

Possible Clinical Benefits of Cardio-Ankle Vascular Index Measurement in Urological Diseases

Emre Bulbul¹, Murat Oguz Ozilhan², Ali Sezer³, Mustafa Yetisen⁴, Fahri Yavuz Ilki⁵

¹Department of Urology, Trabzon Vakfikebir State Hospital, Trabzon, Turkey; ²Department of Cardiology, Ankara City Hospital, Ankara, Turkey;

³Department of Urology, Konya City Hospital, Konya, Turkey; ⁴Department of Cardiology, Erzurum Oltu State Hospital, Erzurum, Turkey;

⁵Department of Urology, Gulhane Training and Research Hospital, Ankara, Turkey

Correspondence: Emre Bulbul, Department of Urology, Trabzon Vakfikebir State Hospital, Trabzon, 61400, Turkey, Tel +90 536 390 06 11, Fax +90 462 841 35 50, Email dr.emre5567@gmail.com

Abstract: Many factors are considered to affect vascular physiology. It is known that one of the reasons for many diseases is a pathology at the microvascular level. Therefore, the relationship between endothelial dysfunction and many diseases is currently being investigated. The clinical evaluations of arterial stiffness have made it possible to perform necessary risk assessment regarding cardiovascular diseases. In this way, protective measures can be taken against microvascular pathologies in many organs. In this paper, we present a review of studies investigating the relationship between urological conditions and the cardio-ankle vascular index (CAVI), a marker of arterial stiffness. As with erectile dysfunction, some studies have shown that conditions such as lower urinary tract symptoms, overactive bladder, and chronic kidney disease are also associated with an elevated CAVI. The association of erectile dysfunction and chronic kidney disease with vascular pathologies has been clearly demonstrated. In addition, lower urinary tract symptoms may be the first sign of silent vascular dysfunction. Assessing arterial stiffness with CAVI can help prevent future cardiovascular events in these patients.

Keywords: arterial stiffness, cardio-ankle vascular index, cardiovascular disease, erectile dysfunction, lower urinary tract symptoms, overactive bladder

Plain Language Summary

- Erectile dysfunction and cardiovascular disease can be seen together with systemic vascular pathologies.
- The cardio-ankle vascular index is higher in patients with erectile dysfunction and overactive bladder than in healthy people.
- The risk of cardiovascular disease in patients with chronic kidney disease is much higher than in the general population.
- Lower urinary tract symptoms are associated with increased arterial stiffness.

Introduction

The human body has a unique vascular anatomy, and many factors are considered to affect vascular physiology. It is known that one of the components of many diseases is a pathology at the microvascular level.^{1–3} Therefore, the relationship between endothelial dysfunction and many diseases is currently being investigated.^{4,5}

Erectile dysfunction (ED) may be the first sign of cardiovascular disease (CVD) risk. The luminal diameter of the penile artery is smaller than that of the coronary arteries, and the erection mechanism is affected earlier by microvascular pathologies. Therefore, ED may be a marker of systemic microvascular pathologies. Furthermore, ED and CVD can be seen together with systemic vascular pathologies.⁶ The European Association of Urology guidelines recommend that patients with ED be evaluated in detail regarding CVD risk.⁷ In the literature, it has been stated that urological diseases, such as overactive bladder (OAB) and benign prostatic enlargement (BPE) are also associated with vascular pathologies.^{8,9} It has been noted that as the local atherosclerosis of the prostatic artery increases, the prostate volume also increases. It has also been reported that oxidative stress markers are effective in the mechanism of BPE.¹⁰ CVD risk

Table 1 The Principal Finding of Each Paragraph

<p>Introduction</p> <ol style="list-style-type: none"> Paragraph: Microvascular pathology Paragraph: Urological diseases and cardiovascular disease risk Paragraph: Endothelial dysfunction markers Paragraph: Arterial stiffness: PWV and CAVI Paragraph: Urological conditions and CAVI
<p>Association between erectile dysfunction and cardio-ankle vascular index</p> <ol style="list-style-type: none"> Paragraph: Erectile dysfunction and cardiovascular disease risk Paragraph: Cardiovascular disease risk and arterial stiffness Paragraph: Erectile dysfunction and arterial stiffness
<p>Lower urinary tract symptoms and arterial stiffness</p> <ol style="list-style-type: none"> Paragraph: Lower urinary tract symptoms and cardiovascular disease Paragraph: Lower urinary tract symptoms and arterial stiffness
<p>Cardio ankle-vascular index in overactive bladder</p> <ol style="list-style-type: none"> Paragraph: Overactive bladder and CAVI Paragraph: Arterial stiffness and tissue healing Paragraph: Antimuscarinic treatment and CAVI
<p>Cardio ankle-vascular index and renal function</p> <ol style="list-style-type: none"> Paragraph: Renal dysfunction and arterial stiffness Paragraph: Renal dysfunction and CAVI Paragraph: Chronic kidney disease and cardiovascular disease

Abbreviations: PWV, pulse wave velocity; CAVI, cardio-ankle vascular index.

