TÜRKİYE VE DÜNYADA KALP VE DAMAR CERRAHİSİ

Editör: Dr.Öğr.Üyesi Volkan Burak TABAN

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İÇİNDEKİLER

CHRONIC VENOUS INSUFFICIENCY

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1. INTRODUCTION

Lower extremity venous insufficiency and the varicose veins that arise as a result are significant diseases that are quite common in society, impair quality of life, and can present with serious complications in some cases. Chronic venous disease is often an undiagnosed condition that progressively reduces the patient's quality of life while also placing an increasing burden on healthcare resources. Chronic venous disease includes telangiectasia, reticular veins, varicose veins, chronic venous insufficiency (CVI) and chronic venous hypertension. Symptoms of CVI include pain, skin discolouration, oedema and ulceration. More specifically, CVI is a condition that affects the lower extremity venous system and involves complex venous pathology.

2. ANATOMY

The lower extremity venous system is divided into the superficial venous system, deep venous system, and perforating venous system. Bicuspid valves located throughout the venous system regulate hydrostatic pressure by ensuring unidirectional blood flow. In the lower extremities, the valves divide the hydrostatic column of blood, enabling flow from the surface to the deep tissues and from caudal to cephalic..[1]

The superficial venous system consists of the Great Saphenous Vein (GSV) and the Small Saphenous Vein (SSV). The VSM starts at the dorsum of the foot and runs along the medial aspect of the thigh, draining into the femoral vein at the saphenofemoral junction. During its course in the saphenous compartment, it is in contact with arterial and nerve structures. This vein, which runs within the saphenous fascia on the medial aspect of the thigh, is one of the most important superficial venous structures in the development of venous insufficiency. The VSP originates from the outer part of the arcus venosus dorsalis pedis on the upper surface of the foot and extends posteriorly and laterally, passing over the lateral malleolus. It runs alongside the sural nerve throughout its course. Protection of this nerve is important during thermal ablation.[2, 3]

The deep venous system runs alongside the arteries within the muscular fascia. With the exception of the femoral vein and popliteal vein, they are generally present in pairs. They have numerous collateral connections with the superficial veins.[1]

Perforating veins are effective in regulating venous hypertension by connecting deep and superficial veins. They are named Dodd, Boyd, and Cockett according to their location. The Cockett perforating veins located medially in the calf region are clinically significant and are responsible for perforating venous insufficiency.[2, 3]

3. EPIDEMIOLOGY

CVI affects approximately 25-40 per cent of the adult population. It is seen in 30-50 per cent of women and 10-30 per cent of men. [4] It affects socio-economic status in communities where it is prevalent. The prevalence rate in advanced stages is 5%. Active or healed ulcers occur in 1-2%, telangiectasias in 80%, and varicose veins in 20-64% of cases.[5]

4. ETİOLOGY

Chronic venous insufficiency (CVI) is a condition resulting from the functional insufficiency of the venous system's valve structures and the deterioration of the venous walls. This pathology leads to increased venous pressure in the lower extremities and impaired venous return. Etiological factors include genetic predisposition, venous hypertension, history of deep vein thrombosis (DVT), prolonged immobilisation, and age-related loss of venous elasticity. Environmental factors such as pregnancy, obesity, and prolonged standing also increase the risk of CVI.

4.1. Primary CVİ

Primary chronic venous insufficiency is a chronic progressive disease that leads to pathological reflux by causing abnormal dilation and weakness in the vein wall or venous valves. It is frequently seen in the saphenous veins and is known as trunkal (trunk) insufficiency. Primary CVI is the result of various genetic and environmental factors. The majority of individuals with insufficiency carry certain polymorphisms or genetic variants that are partly responsible for the development of the disease.[6]

4.2. Secondary CVİ

Secondary CVI refers to the development of venous insufficiency as a result of a previous event. This condition is divided into two main groups: secondary intravenous causes (direct involvement of the vein wall and valves) and secondary extra-venous causes (local or systemic venous haemodynamic imbalances). Secondary extra-venous causes include central venous hypertension, extrinsic compression due to a tumour mass, arterial compression in May-Thurner syndrome, and muscle pump dysfunction.[6]

4.3. Congenital CVİ

Genetic factors play a significant role in the pathogenesis of chronic venous insufficiency (CVI), and family history is a strong risk factor. Klippel-Trenaunay syndrome, Parkes Weber syndrome, Lymphedema Distichiasis Syndrome, CADASIL, and Ehlers-Danlos Syndrome are congenital disorders that include venous hypertension and venous ulcers.[6, 7]

