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Review Article

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Abdominal aortic aneurysm: risk factors, new diagnosis realities and complications prevision

Abstract

Introduction: An abdominal aortic aneurysm is defined as a permanent expansion of aorta, superior or equal to 3, 0 cm, which has a 60-80% mortality in case of rupture. Its formation is caused by the weakening of aortic wall, that occurs when elastic and collagen fibers are degraded, being an entity that has an elevated risk for human live.[1–3]

Purpose: To put in evidence the contribution of modifiable and non-modifiable risk factors to the development of abdominal aortic aneurysms and to present new diagnosis paths and ways of previse complications of this entity.

Methodology: Review article based on the search made between February and April of 2015, through the search engines *ScienceDirect* and *Pubmed*. From the 2569 articles found, 33 were selected, with only 28 being used, dated between 2007 and 2015.

Discussion: The majority of the risk factors contribute to enhance the development of an abdominal aortic aneurysm, being the search of the individuals that present them fundamental. This search is especially important in women aged between 60 - 85years with cardiovascular risk factors or in those aged over 50years with an abdominal aortic aneurysm's family history, but also in those aged over 65years that present smoke or cardiac disease history.[4] Considering the complications associated with an abdominal aortic aneurysm, its frequent evaluation is crucial, having the purpose of determining the moment when the aneurysm reaches the minimum proposed diameter for surgery, which matches with a higher rupture risk and, consequently, a higher life danger.

Conclusion: The abdominal aortic aneurysms have risk factors similar to those found in atherosclerotic disease, although it is thought that these two entities feature different etiologies. Some 3D methods have emerged (speckle tracking), which has allowed a more detailed study of abdominal aortic aneurysms, bridging the limitations found in 2D methods. 3D methods, in association with mortality and morbidity prognosis studies, can justify and support the decision of intervening abdominal aortic aneurysms, which must be performed when the rupture risk exceeds the surgical ones.

Keywords: abdominal aortic aneurysm, risk factors, ultrasonography, diagnosis, prognosis

Volume 3 Issue 6 - 2015

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Received: November 10, 2015 | **Published:** November 17, 2015

Abbreviations: AAA, abdominal aortic aneurysm; ABC1, ATP, binding cassette, sub-family A, member 1; ACEI, angiotensin: converting enzyme inhibitors; AGE, advanced glycation end-product; CAD, coronary arterial disease; COPD, chronic obstructive pulmonary disease; CTA, computed tomographic angiography; EVAR, endovascular aneurysm repair; FEV1, forced expiratory volume in 1 second; FVC, forced vital capacity; LRP1, low density lipoprotein receptor related protein I; MMP, matrix metalloproteinase; RIFLE, risk injury failure loss of kidney function and end-stage kidney disease

Introduction

An abdominal aortic aneurysm (AAA) is an entity that carries a high risk to human life, whose rupture has a mortality rate of around 60-80%. It is defined as a permanent dilatation of the aorta, superior or equal to 3,0cm, which is due to the degradation of their elastic fibers and collagen and the consequent weakening of their walls. It becomes important to understand and study this pathology in order to build a clinical framework that may allow the prevention of possible complications.[1–3]

The AAA is mostly silent until the moment when the aortic wall becomes severely weakened, due to the expansion of the aneurysmal sac, culminating in his rupture. This aggravates the clinical framework, being the most frequent symptoms abdominal and dorsal pain, nausea, vomiting, fainting and systemic shock.[2]

It is therefore crucial an early diagnosis of the AAA, especially in high risk groups, in order to prevent their rupture and the consequent complications. The physical examination appears, most of the time, as the first indicator of the presence of a pulsating mass in the abdominal area (AAA), which is then confirmed by imaging exams, namely the abdominal vascular ultrasonography exam and the computed tomographic angiography (CTA), considered the gold standard for aortic imaging study, giving exact information of the anatomic location and the AAA size.[2] The high risk associated with the surgery intervention advises waiting until the AAA reaches a diameter of 5 to 5,5cm, in order to do it. Higher risk interventions, such as the thoracic or thoraco-abdominal aorta, should be done only when the aneurysm reaches a diameter greater than 6cm.[2]

This intervention is presented as the only available treatment for the AAA, it can be performed in two distinct ways. The first way is the classical surgery, in which an abdominal incision is made and the aorta is clamped above and below the affected portion. Then, the aneurysmal sac is opened and the aorta is sealed with a synthetic graft, sutured from end-to-end. The other way is the endovascular

repair (EVAR), where, via femoral puncture, a stent graft is inserted inside the aorta, between two healthy portions, thereby strengthening their weakened walls and sealing the aneurysmal sac. Given the less invasive nature of the EVAR, the patients have shorter recovery and internment time compared to the classical surgery[2] (Figure 1).

