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Clinical Study

Supplementation with alkaline minerals reduces symptoms in patients with chronic low back pain

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Abstract

The cause of low back pain is heterogeneous, it has been hypothesised that a latent chronic acidosis might contribute to these symptoms. It was tested whether a supplementation with alkaline minerals would influence symptoms in patients with low back pain symptoms. In an open prospective study 82 patients with chronic low back pain received daily 30 g of a lactose based alkaline multimineral supplement (Basica®) over a period of 4 weeks in addition to their usual medication. Pain symptoms were quantified with the "Arhus low back pain rating scale" (ARS). Mean ARS dropped highly significant by 49% from 41 to 21 points after 4 weeks supplemention. In 76 out of 82 patients a reduction in ARS was achieved by the supplementation. Total blood buffering capacity was significantly increased from 77.69 ± 6.79 to 80.16 ± 5.24 mmol/L (mean ± SEM, $n = 82$, $p < 0.001$) and also blood pH rose from 7.456 ± 0.007 to 7.470 ± 0.007 (mean ± SEM, $n = 75$, $p < 0.05$). Only intracellular magnesium increased by 11% while other intracellular minerals were not significantly changed in sublingual tissue as measured with the EXA®-test. Plasma concentrations of potassium, calcium, iron, copper, and zinc were within the normal range and not significantly influenced by the supplementation. Plasma magnesium was slightly reduced after the supplemention (–3%, $p < 0.05$). The results show that a disturbed acid-base balance may contribute to the symptoms of low back pain. The simple and safe addition of an alkaline multimineral preparate was able to reduce the pain symptoms in these patients with chronic low back pain.

Key words: low-back-pain, acidosis, alkaline-minerals, supplement, magnesium

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Introduction

A high percentage of the population in industrialized countries experiences low back pain during their life (1). Often, these problems vanish without intense treatment within a few weeks. In many individuals, however, low back pain becomes a chronic situation, leading to a tremendous amount of costs in public health systems. It has been shown that at 12 weeks after beginning of symptoms 35% of patients still have back pain. After 1 year of treatment, 10% of the low back pain population as seen by general practitioners continue to suffer from low back pain (2).

In less than 15% of patients with low back pain an involvement of the spine can be diagnosed. Most of these patients come to the physician with diffuse, unspecific pain symptoms, in which underlying causes cannot be explained simply. These patients often are characterized by having soft tissue rheumathoid disease, fibromyalgia or myogelosis. Nonsteroidal anti-inflammatory drugs and muscle relaxants are effective in the therapy of acute low back pain (3) but for the long term treatment of chronic symptoms other procedures such as back schools or exercise therapy are use-

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ful. However, these procedures produce mainly short-term effects (4).

One explanation for low back pain might be a swelling of connective tissue due to a changed acid-base homeostasis. There is increasing evidence that disturbances in acid base homeostasis might contribute to a wide variety of diseases. Generally, a normal diet has a surplus of acid which must be excreted by the kidney (5). It has been shown that with increasing age the capacity of the kidneys to excrete acid decreases leading to a slight increase in H^+ concentration in blood (6). Chronic acid load due to a high intake of acid producing food in addition to a reduced capacity of the kidney to excrete acids leads to calcium loss from bones and a secondary wasting of muscular nitrogen (7, 8).

A portion of the acid surplus can be bound to extracellular matrix substances such as glucosaminoglycanes, inducing a changed water binding capacity with changed swelling of connective tissue. In addition, it has been shown that a dietary acid load also increases intracellular acidity (9) with possible influences on a great variety of cellular processes.

Therefore, it is reasonable to postulate that a chronically disturbed acid-base metabolism might contribute to the symptoms of patients with low back pain.

An increased adminstration of alkaline minerals might reduce the overacidity in these patients resulting in diminished pain problems.

To test this hypothesis, patients with chronic low back pain were treated with a lactose based alkaline mineral and trace element supplement (Basica®) in an open study. Basica® (being on the German market since 1930) is a traditionally used pharmaceutical preparation which is specifically used for harmonization of chronic acid-base imbalances.

