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Review

Postprandial Hypotension in Elderly

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Abstract

A substansial declining of blood pressure after a meal was first reported in 1977, in 65-year-old male who was diagnosed with Parkinson Disease. He experienced visual disturbance than dizziness following an oral glucose load, which his systolic blood pressure dropped from 200 to 105 mmHg. Not long after that, a prospective study showed that postprandial hypotension (PPH) was common among geriatric patients who stayed in nursing home (LIpsitz et al, 1983). Postprandial hypotension has been defined as a fall in systolic blood pressure > 20 mmHg, or a decrease to ≤ 90 mmHg when the preprandial blood pressure is ≥ 100 mmHg, within 2 hours of a meal. This suddenly decrease in blood pressure is related with a number of symptoms, including dizziness, syncope, visual disturbance, falls, angina, stroke and eventually death (Fisher et al., 2005). All of these manifestations can result in more complications and overuse of heath-care with increasing cost burden (Lipsitz et al., 1986). Postprandial hypotension is a common phenomenon in geriatric population, but are frequently missed and undetected because the majority of the patients don't have any particular signs and symptoms (Barochiner, et al 2014). So far, there is still lack of concern about this topic. This topic not quite much being written in textbooks and there are only small trials and not much studiesthat we can cite about. Therefore, our primary aim of this review are to evaluate current knowledge relating to PPH and then make us as clinicians become more aware in detecting PPH especially in geriatric population, who are increasing in the future. So, we can diagnose earlier and treat them optimally. In this review, we will focus on epidemiology, pathophysiology, clinical manisfestations, diagnosis and management, both nonpharmacologic and pharmacologic.

Keywords: Postprandial hypotension, systolic, diastolic, elderly

INTRODUCTION

Epidemiology

There is still no epidemiology data about PPH in Indonesia. Five prevalance studies in elderly nursing residents had been done, also in hospitalized geriatric patients in 4 studies and one study evaluated prevalence in geriatric patients who treated as outpatient (Le-Couteur, et al 2003). Those studies showed that the prevalence of PPH is about 24-38% in healthy elderly residents and about 20-91% in hospitalized geriatric patients. This fall of blood pressure after meal is more frequently happened in elderly who stay in nursing home than elderly who islive in community or with their family (Son, et al 2012).

From 2 studies in type-2 diabetes mellitus (DM) population, the prevalence of PPH is ± 40%,3 studies in hypertension population is 27-73% and the prevalence of PPH in 2 studies in Parkison Disease is 40-100% (Van-Orshon et al., 2010, Puisieux, et al 2000). Three prevalence studies showed propensity to particular races such as Caucasians Asians and mixed race populat-ions, hence it is unknown whether the races influence the

prevalence of PPH (Lubart, et al 2006).

A study from one of the hospital in Netherland in 2005 showed that there is 67% geriatric patients experienced PPH in total 85 geriatric patients, with the average of dropped blood pressure after meal is 43 ± 4 mmHg. In addition, the study also reported that incidence of PPH is higher than orthostatic hypotension (52%) (Vioet et al., 2005).

Orshoven et al. reported prevalence of PPH is 45% in geriatric patients and 15% in healthy elderly. About 90% of the subjects experienced falling in blood pressure 1 hour after meal especially after breakfast.Another study in Mexico showed that the prevalence rate is 80,1% in elderly both who stay in nursing home or who live in community with their family. The more extreme postprandial hypotension (systolic blood pressure < 100 mmHg or diastolic < 60 mmHg or decrease systolic blood pressure > 40 mmHg or diastolic > 20 mmHg and with symptoms) is occurred in 26.2% of elderly. This study also demonstrated that elderly who live in nursing home (87,3%) are more often experienced PPH than elderly who live with their family (69,8%) (Lagro et al., 2012).

Pathophysiology of PPH

Postprandial hypotension can occur in both elderly population and younger population. Postprandial hypotension in younger population usually is related with several comorbidities such as DM, hypertension, Parkinson disease, regularly hemodyalisis patients, multiple system atrophy (MSA), Fragile X mutation and Shy-Drager syndrome. Whereas, in elderly population, PPH can occur in healthy one, this is associated with aging process (Zanasi et al., 2012). In aging process, there is a declinein autonomic nervous system function, both sympathetic and parasympathetic, which will affect the ability to adapt or respon to both visceral stimulus and changing environment in elderly. In addition, elderly also have a compromised cardiovascular changes related to blood pressure regulation, e.g. decrease in pacemaker cells in sinoatrial node and blunted inotropic-chronotropic-lusitropic responses to β-adrenergic stimulation (Ejaz et al., 2006).

