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CASE REPORT

Immobilisation-induced hypercalcemia following spinal cord injury affecting the kidney function in two young native Greenlanders

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INTRODUCTION: Immobilisation-induced hypercalcemia following SCI affecting the kidney function, is a rare but potentially serious condition. We report immobilisation-induced hypercalcemia affecting the kidney function in two young native Greenlanders with spinal cord injury (SCI).

CASE PRESENTATIONS: Two 15- and 24-year-old male native Greenlanders, both with traumatic C5 SCI were admitted to our spinal cord unit. They were non-smokers without history of daily alcohol intake pre- or immediately post-injury. No physical demanding activities pre-injury. Due to complaints of nausea/vomiting 10–12 weeks post-injury, not explained by usual causes such as urinary tract infection, blood samples were drawn and hypercalcaemia found. Both patients started treatment including increased hydration. Within 1 month calcium ion, plasma-creatinine and plasma-carbamide were normalised.

DISCUSSION: Over the last 20 years our spinal cord unit has only experienced immobilisation-induced hypercalcemia following SCI affecting the kidney function in two young male native Greenlanders. This finding of immobilisation-induced hypercalcemia following SCI affecting the kidney function in two young native Greenlanders, but not in the rest of our primary native Danish patient population, also including youngsters, suggests that ethnicity may be a pre-disposing factor.

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INTRODUCTION

Following spinal cord injury (SCI) the patients are usually immobilised due to SCI-induced motor paralysis, as well as treatment of other severe injuries, that is, head, thoracic or abdominal injuries, extremity fractures and so on.

Patients may develop severe hypercalcemia,[1–5] which in turn may affect the function of the kidneys. Risk factors for this development have been reported to include complete and high-cervical SCI, dehydration, prolonged immobilisation and age < 21 years.[1] The incidence is uncertain but Tori and Hill[3] reported 24% of the children and young adults with SCI to develop hypercalcemia.

We report immobilisation-induced hypercalcemia following SCI affecting the kidney function in two young native Greenlanders.

CASE PRESENTATION

Two young (15 and 24 years) tetraplegic native Greenlandic males with SCI due to trauma-developed hypercalcemia. They were both surgically stabilised and admitted to our clinic for intensive rehabilitation.

They were non-smokers and without any history of daily alcohol intake pre- or immediately post-injury. Pre-injury the younger one went to school and participated in leisure sports a couple of hours a week, while the older one worked as an engine mechanic and did not participate in any physical leisure activities on a regular basis.

Due to complaints of nausea/vomiting 10–12 weeks post-injury, not explained by usual causes such as urinary tract infection, blood samples were drawn and analysed, as illustrated in Table 1.

Both patients were shortly admitted to the nephrological department and started treatment including increased hydration. The younger one received 4.000 I.U. erythropoeitin for 3 months and cyclic treatment with etidronate (200 mg per day, 2 weeks/13 weeks) for 7 months. The older one was treated with clodronate 1600 mg per day for 4 months.

Within 1 month of treatment, calcium ion, plasma-creatinine and plasma-carbamide were normalised, and only a slight anaemia remained.

Following rehabilitation, the older one was still totally dependent of electric wheelchair, whereas the younger one regained ability to walk.

DISCUSSION

Over the past 20 years, more than 600 individuals, including youngsters mainly native Danes, have been admitted to our centre. Immobilisation-induced hypercalcaemia affecting kidney function following SCI was diagnosed and successfully treated in the two young native Greenlanders reported here.

The neurological lesion and the extreme reduction in mechanical stress on bone decreases osteoblast-mediated bone formation and initiates an accelerated osteoclast-mediated bone resorption. During this period of uncoupling of bone formation and resorption, a minority of patients develop severe hypercalcemia,[1] which in turn may affect the function of the kidneys. Immobilisation-induced hypercalcemia has been described in a variety of conditions, inducing complete or incomplete immobilisation of one or more limbs, in both genders, in children and in adults.[1–5] This suggests many risk factors.

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Table 1. Basic characteristics for two native Greenlanders with traumatic SCIs, experiencing hypercalcemia with decreased kidney function

	Case 1	*Case 2*
Age	15 years	24 years
Level of injury	C5	C5
Motor score[a] (right/left side)	At admission 18/24	At admission 10/10
	At discharge 39/43	At discharge 14/14
Maximum calcium ion (Ref. 1.15–1.35 mmol l^{-1})	1.69	1.74
Maximum plasma-carbamide (Ref. 2.5–7.5 mmol l^{-1})	21.5	8.8
Maximum plasma-creatinine (Ref. 0.040–0.090 mmol l^{-1})	0.302	0.168
Minimum hemoglobuline (Ref. 8.0–11.0 mmol l^{-1})	5.1	6.4
Minimum plasma-PTH (Ref. 1.1–6.9 pmol l^{-1})	0.7	0.9
Cr-EDTA glomerulo filtration rate (Ref. 80–130 ml/(min × 1.73 m^2))	Minimum 26	NO DATA
	At discharge 84	

Abbreviations: Ref., reference interval for normal values; SCI, spinal cord injury.
[a]According to International Standards for Neurological Classification of Spinal Cord Injury.[8]

In line with our findings, there seem to be some agreement in the literature on young age, high levels of injury and low-motor score being risk indicators for immobilisation-induced hypercalcemia after SCI.[1–4] Young age is the most frequently mentioned risk indicator. This is in agreement with our finding that the youngest patient, even though he had the highest motor score and the shortest period of immobilisation was the one most affected by hypercalcemia.

Male gender seems also a risk indicator,[4] which may be due to their higher bone mass, although not all studies have been able to confirm this finding. This may partly be explained by the distribution of male/female patients, with more males among traumatic SCI.[3]

Our finding of immobilisation-induced hypercalcemia cannot be explained by unusual pre-injury exercise habits, as none of our individuals participated in any elite training or worked physically hard. Post injury they both received the same amount of physiotherapy/physical exercise as similar SCI patients.

Frantz *et al.* emphasise that moderately restricted fluid intake due to intermittent catheterisation may further increase the probability for developing immobilisation-induced hypercalcemia in SCI patients.[5] This cannot have attributed to our finding, as neither of our patients was on fluid restriction.

We have no firm data on eating habits and are therefore unable to describe any differences in pre-/post-injury vitamin D intake. Inuit diet consists mainly of meat from fish, seal and whale of importance for vitamin D intake.[6] Had they lived as Inuit, their SCI and subsequent hospitalisation would have altered their diet and thereby probably have led to a decrease in vitamin D.

However, as none of the patients lived or ate as Inuit, a slight change in pre-/post-injury food intake is probably of minor importance.

Furthermore, vitamin D metabolites were slightly low or normal and PTH low, which is in accordance with findings in hypercalcemia following immobilisation. This is also in accordance with a population-based survey in an arctic society studying vitamin D and the positive influence of Inuit diet. An effect which was modified by ethnicity.[6] Furthermore, Andersen *et al.*[7] have demonstrated the importance of ethnicity in a study on creatinine excretion among Inuits and Caucasians.

The fact that we only have experienced immobilisation hypercalcemia affecting the kidneys in two young native Greenlanders and not in the many more native Danes received in our clinic suggests that a genetic factor due to ethnicity may be a pre-disposing factor.

Further research is needed and should include genetics describing activity in genes involved in calcium metabolism in SCI of different ethnic groups.

Our finding of immobilisation-induced hypercalcemia following SCI affecting the kidney function in two young native Greenlanders suggest that ethnicity may be a pre-disposing factor together with young age and male gender.

COMPETING INTERESTS

The authors declare no conflict of interest.

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