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# Abdominal Aortic Aneurysm in Patients with Coronary Artery Disease: A Review Article

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## 1. Introduction

Abdominal Aortic Aneurysm (AAA) is defined as a localized and permanent dilatation of the abdominal aorta, beyond 50% of the normal aorta diameter (Schermerhorn, 2009). The prevalence of AAA ranges from 1.3% to 8.9% in men and 1% to 2.2% in women (Sakalihasan et al., 2005; Singh et al., 2001). Incidence of AAA is on the rise in parallel with a globally ageing population, higher clinical suspicion and improved accuracy of imaging methods (Best et al., 2003; Prisant & Mondy, 2004). AAA is an important problem for public health, since AAA rupture is the tenth leading cause of death in American white men 65 to 74 years of age (Upchurch & Schaub, 2006). Most AAA remain asymptomatic until rupture occurs. Half of the patients with a ruptured AAA reach the hospital alive, with an additional operative mortality of 30-60%. On the contrary, an elective AAA repair, recommended in most patients with an abdominal aortic diameter exceeding 50-55 mm or rapid growth (> 1 cm/y), is associated with a mortality risk of 2% to 6% (Kurvers at al., 2003; Sakalihasan et al., 2005). Health organizations recently recommended one-time screening for AAA by ultrasonography for men aged between 65 and 75 years with a smoking history, thereby reducing AAA related mortality rates by 50% (Cosford & Leng, 2007; Ehlers et al., 2008; Ferket et al., 2011; Moxon et al., 2010; Takagi et al., 2010). However, they advised against screening in men below 65 and over 75 years, and in women, since the number of AAA-related deaths that can be prevented by screening these populations is too small. AAA and atherosclerosis share several risk factors, such as male sex, age, smoking and arterial hypertension (Forsdahl et al., 2009). In population – based studies, AAA is independently associated with pre – existing coronary arterial disease (CAD) (Golledge et al., 2006; Kent et al., 2010). The high prevalence of CAD among patients with AAA is well known, with an impact on short term survival after AAA repair (Falk et al., 1997). Indeed, coronary investigation is often required prior to aortic surgery, finding a concomitant CAD prevalence of 31% to 90% (Kioka et al., 2002; Sukhija et al., 2004; Van Kuijk et al., 2009). In contrast, the opposite relationship, namely the prevalence of AAA among patients with CAD, has been explored only in some recent cohorts or in subgroups of patients. The possibility that AAA could be more prevalent in this population, as compared with the general population, has been suggested by these previous studies, with limited, incomplete

and often conflicting results. Accordingly, no specific recommendation is available on AAA screening in patients with advanced atherosclerosis. To our knowledge, we present the first systematic review, based on a thorough literature survey, which delineates the prevalence and predictive risk factors of AAA among CAD patients. We thus aim at identifying CAD patients at high risk for AAA development and discussing the usefulness of AAA screening in such subpopulations.

## 2. Methods

A systematic English-language literature search has been performed using the MEDLINE/Pubmed, Cochrane and Embase databases to identify all studies on the prevalence and risk factors of AAA among CAD patients, published from January 1985 to January 2011. The key words searched for were AAA and CAD. The title and relevant abstracts have been screened for studies which statistically analyzed prevalence data of AAA in CAD patients. In addition, the references of eligible papers were screened for further relevant studies. Two authors independently reviewed and extracted data from each article using a specific data extraction form: publication year, characteristics of study population including CAD severity, AAA definition, size, prevalence, associated risk factors and circulating biomarkers. Discrepancies were resolved by consensus.

## 3. Characteristics of abdominal aortic aneurysm in coronary artery disease patients

Seventeen studies (8308 patients) published between 1991 and 2010 were eligible, meeting the predetermined inclusion criteria (table 1). Study population ranged from 72 to 2819 patients. For some studies, only the subgroup of CAD patients received our attention (Abela et al., 2009; Alcorn et al., 1996; Benzaquen et al., 2001; Bonamigo & Siqueira, 2003; Goessens et al., 2006; Jaussi et al., 1999; Nemati et al., 2009).