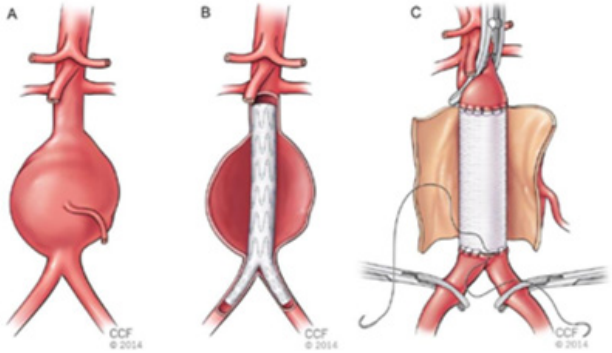

Figure 1 A) AAA
B) EVAR
C) Classical surgery.[2]

Purpose/methodology

The purpose of this article is to put in evidence the contribution of modifiable and non modifiable risk factors for the development of an AAA and to present the new ways of diagnosing this entity and prevent its complications.

To build this article, the main search engines were *ScienceDirect* and *Pubmed*, having the search lasted between February and April of 2015. The search key-words were: Abdominal aortic aneurysm; Thoracic aortic aneurysm; Risk factors; Aorta; Epidemiology; CPOD; Three vessel disease; Fruits and vegetables; Vitamin D deficiency; Hypertension; Diabetes; Familiar risk; Genetics; Gender differences; LRP1; Speckle Tracking; AAA surgery; Renal insufficiency; Spirometry; Respiratory function; Juxtarenal aneurysm; Infrarenal aneurysm. The 28 articles referenced, from a total of 33 selected and 2569 searched, are framed in a chronological profile between the years of 2007 and 2015.

AAA: Risk Factors

Literature refers that there are various risk factors which conditionate a bigger risk of developing an AAA, having been chosen, from the diverse possible classifications, to divide them in modifiable and non modifiable. These factors deserve a special attention, so that it might be possible to identify the individuals that belong to a higher risk group and that, by that, have a need of being study.

Modifiable risk factors

Arterial hypertension: Arterial hypertension is strongly associated with the development of AAAs. Kanematsu *et al* refer that there are various experimental studies that confirm this sentence, namely in animals, which demonstrate that a decrease in arterial pressure, using anti-hypertensive drugs – *amlodipine*-, reduced the AAAs incidence from 49% to 0% and also caused a total absence on adventitia inflammation or medium degeneration. These studies demonstrate the importance of the hemodynamic conditions on the aneurysm formation.[4,5] The hemodynamic stress inherent to this condition leads to an endothelial vascular response (inflammatory process), to elastin degeneration and to a consequent arterial wall dilatation. All this processes weaken the abdominal arterial wall and causes AAAs formation.[6]

Tobacco: Tobacco emerges as a major risk factor to the development of AAAs, being this risk higher in actual smokers when compared to ex-smokers, proportional to number of daily smoked cigarettes and inversely proportional to number of years after quitting smoking. Landenhed et al.,[7] affirm that in the smoking population the risk of AAAs incidence is present in 58/100.000 persons a year, comparatively to the non-smoking population, where the risk is present only in 15/100.000 persons a year.[2,7,8] The risk associated with tobacco varies considering a large scale, being the lower risk related with individuals that smoked till half a pack per day, for less than 10years and quitted for more than 10years; the higher risk is related with actual smokers, that smoke more than a pack per day, for more than 35years.[8]