5. PHYSIOPATHOLOGY

The causes of chronic venous insufficiency are increased venous pressure (venous hypertension) and the effects of this condition on small vessels. In healthy venous circulation, blood flows towards the heart with the help of muscles and venous valves. However, in cases of valve insufficiency or weakening of the vein walls, some blood flows backwards (retrogradely), increasing the pressure within the vein. This leads to increased venous stasis in the legs and chronic venous hypertension.[8]

Venous hypertension increases mechanical stress within the vessel and leads to the development of endothelial dysfunction. Endothelial cells initiate an inflammatory cycle by promoting the release of inflammatory mediators (e.g., interleukin-1 and tumour necrosis factor-alpha). This inflammatory process causes the venous walls to dilate and further impairs valve function. In addition, venous hypertension triggers capillary leakage and oedema formation by increasing capillary hydrostatic pressure.[8]

Venous hypertension is the primary cause of tissue damage in chronic venous insufficiency. Increased capillary pressure causes fluid to leak out of the capillaries, leading to the accumulation of extravascular fluid (oedema). The increased permeability of capillary walls allows plasma proteins and inflammatory cells to pass into the tissue. This leads to hypoxia, tissue inflammation, fibrosis, and ultimately tissue damage (ulceration).[9]

Venous hypertension impairs the small vessel system between arterioles and venules, thereby preventing oxygen from reaching cells. This condition leads to tissue hypoxia (oxygen deprivation) as a result of venous stasis. This oxygen deficiency causes cells called fibroblasts to become more active, leading to changes such as tightening and hardening (fibrosis) in the subcutaneous tissue. In addition, symptoms such as pigmentation changes, hemosiderin accumulation resulting from the breakdown of blood cells, and hardening and thinning of the skin are also effects of these processes.[9]

Insufficiency in the deep venous system is rarely seen and generally occurs secondary to the superficial system, improving with treatment of the superficial system. Deep vein thrombosis (DVT), which is the blockage of deep veins, reduces blood flow by increasing pressure and can damage the valves. This condition can further worsen the insufficiency. This condition cannot be resolved with treatment of the superficial system. Impaired muscle pump function manifests as reflux and increased pressure. After prolonged standing or walking, no significant difference in venous pressure is noticeable because there is an inability to remove blood.[8, 9]

6. CLINICAL FINDINGS AND SYMPTOMS

Chronic venous insufficiency is a condition that significantly reduces patients' quality of life and also causes concerns about their appearance. The most common symptoms of this condition include pain in the legs, a feeling of tiredness, tingling, cramps, a feeling of tiredness throughout the night, and swelling. These symptoms negatively impact patients' daily lives and are important for distinguishing the condition from other illnesses. The pain usually increases as the day progresses and worsens in situations such as hot weather and prolonged standing. Patients may also experience other symptoms such as paraesthesia, night cramps, and fatigue. These symptoms may be alleviated by rest and elevating the legs. Some patients may only seek medical attention due to appearance-related issues.[5, 7, 10]

Chronic venous insufficiency (CVI) can manifest itself in the clinic with a wide range of findings, from simple telangiectasia to chronic and non-healing active venous ulcers. Telangiectasia, the presence of reticular or varicose veins, is one of the most common physical findings in CVI. Varicose veins may be asymptomatic, but in some cases, they can lead to complications such as bleeding and even death. They can also cause inflammation, such as thrombosis, painful phlebitis, and cellulitis in superficial veins. Patients with chronic venous insufficiency may experience functional limitations such as pain during walking

(venous claudication) and difficulty climbing stairs. In more advanced stages of the disease, the severity and frequency of symptoms increase, particularly in parallel with the presence of reflux in the superficial or deep venous system and an increase in the diameter of the great saphenous vein. This significantly reduces patients' quality of life.[5, 7, 10]

7. CLASSIFICATION

Chronic venous insufficiency is a clinically variable disease, and accurate staging is critical for effective treatment planning. Classification systems such as CEAP, VCSS, and Villalta provide standardisation in both clinical practice and scientific research. However, as each system has its own advantages and limitations, combined use can further enhance clinical assessment.

