A recent study examined the role of vitamin E (tocopherol) in preventing CI-AKI and concluded that prophylactic oral α- or γ-tocopherol with 0.9% saline protected against CI-AKI in patients with chronic kidney disease (CKD) undergoing coronary procedures. In this study, 305 patients were randomly assigned to prophylaxis administration with 0.9% saline infusions plus daily oral medication comprised of either (i) placebo (n = 101), (ii) α-tocopherol (n = 102) or (iii) γ-tocopherol (n = 102) starting 5 days before and ending 2 days after coronary procedures. CI-AKI developed in 14.9% in the placebo group, 4.9% in the α-tocopherol group (P = 0.02 versus the placebo group) and 5.9% in the γ-tocopherol group (P = 0.04 versus the placebo group). In patients with diabetes, hypertension, anaemia, aged over 55 years, males or with contrast agent dosages >120 mL, α-tocopherol showed a greater effect than γ-tocopherol when compared with the placebo group (P < 0.05). This strategy should be compared to the abovementioned strategies to ascertain its effect vis-à-vis fluid protocols and NAC use for preventing CI-AKI in elective radiographic procedures.

REFERENCES


Electronic health records from 1997 to 2010 in the CALIBER (CArdiovascular research using LInked Bespoke studies and Electronic health Records) programme, from 225 primary care practices registered with it, were used to set up a cohort of 1.25 million patients, 30 years of age or older and initially free from cardiovascular disease, 20% of whom received blood pressure-lowering treatments. The heterogeneity in the age-specific associations of clinically measured blood pressure with 12 acute and chronic cardiovascular diseases were studied, and estimated lifetime risks (up to 95 years of age) and cardiovascular disease-free life years lost adjusted for other risk factors at index ages 30, 60 and 80 years were calculated. Patients’ blood pressure was measured at initial presentation at primary care clinic closest to index date, and they were classified as hypertensive at a blood pressure higher than 140/90 mmHg. Isolated systolic hypertension (>140 mmHg) and isolated diastolic hypertension (>90 mmHg) were also noted. The cardiovascular diseases recorded were stable and unstable angina, myocardial infarction, unheralded coronary artery disease death, heart failure, sudden cardiac death, transient ischaemic attack, ischaemic stroke, subarachnoid haemorrhage, intracerebral haemorrhage, peripheral arterial disease and abdominal aortic aneurysm. Unclassified stroke was considered an ischaemic stroke, as 87% of all unclassified strokes have been shown as ischaemic origin in previous studies.

During 5.2 years median follow-up, 83098 initial cardiovascular disease presentations were recorded. In each age group, the lowest risk for cardiovascular disease was in people with systolic blood pressure of 90-114 mmHg and diastolic blood pressure of 60-74 mmHg, with no evidence of a J-shaped increased risk at lower blood pressures. Associations with high systolic blood pressure were highest for intracerebral haemorrhage (hazard ratio 1.44 [95% CI 1.32-1.58]), subarachnoid haemorrhage (1.43 [1.25-1.63]), and stable angina (1.41 [1.36-1.46]) and weakest for abdominal
aortic aneurysm (1.08 [1.00-1.17]). Higher systolic blood pressure had a greater effect on angina, myocardial infarction and peripheral arterial disease, while raised diastolic blood pressure had a greater effect on abdominal aortic aneurism. Pulse pressure associations were strongest for peripheral arterial disease (1.23 [1.20-1.27] and inverse for abdominal aortic aneurysm (HR per 10 mmHg 0.91 [95% CI 0.86-0.98])). People with hypertension (blood pressure ≥ 140/90 mmHg or those receiving blood pressure-lowering drugs) had a lifetime risk of overall cardiovascular disease at 30 years of age of 63.3% (95% CI 62.9-63.8) compared with 46.1% (45.5-46.8) for those with normal blood pressure and developed cardiovascular disease 5.0 years earlier (95% CI 4.8-5.2). Stable and unstable angina were responsible for most (43%) of the cardiovascular disease-free years of life lost associated with hypertension from index age 30 years, whereas heart failure and stable angina accounted for the largest proportion (19% each) of years of life lost from index age 80 years.

The widely held assumptions that blood pressure has strong associations with the occurrence of all cardiovascular diseases across a wide age range, and that diastolic and systolic associations are concordant are not supported by the findings of this high-resolution study.[1] The assumption that “lower is better” for blood pressure in relation to vascular events and mortality in patients with vascular disease may not be true. This can be ascribed to a J-curved relationship between blood pressure and cardiovascular events. The Secondary Manifestations of Arterial Disease (SMART) Study followed up 5788 patients with symptomatic vascular disease for new vascular events (i.e. myocardial infarction, stroke or vascular death) and all-cause mortality. During a median of 5.0 years (interquartile range: 2.6-8.1 years), 788 patients experienced a new vascular event, and 779 died. Overall, the covariate adjusted relationship between mean baseline systolic, diastolic or pulse pressure, and the occurrence of vascular events followed a J-curve with increased event rates above and below the nadir blood pressure of 143/82 mmHg. A similar non-linear relationship was found for diastolic pressure and all-cause mortality. Elevated blood pressure was not associated with increased morbidity and mortality in patients with recently diagnosed coronary artery disease, 65 years, and pulse pressure of 60 mmHg. The authors note that low blood pressure could also be a symptom rather than a cause of disease, especially in this subgroup. Blood pressure level below and above 143/82 mmHg was found as an independent risk factor for recurrent events in patients with manifest vascular disease in this study.[3] The lifetime burden of hypertension is enormous, notwithstanding modern treatment. Both studies emphasize the need for new blood pressure-lowering strategies in light of these findings and further randomized trials to assess their impact on human lives.

REFERENCES


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