Diabetes: The prevalence of diabetes and AAAs has increased in the last years, and despite the similar physiopathological mechanisms between both entities, their association is not yet at all clear, although it has been shown that diabetes may be responsible for reducing to half the AAAs prevalence, not only in small size aneurysms but also in those with size equal or greater than 4cm. Shantikumar et al.,[9] and Lederle[10] have equally demonstrated that diabetic individuals have less probabilities of having a ruptured aneurysm in the moment of the repair, as well as lower rates of expansion.[9,10] It is believed that this protective factor is related with hyperglycemia resulting from diabetes, which causes an increase in the collagen synthesis, leading to greater resistance to proteolysis and inhibition of MMP secretion (Matrix Metalloproteinase), promoters of the degradation of elastin and collagen. Diabetes also suppresses the plasmins, responsible for the MMP activation. This corroborates the results found in some studies where it was verified that diabetics have reduced MMP2 and MMP9 concentrations in the coronary arteries, just like in the aorta.[9,10] The stress to which the aortic wall is subjected is a key factor for the development of AAA. The presence, in diabetics, of thicker abdominal aortas and the occurrence of minor degradation of the aortic wall reduces the stress to which it is subject.[9,10] The hyperglycemic stage leads to the reduction of the proteolysis of the aortic walls of diabetics, since it promotes the increase of the stiffness of their walls. This occurs since diabetes causes glycation of extracellular matrix, with consequent formation of glycation end products (AGEs), which will form bridges with several proteins, namely the collagen and elastin from the arterial walls, and promote smooth muscle proliferation.[9] There are two distinct and contradictory theories regarding diabetes medication: one of them relates that if the protective effect is also dependent of the drugs that are taken, so the oldest drugs should be included in the count (insulin, sulfonylureas and metformin) of the most recent studies. But this raises doubts relatively to that protective effect, since it was already mentioned the effects of hyperglycemia in the protective role of diabetes and an insulin therapy decreases this hyperglycemic stage, going against some of the results that have been verified. In this perspective, the drugs are not seen as enhancers of this inverse association. Another perspective has in count comorbidities, like hypertension, atherosclerosis and renal failure that could affect diabetics and lead to the intake of other drugs, namely angiotensin-converting enzyme inhibitors (ACEI), statins and hypoglycemic agents. The ACEI are responsible for reducing vascular inflammation, increasing elastin deposition and inhibiting MMP. Prevent, as well, the aortic expansion and its consequent rupture. The statins decrease the expression of the MMP on the walls of the AAA. Metformin decreases the MMP2 concentration and the proliferation of smooth muscle cells. The rosiglitazone reduces the expression of MMP9 and the development and rupture of the aortic aneurysms. In this perspective, the drugs are presented as favorable elements to this inverse association.[9,10] It is known that the AAAs are associated with intraluminal thrombi and that the rate of expansion of AAAs

Citation: Teixeira F, Silva V, Batista P. Abdominal aortic aneurysm: risk factors, new diagnosis realities and complications prevision. *J Cardiol Curr Res.* 2015;3(6):1–6. DOI: 10.15406/jccr.2015.03.00127

is correlated with the growth of these thrombi, since they have big concentrations of MMP9. This way, the arterial wall with thrombus is thinner, has fewer smooth muscle cells and is less resistant to stress. Recent studies have shown that thrombi in diabetic individuals are denser, less porous and more resistant to fibrinolysis, which leads to a smaller release of MMP9 and to a consequent smaller aneurysmal expansion.[9] This protective effect of diabetes on the formation of AAA emerges as the strongest evidence that there is a big probability of aneurysmal disease and occlusive vascular disease be distinct entities, being expectable to presume that they also have different etiologies.[9,10]

Chronic obstructive pulmonary disease: Meijer et al.,[11] demonstrated a relation, tobacco independent, between COPD and AAAs prevalence and rupture, although it's known that the majority of the COPD cases in aneurysmal individuals are causes by smoking habits. It's also known that COPD remains, normally, unknown when both pathologies are present and that there isn't an association between COPD and the aneurysmal diameter expansion.[11] The relation found between these two pathologies can be justified by the convergence of their pathophysiological mechanisms that conditions a common susceptibility.[11] COPD presents itself as a serious disease in terms of morbidity, health care resources and mortality, being the pre-operatory (of aneurysmal surgery) lung function study as important as the cardiac function evaluation. In aneurismal patients there is a need to make an extraordinary pre-surgery preparation of the pulmonary function.[12,13] Spirometry emerges as a simple tool to determine the long-term mortality prognosis, after treating an AAA, appearing the individuals that present a severe COPD with low FEV1 and FVC values as those with higher mortality risk. A decreased FEV1 value is the major predictor of an increase in long-term mortality.[13] COPD severity and FEV 1 and FVC values must, considering what was said, be included in the pre-operatory risk models stratification, to allow to identify high risk patients with low short-term survival expectancy. Even in large AAAs, over 6cm, the elective treatment in individuals with severe COPD is rarely indicated.[13] In patients who have, simultaneously, pulmonary emphysema and COPD, the AAAs prevalence is higher in those with severe COPD, comparatively to those with moderate COPD. In individuals with AAAs, a similar relation was observed, having the moderate COPD a higher prevalence when compared to mild COPD. This suggests that patients with larger aneurysms may have more severe COPD degrees than patients with smaller aneurysms, which might lead to different post-operatory mortality risks.[13] There is a necessity of making a formal lung function evaluation through spirometry, before treatment and repair of AAAs. Patients with severe COPD have an increased mortality risk, which must be a matter to have in consideration when the balance between post-treatment death risk and AAA's rupture risk is done.[13]