Measurements were made to see if alkaline supplementation significantly reduced pain scores, changed acid-base homeostasis, or altered mineral and trace element metabolism.

Methods

In an open prospective study 82 patients (52 females, aged 20–75 years, mean age 50 years, and 30 males, aged 28–70 years, mean age 46 years) with chronic low back pain received daily 30 g of a lactose based alkaline multimineral supplement (Basica®) over a period of 4 weeks in addition to their usual medication. The daily dose of Basica® provides a total of 43 meq of alkaline salts. Basica® was given by adding it to water, juice, tea or foods like muesli or yoghurt. The mineral composition of Basica® is as follows (mg/daily dose): Ca (405), K (219), Na (375), Mg (20.4) all as citrates and traces of Fe, Sr, Mn, Cu, V, Co, Ni, Rb, Cr, Ti, Te, Bi, Sn, W, Mo as lactates.

Only patients having low back pain without radicular symptoms for more than 3 months were included. Those selected for the study had to meet the criteria of:

1. Exhibiting pain with at least two of five different conditions (pain while sitting, standing, lying, walking, or at night)
2. Having a pain score of at least 6 of a visualized pain scale ranging from 0 (no pain) to 10 (unendurable pain)
3. Patients with known lactose intolerance were excluded.

The patients were advised to keep their usual diet during the study and received no additional treatment (such as massages, exercise therapy etc.). The use of analgesics was allowed as needed and was documented. All patients gave informed consent to participate in the study.

At the beginning and at the end of the study the well known "Arhus low back pain rating scale" (ARS) was applied to the patients (10). This consists of different questionnaires concerning back and leg pain, use of analgesics, disability, and physical impairment. In scoring, the maximum of 120 points represent total invalidity due to low back pain whereas 0 points represent a status fully devoid of low back pain problems.

A non-invasive method was used to measure intracellular mineral content in sublingual cells (11). Sublingual epithelial cells were chosen because they are noncornified, are aerobic, have a turnover time of >3 days, and are easily accessible. Sublingual specimens were obtained by gently scraping the tissue between the frenulum and Wharton's duct. The cells were applied to a low-background carbon slide and dehydrated with a standard cytology fixative (2.5% carbowax, 95% ethanol). These smears yielded many homogenous, well-defined cells for analysis. X-ray microanalysis was used to test for intracellular sodium, potassium, calcium, magnesium, phophorus, and chloride. The cells were examined on a specially configured scanning electron microscope to irridate cells with a focused electron beam; measurement of x-ray fluorescence allows quantification of intracellular elements (EXA®-test, IntraCellular Diagnostics, Inc., Foster City, USA). The coefficient of variance of 40 consecutive determinations of x-rax emission energies for magnesium with a reference standard was 0.82% (11).

Additionally, a venous blood sample was taken with a heparinized syringe. Part of this whole blood sample was used for determination of blood pH and total blood buffering capacity according to the method of "Jörgensen" (12), which can be performed under conditions of a general practice.

Another part of the blood sample was immediately centrifuged for separation of plasma. Plasma was withdrawn and frozen at –20 °C. For detection of potassium, calcium, magnesium, iron, copper, and zinc concentrations plasma aliquots were lyophilized, ashed in a plasma processor (Technics, Munich, Germany) diluted with 1% HNO_3 and measured by sequential inductively coupled plasma emissions spectroscopy (Jobin Yvon ICP-Spektrometer JY 138 Ultrace). Vanadium was used as internal standard. After every 10^{th} sample a standard reference serum (Seronorm, Merck, Darmstadt, Germany) was measured for quality control. The coefficients of variance for 10 samples prepared from reference serum were 5, 7, 6, 6, 9, and 9%, for potassium, calcium, magnesium, iron, copper, and zinc, respectively.

The intake of Basica® ended two days before the last examination to exclude acute effects of Basica® on the acidity parameters in blood and tissue.

