Calcium Supplementation: Cardiovascular Cure or Curse?

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ABSTRACT

Calcium supplements are taken by more than half of middle aged or older US women. Although better bone health is the purported reason, data supporting this association is only marginal. Several epidemiological studies have observed cardiovascular event protection with a high intake of milk and/or dairy products - main sources of dietary calcium. Recent clinical studies however suggest a detrimental effect on the cardiovascular system with their use. This article looks at evidence based data on the use of calcium supplements and their relationship to cardiovascular events.

1. INTRODUCTION

Calcium is a commonly used over the counter supplement. It is predominantly used to help prevent osteoporosis and fractures. (Tang et al., 2007) Calcium supplements reduce bone turnover and slow the rate of bone loss. However, the beneficial data in humans is statistically marginal. Only a few studies have demonstrated a reduced fracture incidence with calcium supplementation, and meta-analyses studies show only a borderline 10% decrease in fractures. (Reid et al., 2006, Reid et al., 2008) There is also a perception that supplemental calcium helps prevent some cancers. (Chung et al., 2011; Bolland et al., 2011) Epidemiological studies have also suggested that there is an inverse correlation of dietary calcium intake on hypertension and stroke. (Bucher et al., 1996; Umesawa et al., 2008) However, a plethora of emerging data indicates that high calcium intake is associated with increased cardiovascular events. (Michaelsson et al., 2013)

2. DISCUSSION

Cardiovascular disease is the number one killer in the world. (WHO, 2013) Recent evidence has suggested that a calcium-rich diet could have beneficial effects on many cardiovascular risk factors, such as obesity, (Torres et al., 2011) insulin resistance, (Fumeron et al., 2011) dyslipidemia, (Ditscheid et al., 2005) hypertension, (Ruidavets et al., 2006) inflammatory stress (Zemel et al., 2010) and cardiovascular events. (Elwood et al., 2010) However several well controlled studies reveal contradictory findings.
2.1. Calcium and cardiovascular disease

Epidemiological studies warn that calcium levels at upper limits of normal appear to be associated with higher risk of cardiovascular events. (Reid et al, 2010) Supplementation may push serum calcium levels higher. A meta-analysis of several studies established an increase in myocardial infarction with calcium supplementation, without co-administered vitamin D. (Bolland et al, 2010) and with vitamin D. (Bolland et al, 2011) A well conducted meta-analysis of trials of calcium supplements, found a 27%-31% increase in risk of myocardial infarction, and a 12%-20% increase in risk of stroke. (Reid et al, 2013) These events also translate into higher mortality. (Reid et al, 2011) Higher death rates from cardiovascular disease (except stroke) have been noted with high intakes of calcium in women. (Michaelsson et al, 2013) Another study indicated that high intakes of supplemental calcium may be responsible for excess cardiovascular deaths in men. (Xiao et al, 2013) The major patho-physiologic mechanisms include detrimental effects on vascular calcification. (Rubin et al, 2007; Shin et al, 2012) Calcium raises fibroblast growth factor 23, which has been associated with higher levels of cardiovascular and all cause mortality. It also detrimentally affects vascular cells, plaque stability, platelet function and blood coagulation.

2.2. Other problems with supplemental calcium

Besides increased risk of major cardiovascular events, excessive calcium intake may also result in milk-alkali syndrome, (Patel et al, 2010) renal stones, (Heaney, 2008) prostate cancer (Butler et al, 2010) and GI symptoms such as constipation, bloating, cramping and indigestion. (Lewis et al, 2012) Calcium supplements may also interfere with the absorption of iron (Bendich, 2001) and zinc. (Wood et al, 1997) There have also been rare reports of lead contamination in calcium supplements, (Rehman et al, 2007) especially if derived from bone-meal, oyster shell and dolomite. (Whiting, 1994)

3. CONCLUSION

Calcium-containing compounds are the second most popular non-prescription supplementation among adults in the United States. The protective role of excess calcium supplements (mainly calcium carbonate or calcium citrate) in the prevention of fractures or osteoporosis remains questionable and marginal. (Warenssjo et al, 2011) Several studies have observed cardiovascular event protection with a high intake of milk and/or dairy products - main sources of dietary calcium. (Elwood et al, 2008) However, similar results have not been duplicated with oral supplementation with calcium. Emerging evidence based data linked increased calcium intake with a higher risk for cardiovascular disease. Caution therefore dictates indiscriminate ingestion of calcium supplements as their non-skeletal risks clearly outweigh any skeletal benefits. They should only be taken by those with a low calcium intake and/or in reduced amounts (Manson et al, 2013) Further studies are needed to investigate these detrimental effects of supplemental calcium use beyond the purported bone health benefits.

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15. Michaelsson K, Melhus H, Warenssjo Lemming E et al. Vitamin D supplements and health outcomes: a reanalysis of trials of calcium supplements, found a 27%-31% increase in risk of myocardial infarction, and a 12%-20% increase in risk of stroke. (Reid et al, 2013) These events also translate into higher mortality. (Reid et al, 2011) Higher death rates from cardiovascular disease (except stroke) have been noted with high intakes of calcium in women. (Michaelsson et al, 2013) Another study indicated that high intakes of supplemental calcium may be responsible for excess cardiovascular deaths in men. (Xiao et al, 2013) The major patho-physiologic mechanisms include detrimental effects on vascular calcification. (Rubin et al, 2007; Shin et al, 2012) Calcium raises fibroblast growth factor 23, which has been associated with higher levels of cardiovascular and all cause mortality. It also detrimentally affects vascular cells, plaque stability, platelet function and blood coagulation.

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