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World Cardiology Summit 2020: Why is Cardiac Morbidity and Mortality Greater Around Christmas, New Year's, Monday Mornings and in the Morning Hours: Potential Roles of Unrecognized Ionized Hypomagnesemia and Release of Ceramides?

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Introduction :

There is a developing incidence of deadly cardiac occasions round Christmas, New Year's and in the morning hours from 4:00 to 10:00 a.m. which is well-established in the USA and in The Southern Hemisphere. In addition, many cardiac deaths frequently manifest on Mondays with no exceptional rationalization. Many of these deaths are, for the most part, unexplained and listed as "death from "natural causes". Although in the USA, the deaths which appear round Christmas and New Years appear in the bloodless –winter months, this does now not account for many cardiac incidences which manifest at some point of the 12 months in the early a.m. hours or on Mondays.

Role of Magnesium in Cardiac Morbidity and Mortality

Ever on account that our laboratories first pronounced that magnesium (Mg) deficiency consequences in vasospasms of small and giant coronary arteries, and that these occasions may want to be accountable for a awesome deal of unexpected loss of life ischemic coronary heart disorder (SDIHD), a wide variety of scientific research have regarded which have established and prolonged these findings. We in the beginning speculated that low dietary Mg consumption and /or mistakes in Mg metabolism may want to be accountable for a massive wide variety of unexpected cardiac deaths (SCD) and coronary heart assaults in the Western world.

In the early 1980's, some scientific research seemed which recommended that of all electrolytes measured in the blood of hospitalized patients, whole serum magnesium (Mg) degrees frequently confirmed reduced levels, e.g., from 80-50% of everyday. However, in general, sufferers admitted to the intensive or coronary care devices regularly tested 60-30% of regular whole blood stages of Mg. When the blood/sera/plasma from these sufferers are examined for ionized Mg levels, in addition to the latter measured total Mg levels, these numbers upward push to 80-70% in the sufferers admitted to intensive and coronary care gadget.. In addition, the crimson blood cells bought from these sufferers are severely poor in ionized Mg (e.g., 60-40% of normal; Resnick, Altura, and Altura, unpublished studies). Why is it so necessary to measure ionized Mg levels, no longer solely whole blood Mg levels?

Mg is a co-factor for greater than five hundred enzyme systems, and is the 2nd most considerable intracellular cation after potassium. It is quintessential in severa physiological, mobile and biochemical features and systems, going for walks the gamut from hormone-receptor binding, transmembrane fluxes of cations and anions, mobile electricity generation, muscle

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contraction, legislation of DNA and RNA structure, law of lipid and carbohydrate metabolism, rules of plasma lipid stages (i.e., cholesterol, triglycerides, and LDL-cholesterol), law of phone and tissue growth, nerve conduction, numerous cardiac features and cardiac stability, manage of vasomotor tone and distribution of blood flows to all organ systems, and mobile dying (i.e., apoptosis and necroptosis), amongst many others. Mg is depleted in ordinary strategies of meals guidance (e.g., boiling, frying, etc.) and processing.

The day by day consumption of Mg has been declining due to the fact that 1900, from the place it used to be about 500-600 mg/day to about 150-225 mg/day, in many USA and European geographic regions, at the existing time. Mg is acknowledged to exist in three forms; free or ionized, complexed, and protein – certain. These three fractions represent the complete serum and mobile Mg. In addition, up till our studies, there had been no dependable strategies to measure ionized Mg on entire blood, serum , and plasma swiftly (within 1-2 min) in the OR and critical-coronary care gadgets

Of almost one hundred sufferers who have been admitted for emergency coronary artery omit surgical procedure (CABS), at our hospitals (e.g., University Hospital and Kings County Hospitals), 88% of them exhibited extensively diminished ranges of serum ionized Mg2+, however now not always complete serum Mg degrees. Of these who have been admitted on Holidays, such as Christmas or Thanksgiving, or in the morning hours (i.e., from 2:00 – 7:00 AM), we found the lowest serum ranges of Mg2+ (i.e., from 0.40 – 0.52 mM vs. 0.57-0.70 mM-controls, $p<0.001$). Patients admitted on Monday mornings (i.e., from 2:00 – 9:00 AM) for CABS exhibited, on common 0.48 ± 0.06 mM vs 0.67 ± 0.03 mM ($p<0.01$). For the most part, many (about 55%) of these CABS sufferers exhibited near, ordinary complete serum Mg levels.