Coronary Disease: It is known that there is an association between coronary arterial disease (CAD) and the prevalence of AAAs, which can be explained by the fact that these diseases share several risk factors.[14,15] However, in the group of diseases that affect the coronary network, only the three vessel disease arises as a risk factor independent of the traditional risk factors for the formation of AAA, regardless of age.[14,15] The prevalence of AAAs is higher in male individuals, so it has been given emphasis, in this gender, to the association between the three vessel disease and the AAAs. Having that in mind, considering Durieux et al.,[14,15] men aged over 65years with coronary arterial disease have a prevalence of 9,5% of developing an AAA. This prevalence becomes higher when we consider the same group of individuals, but who's CAD is specifically the three vessel disease (16%). The prevalence is equally greater if we consider male individuals, regardless of age, and that have the three vessel disease (14, 4%).[14,15] The coronary pathology appears as an independent risk factor to the development of AAAs, especially in men.[14,15]

Alimentation: Fruits and vegetables are rich in antioxidants, which may potentially contribute to reduce the risk of developing AAAs. The combined use of these two foods seemed to reduce this risk. Also the consumption of fish and nuts has shown to have a similar association. These effects are evident when the consumption of these foods is superior to three times per week. On the other hand, the consumption of red meat and fast food is related with the increased prevalence of AAAs.[8,16] Comparing the effect of fruits and vegetables individually, it is verified that the risk of developing AAAs decreases with the increased consumption of fruits, not being this association significant relatively to the consumption of vegetables.[16] The oxidative stress, that is the disequilibrium between the production and the reduction of reactive oxygen and nitrogen species, is related with the physiopathological process of AAAs formation, since it promotes the aortic wall inflammation. The antioxidants have the capacity to reduce the oxidative stress, which helps to explain, from a biological point of view, why the intake of the referred food allows this risk to reduce.[16] Having regard to what was said, it is necessary to understand why fruits and vegetables present distinct effects when consumed alone. One explanation could be the presence of different types of antioxidants in these foods, since fruits are rich in flavonoids, absent in the vegetables, that proved to have a relevant role in the inhibition of the reactive oxygen species, decreasing that way the oxidative stress that may affect the abdominal aorta.[16]

Hypovitaminosis D: Van de Luijtgaarden et al.,[17] concluded that vitamin D deficiency is associated with a mortality excess. It is increasingly clear that vitamin D has a much larger number of actions in the human body than those known about calcium homeostasis and bone metabolism. Vitamin D receptors have a grand distribution through tissues, as vascular wall, where they are found in endothelial cells and in smooth muscle cells, which seems to be related with arterial disease pathogenesis. Hypovitaminosis D has demonstrated to have extra-skeletal effects, as those in cardiovascular system, which contributes to cardiovascular disease, including ischemic cardiac disease, peripheral arterial disease and cerebral vascular disease. Vitamin D influences a big number of molecular paths, which are highly relevant to AAAs pathogenesis. The plasmatic 25-hydroxyvitamin D concentration[25](OH) D is an important vitamin D concentration marker in the organism. It has been observed a relation between 25(OH) D values and the AAAs presence, as a relation of these values with AAAs size, which is independent from traditional cardiovascular risk factors. Also the AAAs severity is inversely related with vitamin D concentration, being equally independent from the traditional risk factors. A relation between vitamin D concentration and increased arterial stiffness was also found.[17,18] Vitamin D modulates the expression of several proteins relevant to the arterial wall, including the factor responsible for endothelial cells growth and the MMP9 protein, which is known to be related with collagen and elastin degradation.[18] The vasoprotective effects of vitamin D include cellular proliferation factors inhibition and angiogenesis inhibition, through cellular apoptosis, along with inhibition of pro-inflammatory factors production, by acting on inflammation cascade. Vitamin D is also related with increases in response of T-2 Helper cells, through selective cytokines production. This response is mainly decreased in AAAs formation.[18]

Non-modifiable risk factors

Race: There is an inverse association between Afro-American individuals and AAAs prevalence. It was also found that Hispanics

and Asians have a lower AAA prevalence when compared with Caucasians, after adjusting to all known risk factors. This difference is not significant when comparing Caucasians with Native Americans.[8]

