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Do's and Don'ts in supportive CKD Care

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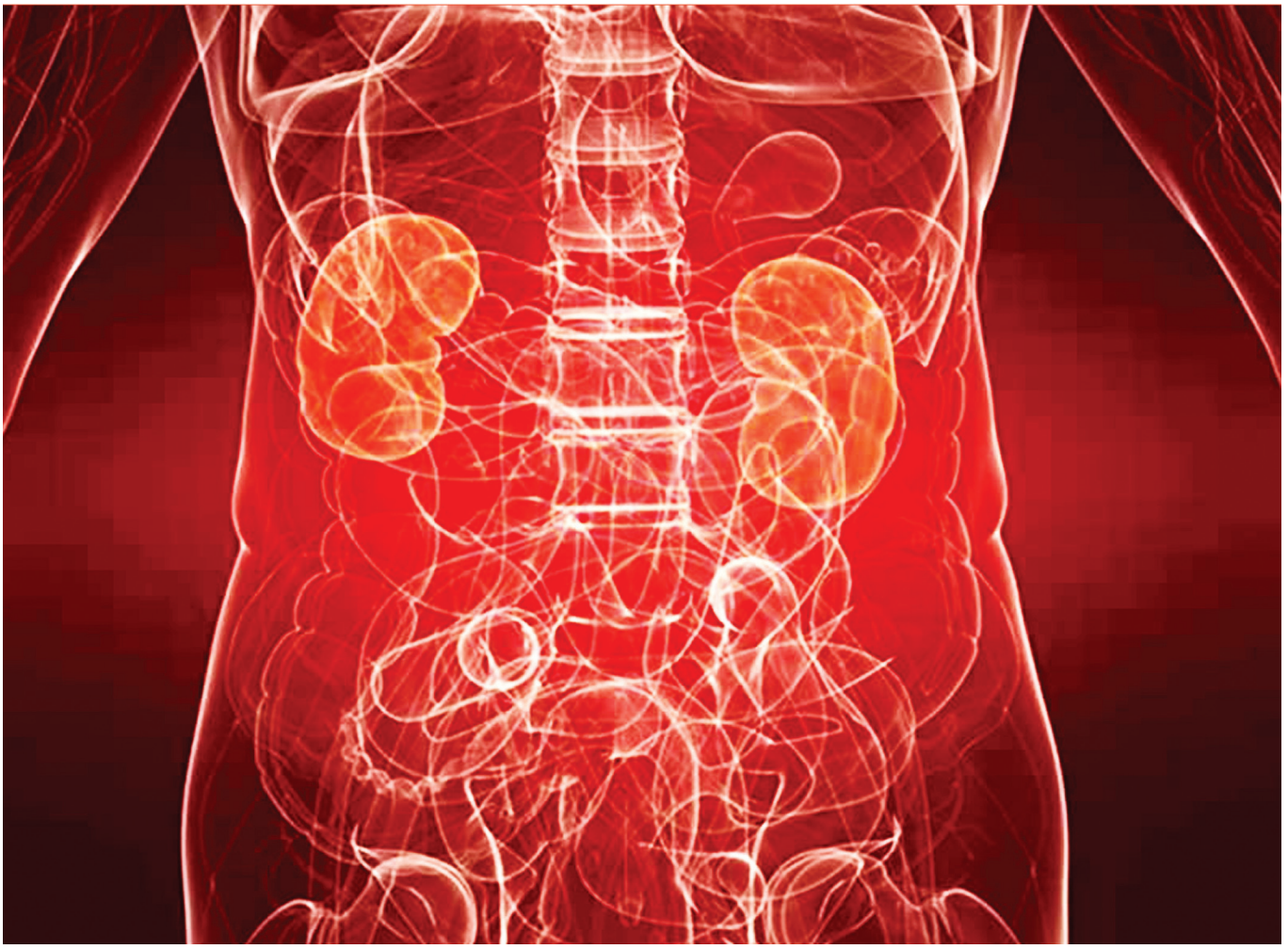

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Diabetes and Antioxidants

SANJAY AGRAWAL

Diabetes is a chronic metabolic disorder with a rapidly increasing prevalence[1] highlighting the importance of continued research and the need for novel methods to both prevent and treat this pandemic. Although obesity and physical inactivity are known to be major risk factors for type 2 diabetes (T2DM), recent evidence suggests that oxidative stress may contribute to the pathogenesis of T2DM by increasing insulin resistance or impairing insulin secretion.[2]