Pathophysiology of PPH is not yet fully understood, however, in the broadest prespectives, a significant postprandial decrease in blood pressure is a sign of inadequate compensation of cardiovascular system in response of increasing splanchnic blood pooling due to meal digestion. In young healthy individual, digestion do not have a significant effect in blood pressure, in contrast, healthy elderly can experience a substantial changes in blood pressure. Currently, scientificevidencessuggest that there are not a single etiopathogenesis or dominant factor in most PPH cases, instead they are multifactorial. Those factors are autonomic-neural dysfunction, changes in gastrointestinal hormonal, meal composition, gastric distention and the rate of delivery of nutrients to small intestine (Luciano et al., 2010).

Lack of sympathetic response, in elderly with PPH, causes the compensation of increasing splanchnic blood pooling is inadequate. Amicroneurographic study of muscle sympathetic activity showed that compared with younger subjects, elders respond to an oral glucose load with smaller increase in sympathetic activity. This sympathetic response is mediated by stretch receptor in the stomach, so called gastrovascular reflex. This hypothesis is supported by the observation study which demonstrated that there is no difference in muscle sympathetic activity between the healthy elderly and young patients in response to an intraduodenal glucose load and also the decrease in blood pressure is milder after glucose oral intake compared to intraduodenal glucose load with the same amount glucose load (Parashar et al., 2016). The magnitude of the sympathetic response, which is induced by gastric distention, is dependent on the distension volume. In healthy elderly, gastric distention with water, even at relatively low volume (300 ml), attenuates the decrease in blood pressure induced by intraduodenal glucose. Although data showed that gastrovascular reflex is weakened in healthy elderly include their sympathetic parameter activities, gastric distention with water associated with a significant pressor response in autonomic failure patients (Lagro et al., 2012).

Moreover, spectral analysis of the heart rate response also confirmed that geriatric patients with PPH demontrasted a lower sympathetic activities response than younger individual or elderly without PPH. This fact also supported by the failure of geriatric patients in increasing appropriate heart rate (Trahair et al., 2014). From this heart rate spectral analysis study also determined that a 200% increase in sympathetic nervous activity would be necessary to prevent postprandial hypotension in elderly patients (Ryan et al, 1992; Masuda, 2003). Inappropriate heart rate compensation in those elderly PPH patients indicate a decrease in baroreflex sensitivity which is mediated by sympathetic nervous system (Van-Orshoven et al., 2008). There is also an increase in plasma noradrenaline level after meal in elderly, but the increase is lower than normal subject (Gentilcore et al., 2009).

As described previously, an intraduodenal glucose load do not influence sympathetic nerve activity mediated by gastric distention, despite an intraduodenal glucose load cause a significant fall in blood pressure in geriatric patients than younger patients. There are possibilities of gastrointestinal (GI) neurotransmitters and hormonals role. Nitric oxide (NO) is a vasoactive neurotransmitter with vasodilatory properties is released endogenously in the gastrointestinal tract and may regulate splanchnic blood flow. The role of NO can be shown using inhibitor of its production, such as, NG-nitro-L-arginine-methyl-ester atau NG-mono-methyl-L-arginine (Gentilcore et al.,

2009). In healthy elderly subjects, the decrease in blood pressure induced by an oral glucose load is attenuated by NG-nitro-L-arginine-methyl-ester, without any change in gastric emptying (Gentilcore,et al., 2008). Future studies are still needed in evaluating NO pathways mechanism in PPH patients.