The CEAP classification was developed in 1994 by the International Union of Phlebology and has been used to improve the assessment and definition of Chronic Venous Insufficiency (CVI) and to make treatment outcomes comparable. The main objective of this classification is to combine different terminologies and criteria in the definition and assessment of chronic venous diseases. This aims to achieve more consistent and objective results in clinical practice and research. CEAP provides a simple system for monitoring the course of the disease and its future development in the clinic. However, this classification has some shortcomings, as it cannot address conditions such as venous neuropathy, venous claudication, the coexistence of arterial and venous diseases, obesity, and coronary phlebectasia. Despite these drawbacks, it is currently the most frequently used classification method by many specialists.[11]

The CEAP classification has successfully categorised chronic venous diseases and has been useful in comparisons across different centres and studies; however, it has been inadequate in comparing and evaluating different treatments performed in studies or the same treatments performed in different studies. Therefore, venous severity scoring systems have been developed with a new classification.

Venous Clinical Severity Score (VCSS): This score is a system used to objectively assess the severity of the clinical findings of venous insufficiency.[12]

Venous Segmental Disease Score: This score, which incorporates anatomical and pathophysiological components, provides a segment-based assessment of venous insufficiency.[12]

Villalta-Prandoni Score: This score, used particularly in the assessment of post-thrombotic syndrome, is widely preferred for measuring the severity of the disease and monitoring clinical progress.[13]

Table I. CEAP Classification

Clinical	
C0	No visible or palpable signs of venous disease
C1	Telangiectases or reticular veins
C2	Varicose veins
C3	Edema
C4a	Pigmentation or eczema
C4b	Lipodermatosclerosis or atrophie blanche
C5	Healed venous ulcer
C6	Active venous ulcer
Etiological	
Ec	Congenital
Ep	Primary
Es	Secondary (post-thrombotic)
En	No venous cause is identified.
Anatomical	
As	Superficial veins
Ap	Perforator veins
Ad	Deep veins
An	No venous location was identified.
Pathophysiological	
Pr	Reflux
Po	Obstruction
Pr, o	Reflux and obstruction
Pn	No venous pathophysiology identifiable

8. DIAGNOSIS

The clinical management of chronic venous insufficiency depends on the accurate and timely diagnosis of the disease. Diagnostic methods consist of clinical evaluation, non-invasive imaging, invasive radiological techniques, and specific functional tests. The correct diagnosis of CVI affects treatment planning and aims to improve patients' quality of life. With a thorough physical examination, a diagnosis can be made without the need for additional tests, and treatment can be started without delay.

The patient's history is critical in the diagnosis of CVI. The patient should be questioned about any history of deep vein thrombosis, thrombophlebitis, pregnancy, prolonged standing, obesity, hormonal treatment, and family history. In particular, the onset, duration, severity, and impact of symptoms on daily life should be assessed.

The physical examination should be performed with the patient standing. Visible varicose veins, oedema, pigmentation, lipodermatosclerosis and skin ulcers are examined. In addition, classic examination methods such as the Trendelenburg and Perthes tests can be used to assess perforating vein insufficiency and saphenofemoral reflux.

Varicose veins and skin changes should be examined during a physical examination performed while standing. Venous collateral development observed in the abdominal and pubic regions is typical of iliac vein occlusion, and every patient must be assessed from this perspective. Furthermore, pitting oedema or resistant oedema during the examination also indicates high hydrostatic pressure.

Duplex ultrasound is one of the most common and useful methods used in the diagnosis of venous insufficiency. It provides information about the anatomy of the lower extremity venous system, including superficial, deep and perforating veins, and the pattern of venous flow. It indicates obstruction or deep vein thrombosis (DVT). It provides information about the presence of reflux, its duration, speed and volume, if any. It can provide anatomical information to guide treatment by determining whether venous valves are functioning and identifying leaks in which areas. However, it may sometimes be inadequate in the assessment of the deep venous system due to pelvic veins and anatomical differences.[14]

Reflux durations in the diagnosis of venous insufficiency:

- Superficial veins >0.5 s,
- Perforating veins >0.35 s,
- If it is >1 s in deep veins, it is pathological.

CT venography enables rapid examination of the venous system using contrast medium and is generally effective in detecting acute thrombosis, compression syndromes, and venous obstruction caused by malignancy. MR venography, on the other hand, is particularly advantageous in young and pregnant patients as it does not use ionising radiation. Additionally, it offers techniques such as time-of-flight, which enable the visualisation of venous structures without the need for contrast agents.

Venography (phlebography): Duplex ultrasound has significantly reduced the use of venography. The procedure is performed in two ways: descending venography and ascending venography. Descending venography is performed by cannulation of the distal iliac vein. It

reveals vascular lumen pathologies, impaired valve function, and reflux. Ascending venography, on the other hand, detects occluded veins, collaterals, and perforators.