Gender: Men are more likely to develop an AAA, although women have faster aneurysmal growing rates and greater proportional dilatations of AAAs. It is however true that in the female gender there is a minor probability of developing smaller AAAs. Since the AAAs' rupture risk is 3 to 4times higher than the one presented in men, having women a higher proportion of ruptured aneurysms comparatively to those who are effectively treated. Recent analyses showed that the peak aortic wall rupture risk is slightly superior in female gender.[4,8,9] Hannawa et al.,[4] documented that there is a higher mortality rate in the female gender relatively to male gender in what concerns about endovascular/elective treatment and AAAs rupture.[4] The occurrence of AAAs in both genders is also influenced by age. It is proven that the prevalence of this pathology in male individuals, over the age of 50years, is 4-5times higher than that of the individuals of the opposite sex on the same age group. With aging, the tendency tends to invert, which means that the prevalence in men increases up to the age of 80-85years, decreasing after that, while in women there is an increase in prevalence after the 90years.[4] In the aortic walls of men there is an increase of the number of macrophages which secrete the MMP9 protein, promoter of the degradation of elastin and collagen, this being one of the reasons that may prove the higher prevalence of AAA in this gender. In women it is believed to exist a regulatory process, like the estrogen protection, that attenuates the aortic wall degradation, by avoiding the inflammatory cells infiltration in the same. Having regard to this, it is now understandable why the development of AAAs in women occurs 10 to 15years later, since it is only in the postmenopausal period that this protection will be lost.[4] The percentage of elastin in the intima-media complex of AAAs' walls is lower in men, resulting in a greater susceptibility. Interestingly, the percentage of collagen present in the AAAs' walls is lower in the female gender, which contributes to a minor stiffness of the same, which confirms the greater rupture risk in women.[19]

Familiar history: Individuals who have a familiar history of AAAs present an increased infra renal aorta maximum diameter, which determines a duplicated risk of developing an AAA. This risk will be as big as the number of relatives affected by this pathology. Joergensen et al.,[20] reported the increase in mortality and morbidity related with aortic enlargement even when under 3,0cm, condition that was associated with familiar history of AAAs.[20,21] Regarding the degree of relatedness, it is known that first degree relatives have a higher risk of developing an AAA, being patients' biological brothers those who have a greater risk.[21] Considering gender, there are differences in the risk that relatives have of developing an AAA. The risk is bigger if the individual with an AAA is a female. If not only the individual with an AAA but also the relative is a female, the risk of the last one will increase significantly.[20] Having in consideration what was said, it is now understandable the reason why the highest risk of developing an AAA is found in female individuals who are first degree relatives of those who have the aneurysm.[20] On the basis of these mechanisms there is an inherited genetic component, explained by some theories that emerged over time and that try to explain how and when the transmitted mutations arise. A possible explanation for this fact is based on the two genes model, proposed by Tilson et al., that defends the existence of a susceptibility factor in the X chromosome and of an autosomal mutation in another gene. This X chromosome susceptibility might be able to clarify why there is a higher risk in female gender. Larsson et al.,[21] defend the existence of a multigenic model, with mutations in more than one gene; on the other side, Sandford et al.,[22] defend a multifactorial model, in which is stated that there is a probable relation between genetic factors and environmental factors (like smoking habits).[20–22]

Lipoproteinemia: The LRP1 (Low density lipoprotein receptor related protein I) is a protein classified as a receptor of the family of the low density lipoproteins, due to their structural similarity, lying on the surface of many cells, namely the hepatics, pulmonary, cerebral, intestinal and muscular, acting not only as a lipoprotein receptor but also showing several functions of modulation of cellular growth.[23,24] It was demonstrated an association between the LRP1 and the AAAs, independent of the remaining cardiovascular risk factors. This lipoprotein has a wide variety of biological activities, regulating different cellular processes, namely the ones implied in the AAAs formation, existing theories defending that the depletion of this protein, especially on the smooth muscle walls, is related with that formation.[1,23,24] Chan et al.,[24] defends that there is a significant association between a variant of the gene responsible for encoding the LRP1 and the AAAs formation, which suggests that this gene, when changed, can be fundamental to the formation of aneurysms, since such association is also independent of the remaining cardiovascular risk factors.[24] One of the reasons that is thought to be related with the depletion of LRP1 is the over-expression of miR-205, a micro RNA that negatively regulates the LRP1 expression, silencing the gene responsible for its formation. One of the consequences of this depletion may be the lower degradation of MMP9 that occurred when these proteins connected to the LRP1. Another consequence is the cholesterol accumulation in the aortic walls, due to the deregulation of ABCA1 (since the LRP1 works as an agonist of ABCA1), a facilitator of the removal of body cholesterol. This accumulation will lead to an inflammatory process that will damage the arterial walls.[23,24]