The results were statistically evaluated with the paired Student's t-test.

Results

The supplementation with Basica® was well tolerated. No adverse reactions were reported. Mean ARS dropped highly significant by 49% from 41 to 21 points after 4 weeks supplemention (Fig. 1). In 76 out of 82 patients a clear cut reduction in ARS was achieved by the supplementation (Fig. 2). There was a significant reduction in all three parts of the ARS, namely pain index (–53%), disability (–49%) and physical impairment (–29%) (Fig. 3).

Total blood buffering capacity was significantly increased from 77.69 ± 6.79 to 80.16 ± 5.24 mmol/L (mean ± SEM, n = 82, $p < 0.001$). Blood pH rose to a small significant amount from 7.456 ± 0.007 to 7.470 ± 0.007 (mean ± SEM, n = 75, $p < 0.05$) even two days after the last intake of Basica®.

Only intracellular magnesium increased by 11% while other intracellular minerals were not significantly changed in sublingual tissue as measured with the EXA®-test (Tab. 1).

Plasma concentrations of potassium, calcium, iron, copper, and zinc were within the normal range and not significantly influenced by the supplementation (Tab. 2). Plasma magnesium was slightly reduced after the supplemention (–3%, $p < 0.05$).

Table 1. Intracellular mineral content in sublingual tissue as measured by the EXA®-test. Mean ± SEM; ***, $p < 0.001$, significant difference between begin and end of the supplementation period according to paired Student's t-test.

[meq/L cells]	before supplementation	after
K	140.2 ± 56.0	140.5 ± 52.0
Mg	31.21 ± 2.94	34.82 ± 3.16***
Na	4.82 ± 1.40	4.83 ± 1.04
Ca	4.83 ± 1.29	5.08 ± 1.56
Cl	4.95 ± 1.60	5.02 ± 1.38
P	16.33 ± 2.28	17.00 ± 2.59

Table 2. Plasma concentrations of calcium, magnesium, potassium, iron, copper, and zinc before and after supplementation. Mean ± SEM; *, $p < 0.05$, significant difference between begin and end of the supplementation period according to paired Student's t-test.

		before supplementation	after
Plasma-Ca	[mmol/L]	2.48 ± 0.29	2.44 ± 0.25
Plasma-Mg	[mmol/L]	0.89 ± 0.09	0.86 ± 0.08*
Plasma-K	[mmol/L]	4.64 ± 0.60	4.49 ± 0.53
Plasma-Fe	[µmol/L]	17.7 ± 7.1	17.5 ± 6.8
Plasma-Cu	[µmol/L]	16.7 ± 5.0	16.4 ± 4.5
Plasma-Zn	[µmol/L]	13.4 ± 2.4	12.9 ± 2.9

Discussion

Most patients with acute low back pain recover quickly within a few weeks, recovery after more than 3 months, however, is slow and uncertain (1). The patient population in this study did not have extreme pain problems as the ARS at the beginning of the study was at 41 points