Plethysmography is a system that works based on the absorption of light in the subcutaneous veins. It provides information about reflux and obstruction by showing calf muscle pump dysfunction and volume changes..[15]

9. TREATMENT

9.1. Lifestyle Changes

Regular exercise is effective in alleviating varicose vein symptoms. Lifestyle changes can also help prevent the development of varicose veins. Athletes who frequently use their calf muscles, for example, have a lower risk of developing varicose veins despite standing for long periods. Conversely, sitting in the same position for long periods or remaining inactive creates excessive pressure in the leg and foot veins and increases the risk of varicose veins. Simple lifestyle changes recommended for patients include elevating the legs and feet, avoiding inactivity, and avoiding being overweight. These practices are beneficial in adhering to treatment and reducing symptoms.[15]

9.2. Compression Therapies

Compression therapy plays a crucial role in the treatment of conditions such as chronic venous insufficiency (CVI) and varicose veins. This treatment improves circulation by reducing venous stasis in the lower extremities and alleviates the patient's symptoms. Compression bandages, Velcro devices, compression pumps, intermittent pneumatic compression devices, and elastic compression stockings are used for compression therapy. When deciding which treatment method to choose, the patient's CEAP classification, personal circumstances, and economic situation are taken into account. Elastic compression stockings are preferred for patients with mild symptoms or those at high risk of surgical intervention. These stockings are classified according to different pressure levels based on their use: Class A: 10-14 mmHg (very low pressure), Class I: 15-21 mmHg (light pressure), Class II: 25-32 mmHg (medium pressure), Class III: 34-46 mmHg (high pressure), Class IV: >49 mmHg (very high pressure). The appropriate pressure level for each patient should be determined based on individual needs. In patients with skin lesions (rash, open areas), stocking use should begin after the lesion has healed. In all patients recommended for compression stockings, the presence or absence of arterial insufficiency should be considered. Compression stockings can be used safely in patients with an Ankle-Brachial Index (ABI) >0.5 and ankle pressure >60 mmHg. Stockings should be put on in the morning, before getting out of bed, when the veins are pulsating and the pressure is at its lowest, and after the legs have been elevated for 5-10 minutes.

9.3. Medication

Herbal or synthetic drugs are used in the treatment of CVI. These drugs are generally venoactive. Venoactive drugs exert their effects by alleviating the oedema and symptoms of CVI, primarily through their antioxidant mechanism. Such drugs cannot completely cure the existing disease but provide symptomatic improvement by alleviating the symptoms.

Venous active drugs are a group of medications consisting of different types used to alleviate the symptoms of venous insufficiency. These drugs cannot provide a complete cure for the

disease but alleviate the patient's symptoms. They reduce symptoms such as oedema, pain, discomfort, heaviness, and muscle cramps. Thanks to their antioxidant properties, they reduce the damage caused by free oxygen radicals in the vessel walls. By preventing oedema formation, they provide improvement in symptoms. They reduce the permeability of blood vessels by decreasing fluid and protein leakage from capillary vessels. They reduce PG by preventing leukocytes from adhering to and interacting with vessel walls, thereby exhibiting an anti-inflammatory effect. It helps increase venous tone by increasing vascular contraction with noradrenaline via alpha-1 and alpha-2 adrenergic receptors. It facilitates the resolution of oedema by supporting lymphatic circulation and facilitates return in the lymphatic system.

These medicines may be plant-based or synthetic. [16]

1- Natural

Coumarin (benzopyrones) Flavonoids (benzopyrones) Hydroxyrutosides Micronised purified flavonoid fraction (MPFF) Saponins, Other plant products

2- Synthetic

Adenosine phosphate Benzaron Calcium dobesilate

9.4. Interventional Treatment Methods

9.4.1. Sclerotherapy

Sclerotherapy is performed by injecting an irritant substance into a vein. These substances irritate the inside of the vein, causing inflammation and subsequently fibrosis (hardening). This erodes the inside of the vein. This technique has been used for spider veins for over 150 years.

Over time, this technique was developed to include foam sclerotherapy and ultrasound (USG) guidance for use in the treatment of longer and wider varicose veins. Foam sclerotherapy is obtained by mixing the sclerosant with a three-way valve using formal air or carbon dioxide as the waste gas, producing a micro-bubbled foam solution. This method is echogenic and directly visualisable with USG. The foam is injected into the varicose vein. This procedure creates intense vasospasm and ensures contact with the endothelium, which maximises the sclerosant effect and allows blood to be displaced from the vein.[8]

Sclerotherapy is a common treatment used by dermatologists in the West for people who have varicose veins in their legs. There are three main types of sclerosing solutions, which work in different ways: chemical irritants, osmotic agents, and detergent agents. Some examples of these sclerosants include STS, polidocanol, sodium morrhuate, and hypertonic saline. These solutions damage the inner lining of blood vessels, called the endothelium, by changing the tension around the cells. This process is known as the protein theft mechanism.[16]