Age: Individuals aged under 55years are those that present a lower prevalence of AAAs (1, 83%). This rises till the age group of 70 - 74years (prevalence of 23, 48%), with 4,87% in the age group of 55 – 59years, 13,26% in the group of 60 - 64years and 20,14% in the group of 65 - 69years. Above the age group of 70 - 74years the prevalence decreases, being the prevalence in age group of 75 - 79years of 22,07% and in that of 80 - 84years of 14,28%. It is important to point out that these prevalences are gender independent.[8] If genders are considered, AAAs prevalence in men aged over 50years is 4 - 5 times superior than that of women in the same age group. With aging, the trend tends to be reversed, so that the prevalence in men increases till the age group of 80 - 85years, decreasing from this point, while in women there is an increase in the prevalence after the age of 90years.[4] It should be noted that 29, 5% of the AAAs cases appear in men with smoke history and in the age group of 65 - 75years, being this the group of higher risk.[8] The presented factors contribute to potentiate AAAs' development. When this entity is present, its frequent evaluation is crucial, so that it may be possible to determine the moment when the aneurysm reaches the minimum proposed diameter for surgery, at which the rupture's risk is incremented.

Tri-Dimensional Diagnostic: Computed Tomographic Angiography presents itself as a gold standard practice in the diagnosis of an AAA. However, other techniques have been developed, such as tri-dimensional diagnostic, an improved imaging technique that allows a more precise study if the individuals in risk, being useful for the rupture risk stratification and to make a decision about the right moment to intervene.

Tri-Dimensional Diagnostic: A new in-development technique is the tri-dimensional ultrasound vascular study by speckle tracking, currently applied in echocardiography and that is being adapted to

the AAAs study. Presents itself as an individual prospective and non-invasive study of an AAA rupture risk, that will allow a radial, longitudinal and circumferential analysis of this entity, bridging the limitations presented on the current study ultrasound methods, that evaluate only a sectional plan of the abdominal aorta (bidimensional method). This further study will allow to make a 3D reconstruction of the AAA.[3] It is known that the aneurysmal rupture occurs when there is a mechanical failure of the vascular wall. The irregular geometry of the AAAs, along with the variations in the stiffness of their wall, lead to a heterogeneous distribution of the wall stress and to a disproportional degradation of its structural elements, which conditions local differences related with the tensional strength. The rupture occurs when the wall's stress surpasses the tensional strength of the vascular tissue, which puts in evidence the importance of the knowledge and the study about local mechanical parameters of the aortic wall, as well as other parameters already studied, as the aneurysmal diameter.[3,25] This new method allows a real time study of the mechanical changes that affect the aneurysmal wall, which demonstrates, in a more accurate manner, the tensional differences present in the AAAs, along with the different maximum peak values. Studies done with this technique also allowed concluding that aneurysms present distinct regional inflammatory activity and also local differences in expansion, intraluminal clots formation and calcification, which proves that aneurysms are complex structures, in which biomechanical properties varies in space.[3] Determine the rupture risk from mechanical parameters is more accurate than determine this risk only having in consideration the aneurysmal diameter. The 3D diagnostic technique is capable of detecting the heterogeneity in the local mechanical parameters found in an AAA, presenting itself has a prospective method of study of every type of AAAs, not only the larger ones but also the smaller, that may not have the agreed size for analysis and intervention but that present rupture risk.[3]Data acquisition takes 15minutes and the off-line processing about 30minutes. It has a similar limitation as that found in the conventional 2D ultrasound exam, which is the loss of information when the ultrasound spread is prevented.[3,25] It is important to emphasize that the installation of this technique is an asset for the patient, considering that, when compared with the gold standard (CTA), it cancels the radiation factor and minimizes the exam costs.[25] Considering all the presented risk associations that may lead to AAAs formation, scanning the individuals that present them assumes a major role, not only because severe complications may come from the AAAs that aren't diagnosed nor consequently treated, but also because there are an amount of prognostic and prevision studies that can be done and that may improve after-surgery results[4] (Figure 2).