When we mimicked these, decreased serum Mg2+, in vitro, the usage of isolated, ordinary canine, baboon, monkey, human or piglet coronary/ cerebral arteries, they went into one-of-a-kind tiers of vasospasm which may want to solely be at ease with accelerated tiers of Mg2+, no longer with calcium channel blockers or a range of commonly-used vasodilator capsule. The artificially-lowered stages of Mg2+ additionally resulted in potentiation of the contractile movements of all kinds of circulating neurohormonal vasoconstrictor retailers (e.g., catecholamines, angiotensin II, serotonin, and a range of peptides such as vasopressin, etc.).

From our studies, we trust the records are steady with the speculation that human topics admitted for emergency CABS on predominant holidays, in the morning hours , or on Monday mornings no longer solely display abnormally low Mg2+ degrees however most probably are predisposed to vasospasm of the coronary and cerebral arterial vessels which would end result in multiplied morbidity and mortality. So, it makes eminent feel that human subjects, worldwide, would be predisposed to extended morbidity and mortality on vacations such as Christmas, New Year's Day and Monday mornings.

An essential aspect worried in these predilections, at these a number instances of the year, are additionally most probable due to the extra ingesting of alcoholic beverages, espresso and sodas (with caffeine), which have been proven to unexpectedly use up vascular easy muscle, cardiac muscle and endothelial cells of intracellular degrees of Mg2+. In addition, when you consider

that many humans get coronary heart – burn after heavy meals, they have a tendency to ingest protonpump inhibitors which additionally can minimize Mg ranges. One have to additionally reflect onconsideration on the opportunity that a quantity of the topics death from SDIHD and SCD may also have been on long-term therapy with cardiac glycosides and/or thiazides, positive antifungal sellers (i.e., amphotericn B), aminoglycosides (e.g., gentamicin, tobramycin), loop diuretics (e.g., furosemide), immunosuppresents(e.g., cyclosporine, sirolimus), or even sure chemotherapeutic dealers (cisplatin, amsacrine), all of which burn up the physique of Mg; plausible interactions with consuming of alcoholic liquids and/or caffeine –beverages would have a tendency to strengthen (and potentiate) the tendency for considerable, fast Mg depletion. Unfortunately, such interactions have no longer been a focal point of any epidemiological research to our knowledge.

The cellular, biochemical, and molecular mechanisms of how diminished mobile stages of Mg^{2+} purpose vasospasm and reduced peripheral, coronary, and cerebral blood flows, inflammation, ischemic activities , atherogenesis, and various types of mobile loss of life have been a long-time focal point of our laboratories which are introduced and mentioned some other place. In this context, the use of proton –nuclear magnetic spectroscopy (NMR), P31 – NMR, and brand-new ELISA assays, we have located that low tiers of extracellular Mg^{2+} ($[Mg^{2+}]0$) hastily generated ceramides and different sphingolipids which, heretofore, had been absolutely unknown as workable causal elements in SDIHD, surprising cardiac demise (SCD), congestive coronary heart failure (CHF) and coronary artery disorder (CAD) . This work led us to hypothesize that dietary deficiency and/or inborn metabolic-induced deficiency of Mg may want to end result in elevated morbidity and mortality from coronary and cerebral arterial vasospasms. But, how would technology of ceramides and/or different sphingolipids(e.g., sphingosine; sphingosine-1-phosphate) end result in susceptibility to SDIHD, SCD, CHF, and CAD?