9.4.2. Thermal Ablation Methods

Endovenous thermal ablation (EVTA) is a treatment that is usually performed on an outpatient basis under tumescent anaesthesia. During the procedure, the catheter is accessed from the lowest part of the reflux and placed 1-2 cm below the saphenofemoral junction (SFJ) at the tip of the catheter. Ascending to the highest point without damaging the femoral vein helps prevent the recurrence of accessory anterior saphenous veins. Tumescent anaesthesia is

applied to the surrounding tissues during the procedure. With tumescent anaesthesia, approximately 300-500 mL of solution is injected into the fascia, not under the skin. This method prevents damage to the perivenous tissue due to heat while also reducing vein constriction and pain.

Thermal ablation methods are as follows:

9.4.3. Endovenous laser ablation (EVLA)

Endovenous laser ablation (EVLA) is a minimally invasive procedure performed under tumescent anaesthesia and ultrasound guidance for patients with chronic venous insufficiency. In this treatment, once the entry point of the insufficient vein is identified, a laser fibre is placed into the vein using a sheath and heat is applied to the target vein, causing occlusion. EVLA uses laser energy transmitted through an optical fibre that is absorbed by blood components, creates vapour bubbles at the tip of the fibre, and causes thermal damage to the endothelium.

Parallel to technological developments, significant advances have been made in the fields of laser devices and fibre design. In particular, the development of lasers with longer wavelengths, such as 1320 nm and 1470 nm, has increased the use of water-sensitive lasers, resulting in less heat damage and lower pain levels.

All wavelengths are effective in the ablation of varicose veins, but patient comfort and side effects are important factors in their selection.

EVLA has rapidly gained popularity due to its minimally invasive nature, high success rate and low complication risks, and has now become a standard treatment for chronic venous insufficiency.[17]

9.4.4. Endovenous Radiofrequency Ablation (EVRF)

This method aims to close the venous channel using thermal energy and is of great importance as it is less invasive than surgical techniques. The RFA procedure generates heat between 85–120°C in the vessel wall, causing collagen degradation and damage to the vessel's inner surface. This effect ensures the fibrous closure of the vessel, while tumescent anaesthesia preserves the integrity of the surrounding tissue.

The basic mechanism of radiofrequency ablation involves the transmission of radiofrequency energy to the vessel wall through contact between the catheter at the tip of the device and the vessel wall. This energy generates heat, causing thermal damage to the vessel wall, collagen degradation, and damage to endothelial cells. This results in an occlusive effect within the vessel. During the RFA procedure, a pair of bipolar electrodes are firmly attached to the inner surface of the vessel. Radiofrequency energy is then transmitted between the electrodes. For this energy to pass, it must travel through the vessel wall between the electrodes. At this stage, it is essential that there is no blood in the vessel and that the catheter is firmly attached to the inner surface of the vessel. If this condition is met, the cells and proteins heat up due to the resistance they exhibit to the passage of alternating current.[18]

9.4.5. Cyanacrylate ablation (SAA)

A new method can be used for superficial venous insufficiency. This method is minimally invasive and does not involve heat. In this method, a cyanoacrylate-based biological adhesive

is injected into the vein and the inside of the vein is mechanically closed. Unlike thermal ablation methods, this treatment does not require local anaesthesia. This reduces the incidence of pain and bruising after the procedure, thereby increasing patient comfort.[19]

Avoiding high temperatures largely prevents thermal problems such as nerve damage. Furthermore, as tumescent anaesthesia is not required, the procedure becomes less invasive..[20]

When administered intravenously, cyanoacrylate rapidly hardens as it mixes with blood and mechanically seals the inside of the vessel, thereby stopping blood flow. This process causes inflammation and fibrosis (constriction) in the vessel wall, resulting in permanent closure.[20]

Phlebitis is among the most common complications following cyanoacrylate ablation and is usually caused by local inflammation occurring during polymerisation, accompanied by rapid occlusion of the vessel's internal lumen..[19]

The risk of deep vein thrombosis (DVT) is lower with cyanoacrylate ablation than with thermal ablation. This is because this method does not heat the vessels and does not damage the surrounding tissues. Furthermore, the rapid solidification of cyanoacrylate within the vessel and its very limited effect may inhibit thrombus formation..[19]