In these studies, the prevalence or incidence of AAA among CAD patients oscillated between 0.48% and 18.2%, with 8 studies above 9% (upper limit of AAA prevalence in population – based studies). The prevalence of AAA eligible for surgical repair varied from 0% to 5.2 % of the study population, and from 0% to 36.7% of the AAA population (table 2). AAA diameter in these studies ranged between 25 and 87 mm. Five studies included a control population, age and sex matched in most cases, and without angiographic CAD (Bergersen et al., 1998; Bonamigo & Siqueira, 2003; Madaric et al., 2005; Nemati et al., 2009; Wang et al., 2008). AAA prevalence in these control groups were always significantly below the prevalence in the CAD group, ranging from 0 to 3.3%.

## 4. High heterogeneity of studies

The wide variations in AAA prevalence among the precited 17 studies could be explained by differences in severity of CAD, AAA definition, age and sex distribution, geographical localisation, prevalence of associated risk factors and pre-existing atherosclerosis-related morbidity.

Inclusion criteria clearly stated a wide range of CAD severity: current coronary angiographic data with various thresholds of significant coronary narrowing; data on previous coronary events; data on current or previous coronary revascularisation procedures whether endovascular or surgical. Nine out of the 17 studies included patients

| Reference | Study Design, Country | n patients (% male) | AAA (%) |
|---|---|---|---|
| Nevelsteen et al., 1991 | Post CABG, Belgium | 100 (80) | 11* |
| Alcorn et al., 1996 | Aged > 65 years, MI, angina, CABG, significant stenosis on coronary angiography, USA | 1244 | 13.8 |
| Bergersen et al., 1998 | Elective CABG patients, USA | 192 (65.6) | 18.2 (13.0*) |
| Jaussi et al., 1999 | Patients referred for Transthoracic Echocardiography with ischemic heart disease, Switzerland | 72 | 15.3* |
| Benzaquen et al., 2001 | Significant angiographic coronary stenosis, Canada | 99 (75) | 8.1* |
| Bonamigo & Siqueira, 2003 | Male aged > 54 years, severe ischemic disease (stenosis > 70%) and previous coronary surgery, or important lesions on cardiac catheterism, Brazil | 501 | 6.8* |
| Monney et al., 2004 | Male aged > 60, Post CABG, Switzerland | 395 | 10.1* |
| Calderwood & Welch, 2004 | CABG patients, United Kingdom | 118 | 15.3 |
| Madaric et al., 2005 | Age > 60 years, coronary narrowing ≥ 50%, Slovak Republic | 109 (90) | 14.7 (8.3*) |
| Goessens et al., 2006 | "SMART Study", MI, unstable angina or history of CAD or CABG/PCI, The Netherlands | 1034 (83) | 3.7 (2.2*) |
| Wang et al., 2008 | Age > 60 years, coronary narrowing ≥ 50%, China | 209 (72.7) | 0.48* |
| Abela et al., 2009 | Significant coronary stenotic lesions, United Kingdom | 87 (77) | 13.8 |
| Shirani et al., 2009 | Elective candidates for CABG, Iran | 2819 (69.6) | 2.09 |
| Nemati et al., 2009 | CAD (more than 50% constriction), Iran | 184 | 4.3 |
| Dupont et al., 2010 | Post CABG, France | 217 (87) | 6.9 |
| Long et al., 2010 | Acute coronary syndrome with coronary stenosis ≥ 50%, France | 304 (77) | 6.6* |
| Poon et al., 2010 | Elective patients for CABG, China | 624 (79.9) | 1.8 |

Table 1. Summary of studies reporting prevalence or incidence (*) rates of abdominal aortic aneurysm (AAA) among coronary artery disease (CAD) patients. CABG: coronary artery bypass graft, MI: myocardial infarction, PCI: percutaneous coronary intervention

| Reference | AAA diameter mean (SD), [range] (mm) | AAA > 50 mm % / study population | AAA > 50 mm % / AAA patients |
|---|---|---|---|
| Nevelsteen et al., 1991 | [30-65] | 4 | 36.7 |
| Bergersen et al., 1998 | 43.6 (13.1), [25-70] | 5.2 | 28.6 |
| Benzaquen et al., 2001 | 38 (14), [27-65] | 1 | 12,5 |
| Monney et al., 2004 | 38.9 (13) | 1.01 | 10 |
| Calderwood & Welch, 2004 | | 4.2* | 27.7* |
| Madaric et al., 2005 | | 4.6 | 31.2 |
| Goessens et al., 2006 | | 0.09* | 4.34* |
| Abela et al., 2009 | 42 (19.6), [30-87] | 1.1 | 11.1 |
| Shirani et al., 2009 | 30.70 (7.01), [25-61.7] | 0.07* | 3.4* |
| Dupont et al., 2010 | 35.3 (9.1), [26-53] | 0.9 | 13.3 |
| Long et al., 2010 | 33 (3.7), [30-45] | 0 | 0 |
| Poon et al., 2010 | [31.1-59.1] | 0.3 | 18.2 |