A number of GI hormones, including insulin, glucagon-like peptide-1 dan 2 (GLP-1, GLP-2), calcitonin-gene-related peptide, neurotensin, vasoactive-intestinal peptide (VIP), bradykinin and substance P have been implicated in PPH. Postprandial hypotension response is attenuated by somatostatin, which inhibits the release of most gastrointestinal hormones (Jordan et al., 2000). However, the relevance of hormonal mechanisms to PPH remains uncertain because of inconclusive evidences. Among GI hormones that already mentioned before, currently, only GLP-1, GLP-2 and calcitonin-gene-related peptide which are proved to have a role in PPH, in spite of limited evidences.Glucagon-like peptide-1, an incretin hormone, is secreted from the intestinal 'L-cells' in response to oral glucose and acts to slow gastric emptying, which further will attenuate the postprandial fall in blood pressure. Agonist GLP-1 drugs, such as exenatide and liraglutide, which are now used widely in the management of type-2 diabetes, have been reported to have a beneficial effects in treating PPH patients (Masuda et al., 2003). Further investigations are still needed to evaluate GLP-1 role in PPH management. Glucagon-like peptide-2, which is co-secreted with GLP-1, has potent vasodilatory effects including in the mesenteric circulation and may also has a role in PPH. Calcitonin-gene-related peptide, another vasodilatory peptide released in response to meal ingestion, was reported in 1 study to correlate with the magnitude of the fall in blood pressure after an oral glucose load in 20 healthy subjects (Edwards, 1996).

Following a meal, there is an approximate doubling of blood flow through the superior mesenteric artery (SMA), coupled with a decrease in vascular resistance and peripheral blood flow, particularly to skeletal muscle. The postprandial increase of splanchnic blood flow has been shown to be dependent on size of the meal, meal composition and the delivery rate of nutrients to small intestine. However, there are no substansial differences in meal-induced MSA blood flow in PPH patients, implying that the disorder reflects a lack of cardiovascular compensation for the increase (Su, et al 2001).

All the macronutrients have the capacity to decrease blood pressure in older subjects significantly when given orally or intraduodenally, whereas there is no decrease of blood pressure in the healthy young. From those macronutrients, glucose appears to cause the most rapid fall in systolic blood pressure in healthy elderly and patients with PPH. Simple carbohydrate (78% glucose) induced a significant decrease in blood pressure in healthy older subjects, this was not evident following complex carbohydrate (78% starch), probably reflecting a slower rate of small intestinal absorption (Yonenaga et al., 2013). The effects of different nutrient combinations on PPH have not been formally evaluated, despite numerous studies have employed mixed meals for diagnostic purposes.

There is a wide inter-individual variation in the normal rate of gastric emptying, which is between 1 – 4 kcal/min and regulated primarily by inhibitory feedback arising from interaction of nutrients with the small intestine. In contrast, the intra-individual variation is modest because both posture and meal volumes only have minimal effects on emptying rates. Healthy aging appears to be associated with a modest slowing of gastric emptying. The more rapid the rate of gastric emptying, the more rapid the nutrients reach small intestine, the greater decrease in blood pressure occurs. The threshold of nutrient delivery speed to small intestine which may cause hypotension probably between 1 and 2 kcal/min, an effect that is apparently independent of the concentration of glucose infused (Bremholm, et al 2009).

Clinical Manifestations

Postprandial hypotension is related with a number of sequelae which impact adversely on quality of life, as well as increased mortality. However, it should be recognized that all studies relating to the clinical manifestations of PPH have substantial limitations, particularly in relation to the size of the cohorts studied, lack of appropriate control subjects, paucity of longitudinal assessment and potential cofounders, including the diseases associated with PPH, in addition to other methodological issues. Large-scale, propective studies are required and there is also a need for a validated instrument to assess symptoms of PPH (Wicklein, 2007).

The most common manifestations of PPH appear to be syncope, falls, angina, dizziness, nausea, light-headedness, and/or visual disturbance (Jansen et al., 1995a&Lipsitz et al., 1986a), so that there is an overlap with symptoms of frailty (Jansen et al., 1995b). A study of 16 geriatric patients with idiopathic syncope, demonstrated that 50% of the patients was diagnosed with PPH. However, most of the healthy individuals, the decreased of blood pressure as much as 25 mmHg is not sufficient to dysregulate brain autoregulation and based on that, it is likely most of the patients with PPH are asymptomatic. Postprandial hypotension should be suspected in elderly who complaint dizziness or fall soon after a meal (Barochiner et al., 2013). Although well-designed studies to evaluate the relationship of symptoms with the magnitude and time course of the fall in blood pressure are needed, there is anecdotal evidence that symptoms are more likely, and severe, when the reduction is greater.