9.4.6. Mechanochemical Ablation (MOCA)

Thermal ablation techniques have several disadvantages, such as causing thermal damage to nerves, leading to paraesthesia, skin burns, and the need for tumescent anaesthesia. MOCA eliminates these disadvantages by using a catheter with a rotating motor drive unit (MDU) that creates spasms by causing mechanical damage to the endothelium. The MOCA technique not only causes mechanical spasms but also uses a sclerosing agent, which leads to further damage to the endothelium and closure of the reflux vein..[19]

9.4.7. Surgical treatments

Reflux is present in the saphenofemoral junction in 70% of the limbs for which surgery is decided due to CVI. In such patients, intervention in the saphenofemoral junction is essential to eliminate venous hypertension and prevent recurrence. The most important cause of recurrent varicosities is an inadequate and inappropriate approach to the saphenofemoral junction. The aim of surgical treatment is to remove all varicosities along with their source of venous hypertension, to achieve aesthetically pleasing results, and to minimise complications. Pain, heaviness in the legs, superficial thrombophlebitis, pigmentation at the ankle, lipodermatosclerosis, white atrophy, and ulcer formation are symptoms and findings that necessitate surgical treatment in CVI. If such symptoms and findings are present, the decision regarding which surgical treatment method to choose should be made on a case-by-case basis..[21]

9.4.8. Saphen Vein Stripping

CVİ is applied in cases of femorosaphenous insufficiency where the deep venous system is normal. It can also be applied if saphenous vein insufficiency is responsible. While saphenous vein ligation and stripping can be performed, it is known to significantly improve venous haemodynamics, prevent deep venous insufficiency, and contribute to venous ulcer healing by reducing symptoms in advanced stage patients..[21]

9.4.9. High Ligation

If the problem is solely insufficiency of the saphenofemoral junction and the saphenous vein is not dilated and tortuous, it is ligated only proximally along with the saphenous vein branches. The saphenous vein itself is preserved.

9.4.10. Phlebectomy – Mini Phlebectomy

It is mostly performed for cosmetic purposes. It is often performed in addition to the stripping procedure. Varicose veins of the saphenous veins, with or without side branches, are removed separately through separate incisions.[19]

9.4.11. Perforator Vein Surgery

Historically performed surgical procedures (such as the Linton procedure) have not found widespread use due to the extensive tissue damage they cause. However, subfascial endoscopic perforator surgery (SEPS) and perforator ligation, which are currently used methods, are more widely accepted. Making remote incisions in ulcerated or lipodermatosclerotic skin has been advantageous.

9.4.12. Other surgical techniques

Venous bypass procedures are based on the principle of bypassing the occluded area in cases of chronic venous insufficiency (CVI) caused by occlusion. For example, in cases of unilateral iliac vein occlusion, a femorofemoral crossover venous bypass may be performed (Palma procedure).

Reconstructing venous valves involves methods such as openly recreating the valve in cases of segmental valve deficiency, using external support, or transplanting the intact valve portion of another vessel to another location. However, these methods are not generally considered the first choice due to the high risk of thrombosis, small openings, high treatment risk, and the need for specialised skills.[21]

10. CONCLUSION

The choice between surgical or endovascular methods during the treatment of venous insufficiency should be determined on an individual basis, taking into account factors such as the patient's body structure, condition, and CEAP classification, as well as the patient's expectations. Each type of intervention has different advantages, limitations, and potential risks. Initially, conservative treatment may be applied in some patients, but if intervention is necessary, endovascular procedures should be preferred due to their minimally invasive nature. Endovascular methods are generally recommended to be used in conjunction with permanent compression therapy for one week. In some cases, procedures such as phlebectomy or sclerotherapy may also be included in the treatment programme. Ultimately, the treatment approach for each patient should be carefully tailored, and situations requiring multiple procedures, as well as the techniques to be used, should be carefully considered.

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AORT KAPAK CERRAHİSİNDE YENİLİKÇİ BİR YAKLAŞIM: OZAKİ PROSEDÜRÜ

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[1]Sağlık Bilimleri Üniversitesi İstanbul Dr. Siyami Ersek Göğüs Kalp ve Damar Cerrahisi Eğitim ve Araştırma Hastanesi – Kalp ve Damar Cerrahisi Bölümü