Table 2. Characteristics of abdominal aortic aneurysm (AAA) in coronary artery disease patients (*: >55 mm).

who were candidates for CABG or in the post CABG period (table 1). AAA was diagnosed by abdominal ultrasound, by measurement of either transverse or antero-posterior, or both infrarenal aortic diameters. AAA is conventionally defined when infrarenal aortic diameter is ≥ 30 mm (Moll et al., 2011; Wanhainen et al., 2001; Wanhainen et al., 2008). In the present review, fifteen studies used this definition. However, some studies diagnosed AAA when infrarenal aortic diameter was > 26 mm (Calderwood & Welch, 2004) or ≥ 25 mm (Shirani et al., 2009), while others used additional criteria: infrarenal to suprarenal ratio > 1.2 (Alcorn et al., 1996) or > 1.5 (Dupont et al., 2010), infrarenal exceeding suprarenal aortic diameter by at least 5 mm (Bonamigo & Siqueira, 2003), to compensate for individual variation in the diameter of the adjacent aorta. While two studies exclusively included male patients (Bonamigo & Siqueira, 2004; Monney et al., 2004), the other studies recruited a female patient proportion varying from 10 to 34.4%. Because age is an important risk factor for both CAD and AAA in population – based studies, five studies restricted their study population to patients above 60 years. While most recruited patients had a mean age between 60 and 70 years, some studies exhibited a wide age range (from 34 to 88 years; from 29 to 83 years; respectively) (Long et al., 2010; Poon et al., 2010). In contrast to a majority of CAD patients of Caucasian origin in these studies, we found two studies on prevalence of AAA in CAD patients of Chinese origin (Wang et al., 2008; Poon et al., 2010) and two on Iranian patients (Shirani et al., 2009; Nemati et al., 2009). These 4 studies reported the lowest AAA prevalence. It clearly appears that certain ethnic groups experience a disproportionately smaller burden of AAA (Salem et al., 2009). Emphasis should be done upon the fact that the size of abdominal aorta in these populations was quite smaller than in Caucasian population. Wang et al. and Poon et al. consistently found a mean maximal infrarenal aortic diameter inferior to studies of Caucasian population (Allison et al., 2008; Poon et al., 2010; Wang et al., 2008). Despite such heterogeneity between these observational cross – sectional

studies, one can fairly say that AAA prevalence among CAD patients exceeds that observed in population –based studies. It is also of interest that the prevalence of AAA in CAD patients was comparable to that reported in AAA screening studies of patients with peripheral vascular disease (7% -17%) and cerebrovascular disease (8.4% -20.2%) (Bentgsson et al., 1996; Kurvers et al., 2003; Palazzuoli et al., 2008; Simons et al., 1999).

## 5. Abdominal aortic aneurysm risk factors in coronary artery disease patients

We shall now stress on the association between AAA prevalence in CAD patients and the various coronary atherosclerosis risk factors or indicators which may be useful to identify patients at increased risk in a screening context. Of importance, some risk factors were examined in some studies but not in others (table 3).

### 5.1 Gender

Most studies including both male and female patients showed a trend towards a higher male prevalence among AAA cases (table 3) or detected AAA in male patients exclusively, in accordance with results of population – based studies (Dupont et al., 2010; Nemati et al., 2009; Nevelsteen et al., 1991; Wang et al., 2008). Only one study showed a statistically significant association between male sex and AAA prevalence (table 3) (Shirani et al., 2009).