has been found to be higher in patients with OAB. In addition, bladder neck perfusion is low on Doppler ultrasonography. As the severity of OAB increases, the Framingham risk score also increases.¹¹

In the literature, it has been reported that endothelial dysfunction is associated with many urological diseases, including ED, OAB, and BPE.^{12–14} In clinical studies, many markers, such as brachial flow-mediated dilatation, pulse wave velocity (PWV), cardio-ankle vascular index (CAVI), and intima-media thickness have been used to measure endothelial dysfunction.^{15–18}

Vascular age can be determined non-invasively by measuring arterial stiffness. PWV and CAVI are indicators of arterial stiffness.^{19,20} Carotid-femoral measurement is generally used to evaluate arterial stiffness with PWV. Age, rheumatological diseases and blood pressure are parameters that affect PWV independently.²¹ The CAVI can also assess arterial stiffness independent of blood pressure.²²

With the clinical evaluations of arterial stiffness, necessary risk assessment can be made regarding cardiovascular diseases (Table 1). In this way, protective measures can be taken against microvascular pathologies in many organs. In this paper, we present a review of studies investigating the relationship between urological conditions and CAVI, a marker of arterial stiffness.

Association Between Erectile Dysfunction and Cardio-Ankle Vascular Index

The relationship between ED and CVD has been known for a long time. Patients with ED, especially those without a history of CVD may be unaware of the possibility of developing associated cardiological pathologies in future. In patients with ED, urologists usually perform a routine metabolic evaluation (body mass index, fasting blood glucose, triglyceride, cholesterol, etc.). However, other than routine blood pressure measurement, no assessment is made regarding vascular physiology. Only local vascular functions can be evaluated with penile Doppler ultrasonography.

Investigating vascular dysfunction, an essential component of ED pathophysiology, in the whole body can provide significant benefits. Therefore, there is a need for non-invasive markers to show vascular dysfunction.

The patients can be evaluated regarding CVD risk using arterial stiffness measurement. Hayashi et al developed the β formula for this assessment. In their study including 18 cadavers, the authors comprehensively measured vascular wall stiffness and reported that arterial stiffness increased with age. They also stated that increased arterial stiffness played a role in the development of cerebrovascular disease.²³ Later, PWV was used as a marker for measuring arterial stiffness. However, the blood pressure value at the time of measurement affects the PWV value.²⁴ Therefore, in 2006, Shirai et al developed a new method for the measurement of arterial stiffness. In a study they conducted with hemodialysis patients, the authors reported that CAVI was higher in those with signs of atherosclerotic disease than those patients without these signs. In addition, they noted that the correlation of CAVI with blood pressure was weaker compared to that of PWV with blood pressure.²⁵

Arterial stiffness assessment in patients with ED can help identify those at risk of CVD.²⁶ In a screening study of healthy Japanese men, Imai et al reported that arterial stiffness was higher among those with ED.²⁷ In another study, Demirelli et al determined that arterial stiffness increased as ED severity increased.²⁸ The literature also suggests that phosphodiesterase-5 inhibitor therapy reduces arterial stiffness in patients with ED.^{29,30} Bulbul et al compared CAVI between the patients with ED and healthy men and found a significantly higher value in the former. In addition, the authors reported that as the severity of ED increased, CAVI increased proportionally.³¹ In a 2016 study, Motil et al used a device they developed for low-intensity shockwave therapy in the treatment of vasculogenic ED. In that study, patients with vasculogenic ED were successfully identified by measuring CAVI.³²

Lower Urinary Tract Symptoms and Arterial Stiffness

While the cause of lower urinary tract symptoms (LUTS) is mostly BPE in men, the etiology is not yet clear in women. The development of LUTS increases with age and has a multifactorial origin. In the literature, it has been suggested that LUTS may be associated with chronic ischemia. Increased reactive oxygen radicals in the bladder due to chronic ischemia may damage the bladder wall, causing the detrusor to work excessively or be underactive.³³ It has been reported that there is a relationship between nocturia and CVD. Tikkinen et al found that the incidence of nocturia increased threefold in patients with CVD.³⁴

The role of vascular dysfunction in the etiology of LUTS is still being investigated. Uzun et al evaluated arterial stiffness and carotid intima-media thickness in male and female patients with LUTS and detected higher values in the patient group compared to the control group. The authors concluded that LUTS was associated with vascular dysfunction in both men and women.³⁵ Yabe et al performed robot-assisted laparoscopic radical prostatectomy on 80 male patients with prostate cancer. They divided these patients into the atherosclerotic group (CAVI ≥ 9.0) and the control group (CAVI < 9.0). The voiding and international prostate symptom scores were significantly higher in the atherosclerosis group than in the control group at the postoperative third month. There was no significant difference between the two groups regarding the voiding and international prostate symptom scores at six months, but the maximum flow rate was significantly lower in the atherosclerosis group. It was concluded that the increase in arterial stiffness determined by CAVI adversely affected the reduction of LUTS in the early period after robot-assisted laparoscopic radical prostatectomy.³⁶ In a study conducted with 12 healthy men in 2011, 4 mg of doxazosin, an alpha 1 adrenergic receptor blocker, was administered to the patients, and it was determined that CAVI significantly decreased after doxazosin administration compared to the pre-treatment value.³⁷