1. GİRİŞ

İleri aort kapak patolojilerinin tedavisinde ister stenoz ister yetmezlik olsun halen altın standart yöntem aort kapak replasman (AVR) cerrahisidir.[1] Günümüzde güncel guideline'larda mitral kapak cerrahisi için tamir yöntemleri mitral kapak replasmanına oranla daha yüksek öncelikli olsada aort kapak için bu durum söz konusu değildir. Aort kapakta AVR halen daha altın standart yöntemdir.[2] Protez kapak takılan hastalarda, hasta – kapak uyumsuzluğu, rezidü gradient kalması, paravalvüler kaçak, protez kapak endokarditi ve trombozu, ömür boyu antikoagülan kullanma gereksinimi ve düzensiz kullanma sonucu oluşabilecek kanama gibi komplikasyonlar en büyük sorunlardır.[3] Bunlar ve benzeri nedenlerden dolayı günümüzde halen daha ideal bir protez aortik kalp kapağı henüz geliştirilmemiştir. Son yıllarda ileri aort stenozu olan hastalarda transkateter aort kapak replasmanı yapılabilmektedir. Fakat bu yöntem hem maliyeti çok yüksek hem de 10 yıllık sonuçları cerrahi prosedürler kadar yüksek değildir.[4] Aort kapak cerrahisi için protez kapak komplikasyonlarından etkilenmeyecek, rezidü gradient kalmayacak yüksek maliyetli olmayacak bir yöntem ilk defa 2007 yılında Ozaki ve ark. tarafından geliştirilmiştir.[5] Bu yöntem ile hastanın otolog perikardı gluteraldehit ile işlemden geçirildikten sonra uygun ölçülerde kesilerek ve belirtilen teknik ile rezeke edilen aort kapak yerine süture edilerek neo-aort kapak oluşumu gerçekleştirilir.

2. CERRAHİ TEKNİK

Standart bir median sternotomi kesisi yapılır. Sternotomi sonrasında, perikardın parietal yüzeyindeki yağ ve fibröz dokular temizlenir ve yaklaşık 7 × 8 cm'lik bir perikard alanı hazırlanır. Perikardın diyafragmatik kenara yakın kısmı perikardın en kalın ve dayanıklılığı en sağlam olan kısmıdır bu bölge kalem ile işaretlenir ve perikard grefti, koter kullanılmadan yalnızca makasla çıkarılır. Alınan perikard grefti kenarlarından yırtmadan gerdirilerek Ozaki tarafından geliştirilen setin içinde bulunun düz bir plak üzerine sabitlenir. Ardından greft, 0.6% glutaraldehit çözeltisinde 10 dakika bekletildikten sonra plak üzerinden çıkarılır ve bütün halinde yıkama işlemine geçilir. Son aşamada, üç kez 6'şar dakika izotonik solüsyon içinde bekletilerek yıkanması sağlanır.

Cerrahi işlem kardiyopulmoner bypas ve aortik kros klemp altında gerçekleştirilir. Aortotomi, sağ koroner orifisin yaklaşık 1.5–2 cm distalinden transvers şekilde yapılır.(Resim-1)

Nativ aort kapağı ve tüm kalsifikasyonlar, anülüsün bütünlüğüne zarar vermeden rezeke edilir. Her komissür arası mesafe, Ozaki tarafından geliştirilen özel aparat kullanılarak ölçülür. Ölçüm sırasında, her komissür arası mesafenin orta noktası da silinmez bir kalemle işaretlenir. Bu nokta anular devamlı dikişin başlanacağı merkez noktadır. Her yaprakçık için ayrı ayrı bir ölçüm hesaplanır. Eğer iki size arasında kalınırsa daha büyük olan tercih edilir. Yine herhangi iki kusp arasında 1 size dan daha fazla büyüklük farkı olmamalıdır. (Örn; sağ koroner kusp 29, non koroner kusp 31 olarak ölçüldüğünde sol koroner kusp 27 olamaz bunun yerine sol koroner kusp için bir büyük size olan 29 numara ölçüm kullanılmalıdır.)

Perikard yıkanma işlemleri tamamlandıktan sonra aortik anulusta interkomissürel mesafelerde hesaplanan ölçümlere göre, perikardın viseral yüzünden Ozaki'nin geliştirdiği setler kullanılarak yaprakçıklar ve dikilecek noktalar silinmez kalem ile çizilir. Mümkün olduğunca diyaframa yakın olan işaretli yüzey kullanılır; çünkü perikardın en kalın bölgesi burasıdır. Perikard, kanat dikiş noktasını içinde bırakacak şekilde her iki taraftan makasla kesilir ve her bir yaprakçık ayrı ayrı hazırlanır.