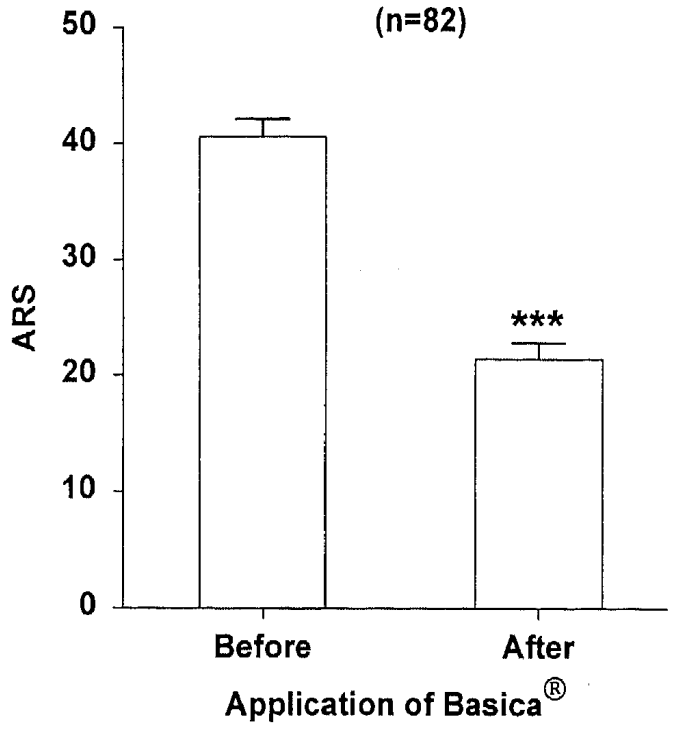

Figure 1. Arhus low back pain rating scale (ARS). Mean ± SEM; ***, $p < 0.001$, significant difference between begin and end of the supplementation period according to paired Student's t-test.

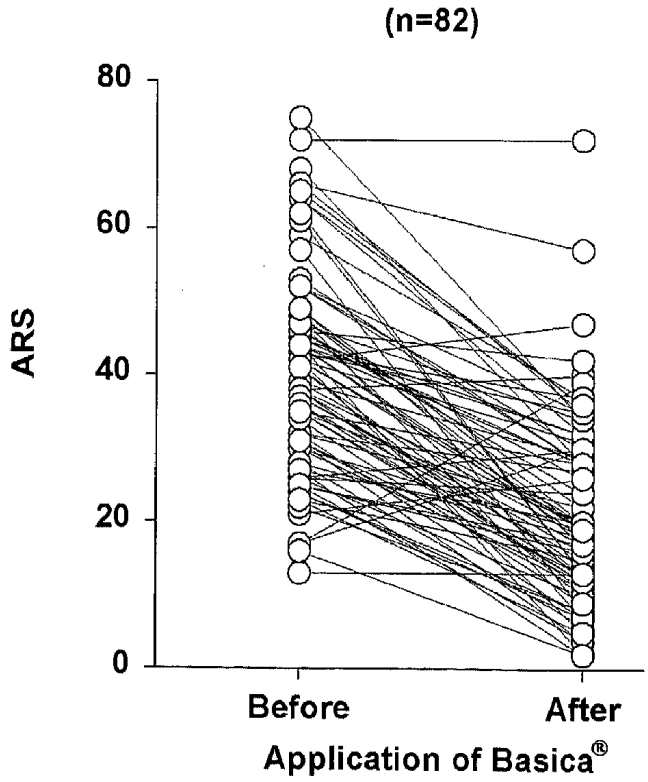

Figure 2. Arhus low back pain rating scale (ARS): Single patient values.

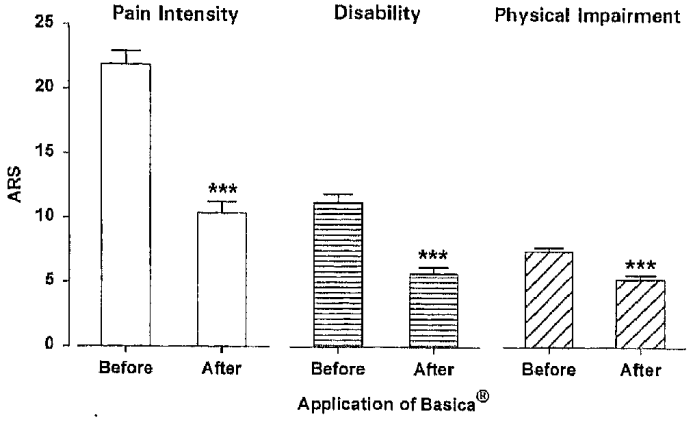

Figure 3. Different parts (pain intensity, disability, physical impairment) of the Arhus low back pain rating scale (ARS). Mean ± SEM; ***, $p < 0.001$, significant difference between begin and end of the supplementation period according to paired Student's t-test.

compared to a maximum of 120 points. A scale of 41 points still represents a situation that is characterized by usual use of analgesics and difficulty in performing simple daily tasks.