Cardio Ankle-Vascular Index in Overactive Bladder

It has been stated that OAB occurs due to sympathetic autonomic dysfunction. Shimizu et al evaluated patients with OAB or an idiopathic large post-void residual volume (>100 mL) using CAVI and carotid intima-media thickness measurements. The CAVI values were significantly higher in the OAB group than in the control group. However, no significant difference was observed between the two groups regarding the carotid intima-media thickness. Furthermore, there was no significant difference in vascular function between the patients with a large post-void residual volume and the control

group. The authors suggested that a large post-void residual volume might be associated with local factors, and the autonomic dysfunction of the bladder might be related to systemic vascular pathologies.³⁸

Increased arterial stiffness may affect tissue healing by causing vascular dysfunction. Koguchi et al cross-sectionally examined OAB symptoms in patients who underwent robot-assisted laparoscopic radical prostatectomy for prostate cancer. They divided the patients into two groups according to their arterial stiffness values as the atherosclerotic group (CAVI \geq 9.0) and the control group (CAVI $<$ 9.0). At the postoperative third month, de novo OAB development was higher in the atherosclerotic group than in the control group. At nine and 12 months, voided volume was significantly lower in the atherosclerotic group than in the control group. The increase in arterial stiffness adversely affected the improvement of storage symptoms after robot-assisted laparoscopic radical prostatectomy.³⁹

Care should be taken regarding the cardiac side effects of antimuscarinic and sympathomimetic agents used in the treatment of OAB. The risks of triggering antimuscarinic autonomic dysfunction and increasing the pulse rate of sympathomimetics should be considered. Hsiao et al investigated the cardiovascular effects of 25 mg mirabegron (beta-3 sympathomimetic) and 5 mg solifenacin in women. After 12 weeks of treatment, blood pressure significantly decreased in the patients that received solifenacin, and the pulse rate significantly increased in those treated with mirabegron. There was no significant difference in CAVI between the two groups after treatment.⁴⁰ In a study evaluating female patients with OAB, the effects of antimuscarinic drugs were compared. After 12 weeks of treatment, CAVI was significantly reduced in patients that received 5 mg tolterodine. However, CAVI did not significantly differ after 12 weeks of 4 mg solifenacin treatment compared to the pre-treatment values. It was concluded that in the treatment of OAB caused by sympathetic autonomic dysfunction, the use of tolterodine was beneficial to help recover vascular function.⁴¹

Cardio Ankle-Vascular Index and Renal Function

Albuminuria is one of the early indicators of renal dysfunction. The albumin/creatinine ratio in urine is used as a marker for albuminuria. If the albumin/creatinine ratio in the urine is \geq 30 mg/g, it is considered to indicate pathological albuminuria. Jiang et al reported that arterial stiffness was independently associated with pathological albuminuria.⁴² Latif et al investigated factors affecting mortality in 5827 hemodialysis patients from six countries and found that high serum uric acid levels ($>$ 8.2 mg/dl) reduced mortality due to CVD.⁴³ Harada et al examined the serum uric acid and CAVI values of 48 hemodialysis patients without CVD. Interestingly, they found an inverse correlation between these two parameters.⁴⁴

The increase in arterial stiffness measured by CAVI is a marker of deterioration in renal function.⁴⁵ The use of intravenous contrast agents in imaging can lead to acute deterioration in renal function. Sahinkus et al performed the CAVI evaluation of 126 patients who underwent contrast-enhanced coronary angiography. CAVI was determined to be significantly higher in the patients who developed contrast-induced nephropathy compared to those without nephropathy.⁴⁶

The risk of CVD and mortality due to CVD are much higher in patients with chronic kidney disease (CKD) than in the general population.⁴⁷ Vascular changes in patients with CKD cause an increase in arterial stiffness.⁴⁸ Blacher et al evaluated the arterial stiffness of 241 patients with end-stage CKD. They reported that increased arterial stiffness resulted in an increase in overall and CVD-related mortality.⁴⁹ In another study, arterial stiffness was found to increase as CKD increased.⁵⁰ Ishimura et al measured CAVI in 107 kidney transplant patients and detected significantly lower CAVI values in those that received everolimus compared to the remaining patients. The authors suggested that everolimus treatment might have a protective effect against the development of atherosclerosis after kidney transplantation.⁵¹

Conclusions

In addition to ED, elevated CAVI values have been reported in studies evaluating patients with LUTS, OAB, and CKD have been. The association of ED and CKD with vascular pathologies is well established. LUTS may also be the first sign of silent vascular dysfunction. Assessing arterial stiffness with CAVI can help prevent future cardiovascular events in these patients.

Disclosure

The authors report no conflicts of interest concerning this work.

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