vibration training on hip density, muscle strength, and postural control in postmenopausal women: a randomized controlled pilot study. J Bone Miner Res 2004;19:352-9.
37. Biering-Sorensen F, Hansen B, Lee BS. Non-pharmacological treatment and prevention of bone loss after spinal cord injury: a systematic review. Spinal Cord 2009;47:508-18.
38. Craven BC, Robertson LA, McGillivray CF, Adachi JD. Detection and treatment of sublesional osteoporosis among patients with chronic spinal cord injury: proposed paradigms. Top Spinal Cord Inj Rehabil 2009;14:1-22.
39. Craven BC, Hawker GA, Bugaresti JM. Importance of screening of secondary causes of osteoporosis among patients with spinal cord injury. J Spinal Cord Med 2008;31:335.
40. Garland DE, Adkins RH, Kushwaha V, Stewart C. Risk factors for osteoporosis at the knee in the spinal cord injury population. J Spinal Cord Med 2004;27:202-6.

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