| Reference | Male sex | Age | Smoking | Diabetes mellitus | Arterial hypertension |
|---|---|---|---|---|---|
| Bergersen et al., 1998 | 1.78 (0.67-4.7) | 5,7 (1,7-18,8)*, § | 7 (2,5-19,1)*,§ | | |
| Bonamigo & Siqueira, 2003 | | 1,09 (1,034-1,152)*, § | 0,96 (0,47-1,95) | 1,29 (0,58-2,86) | 1,53 (0,79-2,99) |
| Monney et al., 2004 | | *, § | 2,07 (1,05-4,1)*, § | 0,4 (0,14-1,16) | 2,67 (1,2-5,96)*, § |
| Madaric et al., 2005 | 1,11 (0,28-4,35) | 1,05 (0,95-1,17) | 4,85 (1,55-15,25)*, § | 0,11 (0,01-0,83)* | 2,66 (0,33-21,75) |
| Shirani et al., 2009 | 3,85 (1,65-8,99)* | age > 65: 1,79* | 1,63* | 2,32* | 1,85* |
| Nemati at al., 2009 | | * | | | |
| Dupont et al., 2010 | NS | 1.34 (0.47-3.83) | infinity* | 1,7 (0,58-4,99) | 0,76 (0,26-2,22) |
| Long et al., 2010 | 2,83 (0,64-12,51) | age > 60: 2,74 (1,07-7)*,§ | 2,01 (0,65-6,18) | 0,54 (0,18-1,66) | 1,07 (0,42-2,7) |
| Poon et al., 2010 | 2.17 (0.49-9.52) | 2.68 (0.74-8.96) | NS | NS | NS |

Table 3. Odds ratio (95% confidence interval) obtained for association studies between abdominal aortic aneurysm presence and some atherosclerosis risk factors, among coronary artery disease patients. *: $p< 0.05$ by univariate analysis, §: $p< 0.05$ by multivariate analysis, NS: non significant

Poon et al. exclusively detected AAA in male CAD patients when using the common AAA definition "infrarenal aortic diameter ≥ 30 mm" (Poon et al., 2010). However, when using an alternate definition "maximum infrarenal diameter ≥1.5 times the group mean infrarenal aortic diameter categorized by gender", AAA prevalence was greater with 2 out of 19 in female patients (infrarenal aortic diameter of 24.1 mm and 25.1 mm). This goes along with the suggestion by Grootenboer et al. by which the definition of 30 mm in population based studies for the average women is probably inappropriate and leads to an underestimation of AAA prevalence (Grootenboer et al., 2009). In these studies on AAA prevalence in CAD patients, one also notes that infrarenal aortic diameter mean (standard deviation), when available, was significantly lower in female compared to male patients : 15.5 (2.2) mm versus 18.8 (5.6) mm, $p<0.0001$ in Dupont et al. (Dupont et al., 2010), 16.8 (3.2) mm versus 20.2 (3.3) mm, $p=0.004$ in Nevelsteen et al. (Nevelsteen et al., 1991).

### 5.2 Age

Nine out of 17 studies reported a statistical evaluation of the association between age and AAA (table 3). Only 2 did not find an association (Dupont et al., 2010; Poon et al., 2010). Age was associated with AAA by multivariate analysis after adjustment for other known risk factors in 4 studies (table 3). In Nemati et al., ROC curve analysis showed that age of patients with CAD (67 years) predicted the presence of AAA with a sensitivity and specificity of 75% and 80%, respectively (Nemati et al., 2009). Although most guidelines contain recommendations that favour one time AAA screening for male patients 65 years or older in the general population, one can note that 6 out of the 17 studies gave data about AAA detection in men younger than 65 years (table 4).

| Reference | AAA age |
|---|---|
| Dupont et al., 2010 | 67.1 (9.8), [54-85] |
| Goessens et al., 2006 | 68.8 (11,5), [45-79] |
| Long et al., 2010 | 69 (11), [51-86] |
| Calderwood et al., 2004 | 64.8, [60-72] |
| Benzaquen et al., 2001 | 67.2 (5.4), [58-76] |
| Abela et al., 2009 | 68.8 (11,5), [45-79] |
| Nemati et al., 2009 | 68.2 (7,09) |

Table 4. Age distribution of abdominal aortic aneurysm (AAA) patients among coronary artery disease patients (mean (SD), [min-max])

Only the AAA that are vulnerable to rupture contribute to the benefit of screening at an earlier age (Ferket et al., 2011). As an example, in Dupont et al., 7 out of 15 AAA were detected in men aged 50 to 65 years with one of them exceeding 50 mm requiring surgical correction (Dupont et al., 2010). In Long et al., 7 out of 20 AAA were detected in CAD patients aged 50 to 60 years (Long et al., 2010).