The effect of the addition of the alkaline mineral supplement to the usual therapy in chronified patients resulted in a highly significant and – more important – clinically relevant reduction in pain symptoms. Overall, more than 90% of all patients reported an improvement. Especially the pain index within the ARS (consisting of pain score and use of analgesics) was reduced. In contrast, the index of physical impairment was reduced to a less degree, indicating that a portion of the chronic changes cannot be reversed or are not due to disturbed acid-base homeostasis.

The efficiency of the supplementation on acid-base homeostasis is shown by the increased blood buffering capacity and also by the increased blood pH. Blood represents an open system, where changes in acidity are very fast and efficiently buffered, thereby avoiding possibly deleterious changes in pH. The measurement of blood acid-base parameters, however, only shows a picture of the acute situation, and it does not allow an estimate of how much acid is channeled through the system within a certain time. Discrete changes in blood pH or buffering capacity, still within the normal range, are indicators of an improved ability to cope with the daily surplus of acid.

Currently, there is no method to measure the total acid binding capacity of the connective tissue or the intracellular space within patients. Such studies are restricted to balance studies in animal experiments. In rats which were fed an acidic diet a net accumulation of intracellular acid could be detected with only minor changes in blood acid-base parameters (9).

An increased intracellular acid content in patients with chronic low back pain can indirectly be derived from the fact, that the mineral supplement led to a significant increase in intracellular magnesium concentration as measured with the EXA®-test. It has been shown intensively, that the measurement of sublingual tissue mineral content is representative for intracellular mineral concentrations also of other tissues (11, 13). The mean intracellular magnesium content in the patients before supplementation was below the reference range for a healthy population of 33.9–41.9 meq/L (14), even though the plasma magnesium concentration was in the normal range of 0.75–1.00 mmol/L (15). After supplementation most patients achieved intracellular magnesium concentration within the reference range. This significant increase in intracellular magnesium concentration cannot be attributed to the magnesium content of the supplement as the used dose provides only about 20 mg magnesium/day which is much too low to represent a magnesium therapy. It is, therefore, more plausible to explain the intracellular magnesium increase by an effect on intracellular acid content.

A small but chronic increase of intracellular H^+ concentration will release a part of the intracellularly bound magnesium (usually 95% of total intracellular magnesium is bound to ligands like DNA, RNA or proteins) leading to an increased free magnesium concentration. Since the free magnesium concentration is tightly regulated, increased intracellular free magnesium concentration is normalized by magnesium efflux, leading to a reduced total intracellular magnesium content. If, on the other hand, the intracellular H^+ is reduced more magnesium is bound to ligands, the intracellular free magnesium concentration falls and is filled up by magnesium influx leading to increased total magnesium content (16). With this mechanism, the effect of the alkaline supplement on intracellular magnesium content can simply be explained by reducing the intracellular trapping of acid. An additional hint for this explanation can be derived from the plasma magnesium concentration. Before supplementation plasma magnesium concentration was within the normal range and after supplementation was even slightly reduced, possibly indicating an intracellular uptake of magnesium.

There is no indication that an overall disturbed mineral metabolism is involved in the symptoms of low back pain, as (with the exception of magnesium) all measured minerals and trace elements were within the normal range and showed no significant alteration after supplementation.

The overall usual diet in industrialized countries provides a daily excess of about 50 meq H^+ which must be excreted by the kidneys (7). The main acid-load derives from a high protein intake, but also phosphate-rich soft drinks contribute to a high dietary acidity. By adding a supplement which provides 43 meq of base the need to compensate for the acid load by various mechanisms can be reduced. It remains to be investigated whether an abnormally high acid intake or an impaired acid excretion capacity leads to the imbalances in acid-base metabolism in patients with low back pain.

In conclusion, the results of this open study show that a disturbed acid-base balance may contribute to the symptoms of low back pain. The simple and safe addition of an alkaline multimineral preparate was able to reduce the pain symptoms in these patients with chronic low back pain significantly.

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