Several studies demonstrated sufficient evidences that PPH is an independent mortality predictor, what is much less certain is the strength of this association.

Postprandial hypotension appears to increase cardiovascular-related mortality. In addition, PPH also appears to be a risk factor for cerebrovascular ischemia and arteriosclerosis. For example, in 401 older hypertensive patients, approximately 73% of the patients had PPH, then followed for 4 years, the presence of a decrease in systolic blood pressure following breakfast was associated with an increase in cardiovascular mortality of 20%. In a study of 179 nursing care residents (mean age 83 years) followed for 4.7 years, mortality was greater in individuals with PPH (145 vs 99 per 1000 person-years) (4) and there was a linear relationship between mortality with the magnitude of the fall in blood pressure so that mortality rates in patients who experienced blood pressure falls of ≤ 10 mmHg, 11-19 mmHg, 20-39 mmHg dan ≥ 40 mmHg being 89.1, 116.9, 144.4 and 156.1 per 1000 person-years, respectively. It seems that mortality does not to be predictable on the basis of symptoms, hence, even asymptomatic individuals with PPH may well be at risk (Uetani et al., 2012).

Risk factors for PPH in elderly include certain medications, type and time of meal, premeal blood pressure and specific comorbid conditions such as diabetes mellitus, Parkinson disease, hypertension, orthostatic hypotension, regular hemodyalisis. Polypharmacy appears to be a risk factor for postprandial hypotension which is frequently being forgotten especially diuretics, cardiovascular and psycotropic medications. Diet high in carbohydrate, having that at breakfast or lunch and being served as warm meal are causing more profound decrease in postprandial blood pressure (Uetani et al., 2012).

Diagnosis

As previously described, most of the PPH cases are not diagnosed due to non-specific symptoms and frequently asymptomatic, so as clinicians we shall have a high index of suspicious if the elderly patients come with a chief complaint of syncope or falls, especially if there are risk factors. The patients should undergo ambulatory blood pressure monitoring especially specific blood pressure measurement after meal, because PPH can occur up to 2 hours postprandial (Oberman et al., 1999). Blood pressure measurement is recommended at certain time include the meal that the patient considers most symptomatic, but 24-hours monitoring is still needed in order to determine the severity and frequency of postprandial hypotension.

The maximum decrease in blood pressure is tipically occurs within the first 35 minutes to 1 hour after a meal. However, monitoring of blood pressure and symptoms should continue for 2 hours after a meal, as the nadir in blood pressure can occur up to 2 hours postprandially. Because blood pressure responses to a meal are similar after an equally timed meal on different days, it is, in fact, possible to make the diagnosis of postprandial hypotension after only one abnormal test (Sidery et al., 1994). In contrast, a negative test cannot rule out the diagnosis and further blood pressure monitoring may be needed. Currently, postprandial hypotension is defined as a fall in systolic blood pressure > 20 mmHg, or a decrease to ≤ 90 mmHg when the preprandial blood pressure is ≥ 100 mmHg, within 2 hours of a meal. There have not been sufficient trials to correlate blood pressure decreases with symptoms. Therefore, diagnosis should be individualized, because patients can have symptomswith decreasing of blood pressure after meal or can also be asymptomatic at all.

Management

Management of PPH can be nonpharmacologic and pharmacologic and the parameter outcomes have usually been assessed by quantifying the magnitude of the postprandial blood pressure decline. Because the association with symptoms is weak, the effect of a treatment on symptoms can only be evaluated in studies of large cohorts, and even larger studies would be required to determine impacts of cardiovascular events/mortality. Previous studies are all short and limited to small cohorts, often including individuls who had more modest postprandial falls in blood pressure, which did not meet the strict criteria for diagnosis of PPH. Accordingly, larger, longer studies of the effects of treatment in PPH represent a priority. Despite these limitations, several management strategies for PPH appear effective (Sidery and Macdonald, 1994).

Nonpharmacologic Management

There are 2 main strategies may be used in nonpharmacologic management, related to the pathophysiologic of PPH that already decribed previously, including slowing or attenuate nutrient delivery to small intestine and or increasing gastric distention (Vioet et al., 2001). In addition, activities or exercise, such as walking for 10 minutes after meal, can also attenuate postprandial hypotension, despite the effect is only temporary (Shannon et al., 2002).