While diabetes management has largely focused on control of hyperglycemia, the rising burden of this disease is mainly correlated to its vascular complications. This is reflected by a 4-fold increase in the incidence of coronary artery disease, a 10-fold increase in peripheral vascular disease, and a 3- to 4-fold higher mortality rate with as much as 75% of diabetics ultimately dying from vascular disease.[3] Oxidative stress may play a role in the pathophysiology of diabetes and cardiovascular disease. Consequently, the question of whether antioxidants could have a beneficial effect on reducing the risk of these conditions, especially cardiovascular disease, has been intensively investigated, but the results remain inconclusive. If antioxidants play a protective role in the pathophysiology of diabetes and cardiovascular disease, understanding the physiological status of antioxidant concentrations among people at high risk for developing these conditions, such as people with the metabolic syndrome, is of interest.[4]

What Does Mold Have to Do With Diabetes?

Nothing. Mold has nothing to do with the direct cause of diabetes. It does, however, provide an early warning sign to diabetics who aren't properly monitoring their blood-sugar levels and those who aren't even aware that they have diabetes.

Mold, an opportunistic fungus that generally wreaks havoc on the human body, is finally—in a rare act of consequential benevolence—proving to be useful. The mere fact that mold thrives on organic material, especially organic material that consists of sugar, is precisely why it is important to observe its presence and whereabouts—not just to remove and prevent it but to gain a glimpse of our own state of health.

Many mold victims complain of mold growing in their clothing and in their toilet bowl. This mold can appear fuzzy, slimy, and gray, black, or brown in color. While this can easily occur from infrequent washing and sanitation of either the person, the clothing, or the toilet bowl, and is also a sign of an overall mold infestation throughout the home, it is a major indication of blood-sugar levels in the sweat and urine of the individual.

If the body is overwhelmed by glucose, it will attempt to flush as much of the excess out of its system by means of sweat, exhalation, and urination. It is quite plausible that the mold found in your clothing or in your toilet bowl is a sign that your body is truly suffering from an abundance of glucose and needs help. It's a commonly overlooked correlation, but it has the potential to save your life.

Diabetes & Oxidative stress

A number of complications arise as a consequence of macro and microvascular complications that result from diabetes; these deficits have a central role in the tissue-damaging effects of chronic hyperglycemia. Since endothelial cells (as well as renal mesangial and Schwann cells) are unable to limit glucose transport as well as other cells do, they are more vulnerable to the toxic effects of hyperglycemia.

Oxidative stress results from an imbalance between radical-generating and radical-scavenging systems, i.e. increased free radical production or reduced activity of antioxidant defenses or both. Hyperglycemia-induced oxidative stress has also been associated with increased endothelial cell apoptosis in vitro and in vivo. Several studies have shown that diabetes mellitus (types 1 and 2) is accompanied by increased formation of free radicals and decreased antioxidant capacity, leading to oxidative damage of cell components.

Decreased antioxidant defenses

Cells have evolved highly complex enzymatic and non-enzymatic antioxidant systems, which work synergistically, and in combination with each other, to protect the body against free radical-induced damage.

There are several lines

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of evidence to suggest that antioxidant defences may be lower in diabetes. These include reports of reduced plasma/serum total antioxidant status or free radical scavenging activity and increased plasma oxidisability in type 2 diabetics, together with reduced levels of specific antioxidants such as ascorbic acid and vitamin E. In addition, the activities of the antioxidant enzymes catalase, superoxide dismutase, and glutathione peroxidase have been described as reduced in diabetics. A diminution in the endothelial synthesis of NO has also been suggested in type 2 diabetics, which apart from detracting from vascular antioxidant defense, would of course compound any defect in the anti-atherogenic signaling role.

In patients with T2DM, the content of oxidized fatty acids is increased, and the anti-inflammatory and antioxidant activities of HDLs are impaired.[5]

ANTIOXIDANT THERAPY & DIABETES

The inhibition of intracellular free radical formation would provide a therapeutic strategy to prevent oxidative stress and the related diabetic vascular complications. Antioxidants may act at different levels, inhibiting the formation of ROS or scavenge free radicals, or increase the antioxidants defense enzyme capabilities. Supplementation with antioxidants and/or factors essential to nitric oxide (NO) production may potentially improve endothelial dysfunction in T2DM by re-coupling eNOS and mitochondrial function, as well as decreasing vascular NAD (P)H oxidase activity.[6] However, in the case of macrovascular/ microvascular complications, the antioxidant therapy is beneficial together with blood pressure control, management of dyslipidemia, and optimal glucose control.[7]