### 5.3 Smoking

Over 90% of all AAA patients have a history of smoking, and nearly half are active smokers at the time of diagnosis (Powell et al., 1996). In population – based studies, smoking may be

more strongly associated with AAA than with CAD (3 fold) or with cerebrovascular disease (5 fold) (Lederle et al., 2003). It also was strongly associated with AAA progression and rupture (Badger et al., 2009) and the strength of its association with AAA persisted independently of the extent of atherosclerotic disease (coronary, peripheral artery disease) (Lee et al., 1997). Five out of the 17 studies on AAA prevalence in CAD patients showed smoking to be significantly associated with AAA by univariate analysis, 3 of them confirming this association by multivariate analysis (table 3). In Shirani et al., even more important was the duration of smoking as 8.6% of CAD patients with a history of smoking ≥ 40 years had AAA compared to an AAA prevalence of 2.6% in their whole CAD population (Odd Ratio (OR): 3.49, p=0.0001) (Shirani et al., 2009). Similar results were found in population based studies (Singh et al., 2001). The effect of smoking cessation has been investigated as well, and showed a slow decrease in risk for AAA of 4% per year (95% confidence interval 2 to 6) (Wilmink et al., 1999). However, when adjusting for smoking duration, the risk of AAA even 20 years after cessation of smoking was not statistically different from the risk of current smokers (Singh et al., 2001). Nontheless, the recent AAA clinical practice guidelines of the European Society for Vascular Surgery recommend smoking cessation to reduce the risk of AAA growth (Moll et al., 2011). This relative slow decline in risk, after cessation of smoking, differs strongly between AAA and CAD. It would be interesting to evaluate the impact of smoking cessation on AAA prevalence and progression among CAD patients.

### 5.4 Diabetes mellitus

Among the 17 studies on AAA prevalence in CAD patients, only 7 did focus on the association between Diabetes Mellitus (DM) and AAA prevalence (table 3). Five out of these 7 studies reported no association, while one showed an association by univariate analysis which was no more significant by multivariate analysis (Madaric et al. 2005). Shirani et al. reported a significantly higher frequency of AAA in diabetic patients compared with non diabetic ones (3.2% versus 1.4%, p=0.033) (Shirani et al., 2009). Of importance, Guijarro et al. studied 159 patients with CAD and found that microalbuminuria, but not DM, was a potent and independent predictor of AAA (OR: 7.56, 95% Confidence interval: 1.8-31.9) (Guijarro et al., 2006). DM, a strong coronary atherosclerosis risk factor, may therefore lack an association with AAA prevalence in CAD patients.

### 5.5 Arterial hypertension

Seven out of the 17 papers on AAA prevalence in CAD patients evaluated the association between AAA and arterial hypertension. Only Shirani et al. and Monney et al. confirmed a significant association (table 3) (Monney et al., 2004; Shirani et al., 2009). Arterial hypertension seems to be more positively associated with coronary heart disease and cerebrovascular disease than with aneurysm formation. These findings indicate that a history of hypertension should not be held as a significant risk factor for AAA among CAD patients.

### 5.6 Lipid profile and obesity

Most CAD patients receive a lipid modifying therapy including statins, fibrates and cholestyramine. In such populations, few studies did evaluate the association between serum lipoproteins levels or history of dyslipidemia and AAA prevalence (Dupont et al.,

2010; Monney et al., 2004; Shirani et al., 2009). None found a statistically significant association. Moreover, in view of current guidelines on atherosclerosis treatment, one cannot construct a study on association between basal lipid profile and AAA prevalence in CAD patients without lipid modifying therapy prescription. We can, therefore, not expect to withdraw conclusions in such a selective population.

As far as obesity is concerned, 3 studies reported on the lack of association between AAA and BMI and/or body weight in CAD patients (Dupont at al., 2010; Long et al., 2010; Monney et al., 2004). A potential association between obesity and AAA or infrarenal aortic diameter in CAD patients requires evaluation by further studies.

To date, in a CAD population, all evaluated potential AAA risk factors are also well – known CAD risk factors. Among these, only age and smoking seem to have a specific impact on AAA prevalence among CAD patients. Of importance, no known specific circulating biomarker of AAA in CAD patients is available.

