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Abdominal fat is dangerous for arteries even in older people

Both cross-sectional and longitudinal studies have shown that body composition changes with ageing, with increase in fat mass and decrease in muscle mass. Ageing leads to increase in the amount of fat, even without changes to body weight [1]. In normal and obese subjects, body weight tends to increase, peaking at an age of about 65 years in men and later in women, and then decreasing with further ageing [2]. Studies using both anthropometry or computed tomography (CT) to evaluate body fat distribution have clearly shown that with ageing, intra-abdominal fat volume increases, while fat in thighs and calves decreases [1]. Very recently, it has been shown that with ageing, there is also increase in the amount of fat inside and around muscles, as well as ectopic fat deposition [3].

Increase in the amount of visceral abdominal fat and ectopic fat deposition occurs with weight gain and also with ageing.

In this issue of *Age and Ageing*, Van Dijk *et al.* [4] confirm the key role of fat distribution on CV risk factors and particularly on blood pressure and arterial stiffness components in older people. In a group of 216 older people with a mean age of 77 years, they report an association between central fat percentage, as evaluated by dual-X-ray absorptiometry (DXA), and aortic blood pressure components. In particular, they found a positive association between augmentation pressure (AP), an index of arterial stiffness, but not augmentation index (AI), and central fat percentage. Their findings regarding the association of AP and not AI with fat distribution confirms and strengthens the importance of this index of arterial stiffness, which is not influenced by the age-related increase in pulse pressure and should be preferred to AI in this population [5].

Fat distribution has been shown to be linked to higher risk of diabetes, hypertension, and dyslipidaemia in young as well as in old subjects. Fat distribution indices are also linked to the so-called metabolic syndrome even in older ages. There are several mechanisms that may link abdominal visceral fat to subclinical vascular damage. Adipose tissue (AT), the major energy storage of an organism, is now well recognised as a complex and highly active metabolic and endocrine organ. Adipocytes, the primary composing cells of AT, not only provide a flexible storage depot for excess nutrients, but are also endocrine cells, secreting hormones that regulate energy intake and expenditure, as well as insulin metabolism and inflammation.

A strong association has been reported between visceral fat and several cytokines, such as interleukin-6, plasminogen activator inhibitor-1 and leptin, that have been shown to be related to endothelial dysfunction [6]. Visceral fat has also been shown to be negatively associated with adiponectin [7], whose protective effect on arteries is well known. Elevated free fatty acid (FFA) levels, whose association with endothelial dysfunction is known, have been shown to be higher in subjects with visceral obesity than in those with subcutaneous obesity [6]. Furthermore, the well-known association between abdominal obesity and impaired lipoprotein metabolism may be another link with vascular damage [8]. Indeed a possible role of angiotensin could be suggested in the

development of endothelial dysfunction. Recently, Yasue *et al*. showed an association between increased values of angiotensin and AT in obese subjects, a novel finding that is likely to open new vistas in clinical application in obese subjects [9].

The findings of the paper of Van Dijk *et al*. are in line with a previous report showing a link between abdominal obesity and arterial stiffness, evaluated by carotid-femoral pulse wave velocity, in a group of older people with metabolic syndrome [10] and with those of Ferreira *et al*. [11] in a young population with a mean age of 36 years. Another study in 169 newly diagnosed hypertensive patients showed an association between arterial stiffness and waist circumference [12].

Actually Van Dijk *et al*. in their paper did not observe any association between peripheral AT and blood pressure and arterial stiffness indices.

A protective role of subcutaneous AT in metabolic alterations and artery distensibility in young as well as in older subjects has been suggested [11, 13, 14]. Shay *et al*. showed that adiposity in the lower extremities may attenuate metabolic risk at a given level of abdominal adiposity in overweight and obese adults [13].

Ferreira *et al*. showed a negative relation between peripheral fat and arterial stiffness in a young population, as evaluated by carotid-femoral PWV as did Snijder *et al*., in a sample of healthy older people [14].

The association between subcutaneous adipose tissue and cardiovascular risk shows pathophysiology plausibility. It is known that subcutaneous AT has a more anti-inflammatory profile. Moreover, subcutaneous peripheral AT is known to have lower lipoprotein lipase activity and low fatty acid turnover than abdominal AT and thus lower FFA serum levels [15].

The paper of Van Dijk emphasises that age-related body composition changes are harmful. It should be noted that the results of the present paper could have been even more relevant if PWV had been used to test vascular stiffness or if imaging methods, such as CT or magnetic resonance, had been used to evaluate visceral abdominal AT instead of DXA. A clear message seems to emerge from the data of this paper: prevention of abdominal fat gain across ages is important to reduction of the risk of cardiovascular disease.

Further studies are needed to confirm the protective role of peripheral fat.

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