

Postprandial hypotension

Postprandial hypotension is commonly defined as a decrease in systolic blood pressure of 20mmHg or more observed within two hours after meal ingestion. It is very common in older patients especially in those living in long-term healthcare homes. Patients with postprandial hypotension may develop symptomatic hypotension, syncope and falls.

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One of the first case reports on postprandial hypotension was published in 1977 and described a 65-year-old man who had dizziness and visual disturbance associated with large decreases in blood pressure after almost every meal.¹ Two decades later, another study looked at 499 elderly residents in a long-term healthcare facility and found that 118 (24%) had a maximal reduction in systolic blood pressure of 20mmHg or higher after a meal.² Other studies have found that elderly patients with autonomic failure who have postprandial hypotension also frequently have orthostatic hypotension.^{3,4,5} It has also been shown that postprandial hypotension is more common than postural hypotension in elderly patients.^{6,7}

Clinical features

Postprandial hypotension may predispose older people to develop falls, syncope, dizziness and fatigue.^{3,7,8} A study in elderly residents showed that patients with a history of syncope or fall in the preceding six months had a greater drop in postprandial systolic blood pressure.²

It can also increase the risk of transient ischaemic attacks, stroke and angina^{7,8} due to impairment of perfusion in the appropriate vascular bed. The cerebral symptoms depend on the extent to which cerebral circulation is compromised. As one ages or in hypertension, cerebral blood flow decreases therefore any small changes in blood pressure following food can reduce the cerebral oxygen delivery needed to maintain consciousness.

Postprandial hypotension is more likely to occur in people who have hypertension or disorders that impair the brain centres controlling the autonomic nervous system such as Parkinson's disease, multisystem atrophy and diabetes.⁵

Although syncope in elderly patients has been associated with postprandial hypotension, this association is rarely considered in the evaluation of syncope.^{2,3}

Pathophysiology

Postprandial hypotension can be partially explained by an inadequate sympathetic response to meal-induced splanchnic vasodilation. This hypothesis has been corroborated by an inadequate or

blunted response of the heart rate, plasma norepinephrine levels, and microneurographic recordings of muscle sympathetic nerve activity to meal ingestion in patients with postprandial hypotension.^{3,9,10,11,12}

After eating a meal, there is splanchnic blood pooling because of splanchnic dilatation as a large amount of blood is needed for digestion. This leads to a reduction in venous return, stroke volume and cardiac output. This should lead to baroreflex compensation, causing a reflex-mediated increase in the heart rate and total systemic vascular resistance, hence maintaining the blood pressure.

In a patient with autonomic nervous dysfunction or in elderly people with impaired sympathetic reflex activity due to a reduction in baroreflex sensitivity, the reflex-mediated increase in heart rate and total systemic vascular resistance do not occur; hence there is a reduction of postprandial blood pressure.

There is a reduction in baroreflex sensitivity with age and in hypertension, hence postprandial hypotension is more common.

The effects of carbohydrates on blood pressure are primarily caused by glucose, which stimulates insulin release. Other carbohydrates, such

as fructose or xylose, have no or minimal effect on blood pressure and do not stimulate insulin.⁴ Insulin has therefore been implicated in the pathogenesis of postprandial hypotension. In diabetic patients with autonomic neuropathy, systolic and diastolic blood pressure decrease considerably after insulin is administered, sometimes resulting in syncope.⁵ It seems that the vasodilatory effect of insulin might be responsible for postprandial hypotension.

Vasoactive intestinal polypeptides have been studied because of their well-known vasodilating effect and their secretion is increased following ingestion of a meal. These may therefore play a role in the pathogenesis of postprandial hypotension.

Diagnosis

Blood pressure should be checked before a meal in the lying position after five minutes of rest. It should then be checked at 15, 30, 60, 75, 90 and 120 minutes after eating while the patient remains seated.

Oral glucose solutions seem attractive for the meal to be used in the diagnosis, but standardised mixed meals with a variety of nutrient compositions are recommended.

In 70% of patients with postprandial hypotension, the blood pressure drop is seen 30–60 minutes after eating.

Treatment

People who have symptoms of postprandial hypotension should not take antihypertensive drugs before meals. It has been shown that drugs such as calcium channel blockers, digoxin, nitrates, angiotensin-converting enzyme inhibitors and diuretics are associated with a significant increase in falls in postprandial systolic blood pressure.² Taking a smaller dose of the antihypertensive drugs may help reduce the effects of this disorder.

Carbohydrate meals predispose susceptible patients to develop postprandial hypotension; therefore patients should

eat fewer carbohydrates hence decreasing the rise in glucose level and the release of insulin. Meal size should be adjusted and frequent small meals should be encouraged. Alcoholic beverages should be avoided before and after meals.

For some people, walking after a meal helps improve blood flow, but blood pressure may fall when they stop walking.

Taking some drugs may help. For example, NSAIDs cause salt to be retained and thus increase blood volume. Caffeine helps maintain blood pressure by causing blood vessels to constrict but it is better taken before breakfast so that sleep is not affected.

Somatostatin or its analogue may help prevent postprandial hypotension by reducing the splanchnic blood flow by causing a direct action on the vasoactive smooth muscle or by redistributing blood flow to the central circulation from the splanchnic vessels.

It has recently been established that the rate of nutrient delivery from the stomach into the small intestine helps determine the hypotensive response to carbohydrate, therefore the fall in blood pressure is greater when gastric emptying is more rapid.¹³ Agents such as guar and acarbose slow gastric emptying and reduce glucose absorption. Guar is guar gum, which is a dietary food extracted from guar bean. It is a polysaccharide that forms a viscous gel with water. It is used as a thickening agent and alters viscosity and hence slows gastrointestinal transit. Acarbose is an alpha-glucosidase inhibitor, which inhibits the enzymes needed to digest carbohydrate and hence reduces postprandial glycaemia, small intestinal glucose absorption and hence insulin release.

In a recent study of 13 patients with postprandial hypotension, patients were given acarbose or placebo 20 minutes before a standardised meal. In the setting of autonomic failure, after adjusting for potential confounders, acarbose significantly attenuated the postprandial fall in systolic and diastolic blood pressures by 17mmHg. Furthermore, acarbose effectively reduced plasma levels of insulin, a known vasodilator, but after adjusting for insulin levels, the attenuation of postprandial hypotension by acarbose remained significant, indicating that additional mechanisms contribute to this effect.¹⁴

Conclusion

Most healthy elderly people have postprandial decreases in blood pressure. These changes in blood pressure tend to be small and asymptomatic. A decrease in systolic blood pressure of 20mmHg or more should be considered potentially dangerous and efforts should be made to decrease the risk of developing symptoms such as postprandial syncope, dizziness and falls.

All physicians looking after elderly patients should be aware of the hypotensive effect of food intake and its important clinical association with syncope, falls, dizziness and stroke.

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