## 6. Atherosclerosis and abdominal aortic aneurysm

### 6.1 Pathophysiology

The pathogenesis of AAA is poorly understood. AAA has traditionally been thought to be caused by atherosclerosis. Recently, this atherosclerosis theory has been challenged on the basis of epidemiologic, genetic and biochemical information, questioning whether atherosclerosis is a "bystander" condition or an active participant in the development and progression of AAA disease (Golledge & Norman, 2010; Johnsen et al., 2010; Lee et al., 1997; Reed et al., 1992; Trollope & Golledge, 2011). As seen in previously described studies on AAA prevalence in CAD patients, not all patients with atherosclerotic disease do present an AAA. On the opposite, several studies reported many AAA patients lacking concomitant atherosclerotic vascular disease. The link between AAA formation and atherosclerosis is also not strong in animal models. Animals developing atherosclerosis by dietary or genetic means scarcely develop an AAA. Conversely, animal models of AAA, by intraluminal elastase infusion or adventitial application of calcium chloride, do not require the presence of atherosclerosis. There are both similarities and differences in the pathogenesis of AAA and atherosclerotic lesions whether coronary, carotid or peripheral arterial diseases. Both involve inflammation, macrophage infiltration, increased vascular smooth muscle cells turnover and thrombus formation. Whereas proliferation of vascular smooth muscle cells (VSMCs) is a typical feature of atherosclerosis affecting vascular media and intima, the density of these cells is low in media and adventitia of aneurismal wall due to apoptosis. Atherosclerosis is also characterized by migrating VSMCs and macrophages, foam cell formation, lipid deposition, leading to endothelial dysfunction and increased intima – media thickness (Palazzuoli et al., 2008). On the other hand, AAA involves dilation of all layers of the aortic wall, destructive remodeling of medial elastin and collagen fibers by matrix metalloproteinases released by activated aortic wall macrophages (Wassef et al., 2007). These different pathological features of CAD and AAA are further emphasized by the results of above mentioned observational studies. Indeed, some of the risk factors for CAD did emerge as being independently associated with AAA in severe CAD populations such as cigarette smoking and age. However, it is not possible to determine from these studies whether these risk factors did so through a pathway of promoting atherosclerosis, in turn increasing the risk of AAA, or if they promoted AAA further leading to CAD development. A third theory could be that both pathways act to some extent, subsequently stimulating the

development of the other. New integrative, hypothesis – driven, carefully designed human and animal studies should shed a light on the sequential development of AAA and coronary atherosclerosis.

### 6.2 Severity of CAD and AAA

Severity of CAD has been differently described in the aforementioned studies. Long et al. described a significant positive association between AAA and previous coronary events (previous acute coronary syndromes with proven coronary stenosis of 50% or greater, CABG or percutaneous coronary intervention), but not with the number of stenosed coronary vessels (Long et al., 2010). Nemati et al. as well as Shirani al. reported no association between AAA and number of stenosed coronary vessels or left main coronary vessel stenosis (Nemati et al., 2009; Shirani et al., 2009). Monney et al. confirmed these results regarding the lack of association between AAA and number of stenosed coronary vessels, NYHA classification of Angina Pectoris and number of bypass (Monney et al., 2004). Nevertheless, there was a reduced risk among patients with a single coronary vessel affection as compared to patients with CAD involving two or three vessels. Likewise, Dupont et al. did not detect an association between AAA and history of unstable/ stable angina, myocardial infarction, coronary angioplasty, and number of diseased coronary vessels (Dupont et al., 2010). Such an association or lack thereof between AAA and CAD severity has not been evaluated in population – based studies. It would also be important to assess the relationship between extent of coronary stenosis and infrarenal aortic diameter and/or risk of AAA rupture.

### 6.3 Concomitant vascular disease to abdominal aortic aneurysm and coronary artery disease

Atherosclerosis is a systemic inflammatory vascular disorder involving multiple arterial beds, so it makes sense to evaluate AAA prevalence among patients when Peripheral Arterial Disease (PAD) and/or Carotid Artery Stenosis (CAS) coexist with CAD. Patients with PAD seem to be at particularly high risk for AAA development with an overall prevalence greater than 10%, twice that of the general population (Barba et al., 2005; Galland, et al., 1991). In Barba et al., patients with tibial disease had a significantly higher AAA prevalence than those patients with aortoiliac or femoro-popliteal diseases. A systematic review of population based screening studies has shown a positive association of PAD with AAA (OR 2.5) (Palazzzuoli et al., 2008). The relationship between AAA and CAS is more difficult to apprehend. In patients aged above 65 years with severe CAD awaiting CABG, a recent study showed the presence of an AAA to be a significant predictor of ≥ 70% internal carotid artery stenosis by univariate analysis (Kiernan et al., 2009). Furthermore, a higher prevalence of AAA has been established in patients with CAS (Gratama et al., 2010; Kang et al., 1999; Karanjia et al., 1994; Kurverts et al., 2003). However, recent case control studies did not find any evidence for more carotid atherosclerosis in AAA patients (Cheuk et al., 2007; Palazzzuoli et al., 2008). In an attempt to better elucidate this relationship, Johnsen et al. reported significantly more carotid atherosclerosis in abdominal aortic aneurysm diameter ≥ 27 mm and in AAA (Johnsen et al., 2010).