The attempts to decrease or delay the exposure of the small intestine to the products of nutrient digestion should be done by consuming smaller meals more frequently and delaying gastric emptying with guar guam or colder meal. There is limited evidence that patients with PPH may benefit from consuming smaller frequent meals (Jian et al., 2008). In a study with autonomic failure and PPH, comsumption of 6 smaller portion of meals may attenuate the decrease of blood pressure as much as 11-20 mmHg if comparing to consumption of the equivalent energy as

3 larger meals.Smaller meals may be related with less splanchnic blood pooling, so requiring less cardiovascular compensation, but a less meal size is also related with less gastric distention which is protective to the postprandial decrease of blood pressure. Further studies are required before clinical recommendations can be made in relation to meal size in PPH.

The effects of water drinking in PPH have been evaluated in 2 studies (Jansen et al., 1995a; Shannon, 2002). The onset of pressor effect to water is immediate, so water drinking should be performed immediately prior to a meal (Gentilcore et al., 2005). In patients with autonomic failure, volumes of 480 ml and 350 ml of water consumed prior to a meal reduced the magnitude of PPH by 21 mmHg and 13 mmHg, respectively. The protective effects were sustained for ≥ 60 minutes (Deguchi et al., 2007). The optimal volume, rate of ingestion of water and the effects to symptoms of PPH have not been formally evaluated and further studies are needed. However, given its nonexistent cost, safety, and apparent efficacy, drinking a modest volume of water prior to a meal can be recommended as therapy for PPH.

Postprandial exercise has been evaluated as a treatment for PPH in 2 studies (Oberman et al., 1999; Puvi-Rajasingham et al., 1998). Exercise is known to be associated with an increase in cardiac output and splanchnic vascular resistance, which may potentially increase postprandial blood pressure. Walking for 10 minutes which is perfomed within 20 minutes after a meal in 14 frail elderly with PPH can increase MAP 18 mmHg (Oberman et al., 1999). These effects sustained only for the duration of walking. Whereas, a study in 12 patients with primary autonomic failure, Postprandial blood pressure were less during light aerobic exercise (Puvi-Rajasingham et al., 1998). Although PPH does not appear to reduce exercise capability in the frail elderly, but it appears unlikely that exercise will be beneficial.

Pharmacologic Management

From studies that we have nowadays, there are several pharmacologic modalities for treating PPH include delaying disaccharide absorption with α-glucosidase inhibitor, blocking the release of peptides potentially responsible for splanchnic vasodilator with somatostatin analogues and vasopressin, direct stimulation of the sympathetic nervous system with caffeine, and lowering preprandial blood pressure with antihypertensive agents (Barochier et al., 2013).

Several studies have already evaluated the effects of α-glucosidase inhibitor in patients with PPH and demonstrated that α-glucosidase inhibitor are effective in attenuating the postprandial decrease of blood pressure but not eliminating PPH. Alpha-glucosidase inhibitor, acarbose, was given in doses of 50-100 mg 3 times a day, and voligbose, in doses of 100 mg once a day, 10 minutes before meals. The mechanisms of α-glucosidase inhibitor for PPH include slowing gastric emptying and inhibiting intestinal disaccharide absorption, both resulting in a reduction in splanchnic blood flow. These are associated with reduction in GLP-1 and GLP-2 secretion due to less carbohydrate in distal intestine. However, α-glucosidase inhibitor does not delay the absorption of simple carbohydrates, such as glucose, and also gastrointestinal side effects is more frequent in α-glucosidase inhibitor, despite these drugs are well tolerated in many patients with type 2 diabetes. Acarbose also appears to be effective in a number of disorders associated with PPH including elderly population, Parkinson disease, pure autonomic failure and MSA (Shibao et al., 2007, Ranganath, et al 1998). Further studies are still required because recent studies were acute and also do not determine the longterm efficacy and impact on symptoms before clear recommendations can be made. Despite this, acarbose may be currently the best pharmacologic treatment for PPH.