Generally, the antioxidant pharmacotherapy can be divided in the use of antioxidant enzyme and substrates, biogenic elements, combined drugs, synthetic antioxidants, and drugs with antioxidant activity. There are also a large number of natural cellular defense mechanisms as the naturally existing antioxidant components, which neutralizes free radical damage. The enzymatic antioxidant systems, such as copper, zinc, manganese superoxide dismutase, gluthatione peroxidase, gluthathione reductase, and catalase may remove the ROS directly or sequentially, preventing their excessive accumulation and consequent adverse effects. Non-enzymatic antioxidant systems consist of scavenging molecules that are endogenously produced such as glutathione, ubichinol, and uric acid or derivatives of the diet such as vitamins C and E, carotenoids, lipoic acid, selenium, etc.[8] Exercise training results in an up-regulation of antioxidant defense mechanisms in various tissues, presumably due to increased levels of oxidative stress that occurs during exercise.[9]

Well-established antioxidants derived from the diet are vitamins C, E, A, and carotenoids, which have been studied intensively. In general, exogenous antioxidants can compensate for the lower plasma antioxidant levels often observed in T2DM and in pre-diabetic individuals, whether their diabetes is primarily genetic in origin or due to obesity and a sedentary lifestyle.[10] Vitamin C (ascorbic acid) and vitamin E (tocopherol) have well-described antioxidant properties. Vegetables and fruits have in their natural composition other substances besides these antioxidant vitamins, which guarantees health benefits associated with its consumption. Over the past decade, evidence has accumulated that plant polyphenols are an important class of defense antioxidants. These compounds are widespread virtually in all plant foods, often at high levels, and include phenols, phenolic acids, and flavonoids.

In a prospective cohort study, vitamin C intake was found to be significantly lower among incident cases of T2DM. In three prospective observational studies, serum α-tocopherol levels were associated with lower risk of type T1DM or T2DM. In another prospective study cohort of more than 4000 non-diabetic subjects over 23 years, vitamin E intake was significantly associated with a reduced risk of T2DM.

However, despite observational studies suggesting an association between antioxidant vitamin intake and reduced cardiovascular risk, this has not been borne out in interventional trials. Studies of the effect of ascorbic acid and tocopherol on endothelial dysfunction in T2DM have yielded mixed results.[18]

Coenzyme Q or ubiquinone may decrease oxidative stress not only by quenching reactive oxidant species but also by 'recoupling' mitochondrial oxidative phosphorylation, thereby reducing superoxide production. Alpha-lipoic acid, a critical co-factor for mitochondrial dehydrogenase reactions, is another compound with free radical-scavenging activity. Lipoic acid was found to increase glucose transport in muscle cells in culture by stimulating translocation of GLUT4 from internal pools to the plasma membrane. In cultured adipocytes, treatment with lipoic acid protected the insulin

receptor from oxidative damage, maintaining its functional integrity. A placebo-controlled explorative study of patients with T2DM indicated that oral administration of lipoicacid significantly increased insulin-mediated glucose uptake, presumably by modulating insulin sensitivity.

In addition to the antioxidants mentioned, a number of commonly used drugs have been reported to have antioxidant activity, in addition to their primary pharmacological property. For example, gemfibrozil, a lipid-lowering fibrate, was previously reported to have antioxidant actions. Anti- hyperlipidemic statins are thought to exert antioxidant effects. In addition, it has been demonstrated that at least a part of the beneficial vascular effects of thiazolidinediones are linked with their antioxidant properties.

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The term "cardiorenal syndrome" has been attributed to a clinical condition that includes a variety of acute and chronic dysfunctions, in which the primary failing organ could be either the heart or the kidney; any primary impairment in one of the two organs promotes and perpetuates a complex combination of feedback mechanisms that further decrease the function of both the heart and the kidney. Several pathways have been proposed as channels through which a crosstalk between the kidney and the heart takes place; the main ones are hemodynamic imbalance, neurohormonal signaling, and inflammatory activation. On one side, pressure fluid overload and sodium retention, altered electrolyte levels and acidosis due to renal failure may contribute to ventricular dysfunction, accelerating cardiac remodeling and increasing the risk of arrhythmias. Conversely, myocardial dysfunction promotes the worsening of kidney function, such that a vicious circle is triggered; hypervolemia, rennin-angiotensin-aldosterone system activation, inflammatory cytokines, nitric oxide dysregulation, oxidative and mechanical stress, and increase in myocardial oxygen consumption are all factors that lead to myocyte injury and death.

-Cardiology

Acute and rapidly reversible left ventricular dysfunction may be triggered by various emotion and physical insults. The most common precipitant being the death of a close family member but the other reported precipitants include court appearance, fear of a procedure, arguments and surprise parties and reunions. This entity is now well known as stress cardiomyopathy or Takotsubo cardiomyopathy.

- JAPI

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