Among CAD patients, 5 studies appraised an association between AAA and PAD, while the association between AAA and CAS was analyzed in only 4 studies (table 5). Regarding PAD, Benzaquen et al. and Dupont et al. reported a significant association (Benzaquen et al.,

2001; Dupont et al., 2010). This was further confirmed by Guijarro et al. in 157 patients with CAD (angina, acute coronary syndromes, or myocardial infarction) out of a cohort of 269 patients with symptomatic atherosclerosis (Guijarro et al., 2006). PAD was a potent and independent predictor of AAA (Odd Ratio: 6.0, 95% CI: 1.4-26.6). Of importance, in Benzaquen et al., difficulty advancing the catheter into the aorta during coronary catheterization was the only independent predictor of AAA presence by multivariate analysis (Odd Ratio: 11.1, 95% CI: 4.6-26.6) (Benzaquen et al., 2001). The association of AAA with concomitant lower extremity PAD may be due to the strong pathophysiologic association of both AAA and PAD with smoking. Both the aorta and lower extremity arteries are especially susceptible to injury by exposure to all forms of tobacco and cigarette smoking in particular (Baumgartner et al., 2008). As for CAS, its presence was associated with an increased risk of AAA in 3 out of 4 studies (table 5). In Shirani et al., AAA frequency was 10.8% in patients older than 65 years and CAS >50% (Shirani et al., 2009). In Dupont et al., severe CAD male patients aged below 75 years with a smoking history had an AAA prevalence of 24% when they also had PAD and/or CAS, versus 4.4% in the absence of either condition (p=0.007) (Dupont et al., 2010).

| Reference | Carotid stenosis | Peripheral arterial disease |
|---|---|---|
| Benzaquen et al., 2001 | | 4.7 (2.1-10.3)* |
| Bonamigo & Siqueira, 2003 | | 0,66 (0,21-2,1) |
| Monney et al., 2004 | 0,9 (0,36-2,24) | 1,88 (0,78-4,56) |
| Madaric et al., 2005 | 3,11 (1,02-9,46)* | 3,07 (0,91-10,38) |
| Shirani et al., 2009 | 4,72* | |
| Dupont et al., 2010 | 4,88 (1,37-17,33)* | 5,45 (1,77-16,78)* |

Table 5. Odds ratio (95% confidence interval) obtained for association studies between abdominal aortic aneurysm presence and carotid artery stenosis or peripheral arterial disease, among coronary artery disease patients. *: p< 0.05 by univariate analysis

## 7. AAA screening

AAA appears to meet many of the classic criteria of disease screening (Wilson & Jungner, 1968). First, AAA is an important health problem disease with life – threatening consequences when rupture occurs. Its prevalence in cross sectional studies is on the rise, not only because of better case – finding by abdominal ultrasonography, but also due to ageing of the population. In carefully designed randomized controlled trials, it has been well established that AAA screening in men aged 65 to 74 years is effective and decreases the risk of aneurysm rupture by almost 50%. In this group, after a single abdominal ultrasonographic evaluation, death from aortic rupture is rare within the subsequent 10 years (Ashton et al., 2002; Lindholt et al., 2002; Lindholt et al., 2005; Thompson et al., 2009; Wilmink et al., 2003). The evidence of clinical effectiveness was reinforced by a subsequent Cochrane Review—which estimated a 40% relative risk reduction in aortic rupture (absolute risk reduction from 0.27% to 0.16%)—and then by results from MASS after 7 years of follow-up (Cosford & Leng, 2007; Kim et al., 2007). A second point is that abdominal