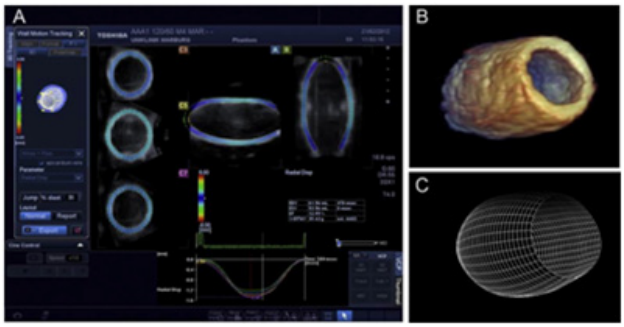

Figure 2 A) *Speckle Tracking*

B) 3D Image;

C) Silicon vascular model reconstruction.[3]

Complications and their prevision: Intervened aneurysms can be accompanied by risks and complications, being death the major complication, the prevention of this complication depends ontwo factors: an attempted diagnosis and a consequent repair with good long-term results. This reparation is advised when the AAA's rupture risk surpasses surgical risks. It is essential to evaluate every patients proposed to intervention, not only to determine the post-intervention prognosis, but also to search possible morbidities related to surgical interventions.[26]

Prevision of renal insufficiency incidence after treatment: Several studies indicate that the AAAs' classical surgery has a mortality rate of about 5% and a 1% to 6% prevalence of development of postoperative renal failure, being the post-EVAR complications less frequent.[27] Given the increased death' risk, within 30days, that the development of renal failure entails, the identification of the individuals at risk of developing it becomes very important, existing a set of predictive models and risk factors that justify the likelihood of developing this condition,[27] Grant et al.,[27] mentioned some independent preoperative risk factors that condition an increased risk of developing postoperative renal failure: smoking habits, often presented in patients with AAAs, with development of atherosclerotic disease and concomitant cardiovascular and renal disease; medication used to treat cardiovascular diseases, in individuals with AAAs, which can increase the risk of renal failure; clamping of the suprarenal aorta, during the treatment of the juxtarenal aneurysms, which could lead to embolism and renal hypoperfusion, since the flow is conditioned.[27] Grant et al.,[27] and Hoshima et al.,[28] shown that the levels of creatinine or the estimated glomerular filtration rate are two important indicators for the evaluation of the preoperative renal function. Creatinine values higher than 150μmol/L have been shown to be associated with increases in the risk of AAAs' postoperative mortality. For the assessment of the glomerular filtration rate it is often used the RIFLE classification (Risk, Injury, Failure, Loss of kidney function and End-stage kidney disease), useful to evaluate acute renal injuries.[27,28] The identification of the higher risk individuals becomes important, as they will be the ones that should be followed by a health technician, in preoperative context, in order to define the strategies of renal optimization that improve the post-surgery prognosis, namely the reduction of exposure to nephrotoxic agents on the day of surgery, such as the ACEI and the antagonists of the angiotensin receptors. It is also pertinent to avoid the use of contrast in preoperative exams. This optimization can lead to a reduction of the preoperative renal complications as well as of the potential mortality risk that is associated.[27] It is important to point out that individuals older than 75years have a decreased renal vascular reserve, which leads to a smaller ability to respond to stress situations, as is a major surgery.[27] Another interesting relation to be established is relatively to the symptomatic and asymptomatic AAAs. The symptomatic individuals may be subjected to high risk surgeries that would be inappropriate if they were asymptomatic. These individuals can also be intervened more urgently than the asymptomatic ones, without proper preoperative renal optimization. It is also necessary to consider the fact that if they are symptomatic, that can be due to inflammatory processes or degradation of luminal thrombi, which increases the risk of renal complications after surgery.[27] The presence of respiratory disease is also associated with a higher risk of renal complications. The chronic renal failure rates are higher in patients with chronic respiratory disease, being equally true that the pulmonary disease is a predictive factor of renal failure in the post-surgery. Hypertension is also presented as a predictive factor, since it is one of the major risk factors for the development of renal failure.[27]