The effects of the somatostatin analogue, octreotide, in PPH have been reported in several related conditions, such as autonomic failure, hypertension and healthy elderly in doses ranging from 0,4 mcg/kg – 0,8 mcg/kg. Octreotide has unique properties that it is the only pharmacologic treatment which may completely prevent PPH. The effects of this drug may relate to inhibition of the release of vasoactive gut peptides, with a consequent reduction in splanchnic blood volume and or directly cause splanchnic vasoconstrictor. However, there are some limitation in octreotide, such as expensive, requires daily injections (injection sites pain), side effects (abdominal pain), and QT prolongation. Long acting somatostatin analogue, such as lanreotide, are often tolerated better than octreotide, but there is no information about their use in PPH.

Caffeine is one of the pharmacologic treatment modalities for PPH, but the evidence to support its efficacy is still limited. The doses studied have ranged from 50 mg to 250 mg, as a tablet, coffee, or tea. Studies in older patients with PPH and in patients with autonomic failure showed that caffeine has a modest effect to reduce the magnitude of PPH, by direct stimulation of the sympathetic nervous system. While caffeine has been reported to be well tolerated, only 3 studies in more than 10 subjects, so the recommendation of using caffeine in PPH still have to be evaluated further (Trahair et al., 2014).

The effects of antihypertensive therapy on postprandial blood pressure have been reported twice (Connelly et al., 1995; Jansen et al., 1988), both studies evaluated elderly hypertension population without known PPH. The drugs used were isosorbide dinitrate (20 mg) or nicardipine hydrochloride (20 mg) in a 3-week study (Connelly et al., 1995), and nitrendipine (20 mg) or hydrochlorothiazide (50 mg) in a 12-week study (Connelly, 1995)). Both study demonstrated improvement

of the modest postprandial falls in blood pressure, but not abolishment. The mechanism of the improvement probably reflects lowering of pre-meal blood pressure, as opposed to a direct effect of these antihypertensive medication on postprandial blood pressure.

In patients with PPH, a combination of denopamine 10 mg (β-adrenergic agonist) and midodrine 4 mg (α1-adrenergic agonist) was reported to attenuate the postprandial fall in blood pressure. Midodrine can increase vascular resistance and denopamine can increase cardiac output, then acting on 2 pathways of hemodynamic dysregulation implicated in PPH. Intravenous vasopressin, that is given continuously following oral glucose, could attenuate the postprandial decrease of blood pressure in 5 patients with MSA (Hiraya 1993). Randomized placebo-controlled study is needed to confirm the positive effects in PPH patients.

There were several drugs that had already been studied for preventing and attenuating the postprandial decrease of blood pressure including cimetidine 300 mg, diphenhydramine 50 mg, dihydroergomtamine 50 mg and propranolol 40 mg. Indometacine 50 mg and 3,4-DL-threo-dihydroxyphenylserine (DL-DOPS) are reported to attenuate PPH in autonomic failure (Hiraya et al., 1993).

Withrawal of furosemide therapy may benefit patients with PPH. In 24 elderly subjects with heart failure, a single dose of 40 mg could increase the hypotensive response to a mixed meal from 19,6 ± 2,1 mmHg to 28,5 ± 6,2 mmHg (94). Another study in 20 patients heart failure (mean ages 75 years), 55% of whom had PPH, withdrawal of furosemide therapy reduced the magnitude of PPH at a 3-month follow-up (Mehabnoul-Schipper et al., 2002).

CONCLUSION

Postprandial hypertension, an important clinical problem, is a unrecognized cause of syncope and falls in elderly population and cause an overall increased risk of mortality. Not only PPH is often occurred in healthy elderly, but also PPH can occur in several medical conditions, such as type 2 DM, Parkinson Disease and dysregulation of autonomic nervous system. The pathophysiology of PPH is multifactorial including the increase in splanchnic blood flow, the rate of small intestinal nutrient delivery, gastric distention, neural and hormonal mechanisms. Clinians should have a high index of suspicious of PPH because most of PPH patients are asymptomatic or present non-specific symptoms. Management of PPH can be both nonpharmacologic and pharmacologic, and currently proof to be effective are drinking water immediately before a meal and acarbose. The main purpose of the management of PPH is attenuating the postprandial fall of blood pressure accompanied with improvement of related-symptoms.

Futher studies are still needed particularly in pathophysiologic and management aspect.

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