ultrasonography is an ideal method for AAA screening as it is safe, non – invasive, cheap, lasting only few minutes, with a high sensitivity and specificity (Lederle et al., 1988; Lindholt et al., 1999). Thirdly, the best therapy is pre-symptomatic elective surgery in appropriately selected individuals, with a mortality of 5% for elective surgery, one tenth the mortality of emergency interventions (Golledge & Norman, 2009). Accordingly, population-based screening is currently being implemented in several countries (Scott et al., 2008; Wannheinen et al., 2005; Swedish council on technology assessment in health care, 2008). Most clinical practice guidelines contain recommendations in favor of one – time AAA screening for men 65 – 75 years old with a history of smoking using ultrasonographic scans (Ferket et al., 2011). However, this recommendation is poorly followed up in this target population, partly due to a lack of patients' compliance with an acceptance rate around 65% (Boll et al., 1998; Collin et al., 1988; Lederle et al., 1997; Lindholt et al., 1997; Simoni et al., 1995). Moreover, a recent study estimates that about half the patients with AAA were not eligible for screening under current guidelines and would have been missed (Kent et al., 2010). A different approach, with screening of specific high-risk groups has been suggested (Derubertis et al., 2007) and is implemented within the Medicare programme in the United States (Lederle, 2008). No specific guidelines for AAA screening in CAD patients are available. In those articles dealing with AAA prevalence among CAD patients, we found a higher AAA prevalence among Caucasian patients (between 3.7 and 18.2%) compared to population – based studies, with a high prevalence of AAA above 50 mm, independently of CAD severity. Such prevalence seems to be higher, according to our review, when CAD patients have a smoking history and symptomatic atherosclerotic affection of other vascular beds (CAS and PAD). These data lend support to further urgent controlled randomized studies, dealing with AAA ultrasonographic screening in this specific subpopulation, with a longer follow up of included subjects. End goals would include cost effectiveness, AAA operability, long term survival, sufficiency of one – time screening and quality of life. The impact of cardiovascular co – morbidities has been implicated, by predictive scoring systems, to influence AAA prevalence in the subgroups of women and middle – aged men (50 to 65 years old) (Kent et al., 2010; Wanhainen et al., 2008). The screening of hospitalized CAD patients would be easily organized without adding administrative burden and of high efficiency, as evidenced by full attendance rates in studies of Monney et al. and Dupont et al.; as abdominal ultrasonography was proposed during hospitalization (Monney et al., 2004; Dupont et al., 2010).

## 8. Conclusion

This review of literature shows, despite the limited number of studies, that CAD patients present with a higher AAA prevalence compared to population – based studies. Some classic atherosclerosis risk factors (history of smoking, age, associated atherosclerotic affection of other vascular beds) maintain an association with AAA in such CAD populations. This has a two-fold consequence: first, AAA may not simply be an atherosclerotic complication, as suggested by a recent work based on TROMSO study, warranting further studies in AAA pathogenesis (Johnsen et al., 2010). Secondly, specific CAD subgroups (those showing the above mentioned common risk factors) may be at risk for a higher AAA burden, strongly justifying the need for further studies to evaluate the impact of AAA screening in such patients subgroups.

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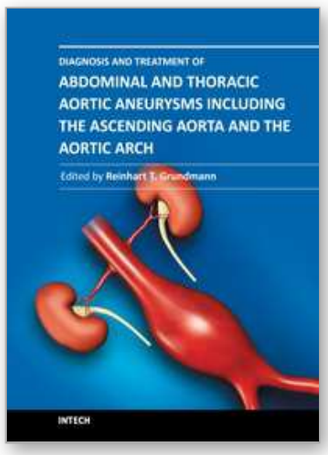


**Diagnosis and Treatment of Abdominal and Thoracic Aortic Aneurysms Including the Ascending Aorta and the Aortic Arch**
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This book considers diagnosis and treatment of abdominal and thoracic aortic aneurysms. It addresses vascular and cardiothoracic surgeons and interventional radiologists, but also anyone engaged in vascular medicine. The book focuses amongst other things on operations in the ascending aorta and the aortic arch. Surgical procedures in this area have received increasing attention in the last few years and have been subjected to several modifications. Especially the development of interventional radiological endovascular techniques that reduce the invasive nature of surgery as well as complication rates led to rapid advancements. Thoracoabdominal aortic aneurysm (TAAA) repair still remains a challenging operation since it necessitates extended exposure of the aorta and reimplantation of the vital aortic branches. Among possible postoperative complications, spinal cord injury (SCI) seems one of the most formidable morbidities. Strategies for TAAA repair and the best and most reasonable approach to prevent SCI after TAAA repair are presented.



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