Prognosis in patients with renal disease: Chronic renal disease presents a significant number of cardiovascular co-morbidities and a reduced long-term chirurgical survival rate, proportional to the

severity of the disease. The higher this severity, the higher will be operatory mortality rates and associated complications. The operatory mortality and morbidity reflects the effect of the moderate and severe chronic renal disease, being this independent of other operatory risk factors.[26] Patients that go under classical AAA repair surgery present increased mortality and morbidity rates comparatively to EVAR. The risk in the classical surgeries increase with the increasing in the severity of the renal disease, has already said. For example, patients with mild chronic renal disease have operatory mortality rates of 8,4% comparatively to 9,9% of patients of moderate and severe chronic renal disease. However, these values are low if we have in consideration the mortality rate of 42% found by Katz *et al* in his study, related with the higher risk group (renal disease in advanced stages). The analysis of other studies suggests that the negative effect of renal dysfunction on the operatory mortality and morbidity rates after classical repair maintains relatively unchanged although the advances made on the medical care over time.[26] Patel et al.,[26] affirms that high risk patients for classical surgery were proposed to EVAR reparation. These patients had 30-days mortality rates of 9% and a long-term survival of 40%, after 4years, which allows to verify that there isn't a beneficial contribution of this technique in the decreasing of the mortality rates of these patients, when compared to classical surgery.[26] The moderate and specially the severe chronic renal disease are of the maximum importance on the decision about the treatment, this being the classical surgery or EVAR. High risk and symptomatic patients or those with rapid growth AAAs must be considered for reparation after a risk-benefit analysis.[26]

Prognostic in patients with infrarenal and juxtarenal aneurysms: The post-operatory prognostic differs according to the aneurysm location. Individuals that present juxtarenal AAAs have, normally, more complications than the individuals with infrarenal AAAs, this because the first ones need to clamp the suprarenal aorta, which might be accompanied by extensive dissections that will cause ischemic lesions in several organs and reperfusion injuries, along with possible embolization of atherosclerotic material to the renal arteries. Temporary renal ischemia presents itself as the most influent factor on the weak post-operatory results. It is important to refer that juxtarenal AAAs are entities that are characterized by the involvement of the infrarenal abdominal aorta segment, with superior extent that might also involve the renal arteries inferior margins.[27,28] Grant et al.,[27] proved that the renal insufficiency incidence is superior in individuals submitted to the juxtarenal AAAs surgery. In these individuals, the incidence rate of renal insufficiency ranges between 18-41% and the new hemodialysis rate after-surgery between 4-7% [27–30] (Figure 3 & 4).

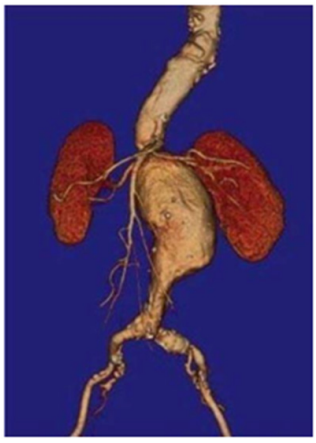

Figure 3 Juxtarenal aneurysm.[29]

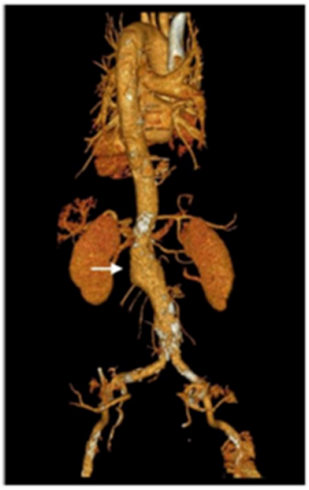

Figure 4 Infrarenal aneurysm.[30]

Conclusion

The AAAs feature a group of risk factors and associations that enhance its formation and/or growth. Many of them are similar to atherosclerotic disease's risk factors, although lately it has been referred that the atherosclerotic and aneurysmal diseases have different etiologies, namely the protective effect that the diabetes presents on the formation of AAA, something that does not happen in atherosclerotic disease. New diagnosis methods have been developed, being tri-dimensional diagnosis, by speckle tracking technique, the most recent bet, which bridges the limitation presented in the bi-dimensional diagnosis techniques, being possible to do the study of the biomechanical parameters of an AAA and to obtain more detailed information about the same. The prediction of eventual complications appears as an aspect of extreme importance, especially in the high risk groups. The spirometric studies and the vascular eco-doppler studies are pertinent exams in the prevision of mortality and morbidity, the information obtained should be framed in the therapeutic decision, evaluating the pros and cons of surgery, to determinate a chirurgical risk factor, face the eventual AAA's rupture risk. It is important to mention the work developed by the health technicians in patients with AAAs, having an essential role in the diagnosis and study of the biomechanical proprieties of this entity, but also in the prognosis and predictive evaluation of complications.

Acknowledgments

None.

Conflicts of interest

Author declares there are no conflicts of interest.

Funding

None.

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