Neokusplar sırasıyla sağ, sol ve non-koroner olarak anülüs halkasına dikilir; ancak bu sıralama şart değildir. Bazı olgularda sol koroner kusptan başlandığı da olur. Dikişler için 4-0 polipropilen tercih edilir. Neokusplar, viseral yüzeyleri ventriküle bakacak şekilde yerleştirilir. Önce komissür arası orta nokta ile neokusp orta noktası karşı karşıya gelecek şekilde dikiş geçilir ve neokusp anülüsün altına bağlanır (3 düğüm). Dikiş tekniğinde; neokusp üzerinde aortik taraftan ventriküle doğru, anülüs üzerinde ise ventrikülden aort tarafına doğru geçilir. İlk üç dikişte anülüs/neokusp geçiş oranı 1/3 olur, kalan mesafe eşitlenince oran 1/1'e döner. Sürekli dikiş komissürde sonlandırılır ve sütür aortun komissüründen 2–3 mm aşağıdan çıkarılır. Komissür dışına çıkılan son dikişten bir önceki dikiş, "big bite" (büyük ısırık) olarak adlandırılan ve anülüsün daha derin ve daha kalın alınmış bir dikişidir.

Tüm neokusplar aynı yöntemle tamamlanır. Ardından yeni bir 4-0 dikiş, komşu neokuspların köşelerinden ve ardından kanatlarından geçirilir; kanatların aort duvarına temas etmesi için sütür aortun komissüründen 2–3 mm yukarıdan çıkarılır. Her komissürde toplam 4 dikiş bulunmalıdır: 2 üstte, 2 altta. Bu dikişler komissürün dışında 5 × 10 mm genişliğinde bir plejit ile sabitlenir. Böylece rüzgar gülü görünümünde yeni bir kapak oluşturulmuş olur.(Resim-2) Kardiyopulmoner baypastan ayrıldıktan sonra neokapağın koaptasyonu transözofageal ekokardiyografi ile kontrol edilir.

3. AVANTAJLARI ve DEZAVANTAJLARI

Ozaki tekniğinin en büyük avantajı postop dönemde herhangi bir antikoagülan tedaviye ihtiyaç olmamasıdır. Dolayısıyla antikoagülan tedavinin neden olabileceği kanama, serebrovasküler olaylar, düzenli inr takibi gereksinimi tüm komplikasyonlardan hastayı korumuş oluruz. Yabancı bir materyal içermediğinden enfeksiyon oranı diğer protez kapak takan hastalar oranla daha düşüktür.[6] Bir diğer en büyük avantajı ise aort kapakta postop dönemde nativ kapağa en yakın basınç gradientleri ve nativ kapağa en yakın serbest aort kök hareketlerine sahip olmasıdır. Ozaki yöntemi ile oluşturulan yeni kapağın postop dönemdeki

hemodinamik fonksiyonu nativ kapağa en yakın çalışan cerrahi prosedür olarak literatürde yerini almıştır.[7] Özellikle dar aort köküne sahip hastalarda bu teknik kapak replasmanına göre belirgin avantaj sağlar. Bir kere alınan ve tekrar tekrar kullanılabilen Ozaki Seti dışında herhangi bir maliyetinin olmaması ekonomik açıdan da bu tekniği avantajlı kılar.[8]

Bu kadar avantajını sıraladığımız Ozaki yönteminin en büyük dezavantajı ise çok daha yeni teknik olduğundan dolayı uzun dönem sonuçlarının net olarak ortaya konamamasından kaynaklıdır. Literatüre baktığımızda erken – orta dönem sonuçlarına göre 10 yıllık sağ kalım oranları yüzde 80'lerin üzerinde olup bu dönemler için yüz güldürücüdür. Bir diğer önemli dezavantajı ise protez kapak ile aort kapak replasmanı operasyonlarına göre daha uzun kross-klemp ve kalp akciğer pompa süresinin olmasıdır.[9]

4. SONUÇ

Tekniği geliştiren Ozaki ve ekibinin yayınladığı 10 yıllık takip sonuçlarına göre %85,9 sağkalım ve sadece % 4.2 reoperasyon oranlarına sahiptir. %7,3 oranında hasta ise orta ve ileri rekürren aort yetersizliği nedeniyle takip edilmiştir. Reoperasyon oranlarında en sık enfektif endokardit neden olurken yine de bu oran protez kapaklara göre daha düşük bir orandadır.[10]

Sonuç olarak Ozaki tekniği standart AVR yerine uygulayabileceğimiz postop dönemde antikoagülasyon kullanımı gereksinimi olmayan ve kapakta minimal rezidü gradient ile kapak replasmanı yapmamızı sağlayan, ekonomik maliyeti standart AVR'ye göre çok düşük, otolog perikard kullanıldığımız bir aort kapak replasman/tamir yöntemidir.

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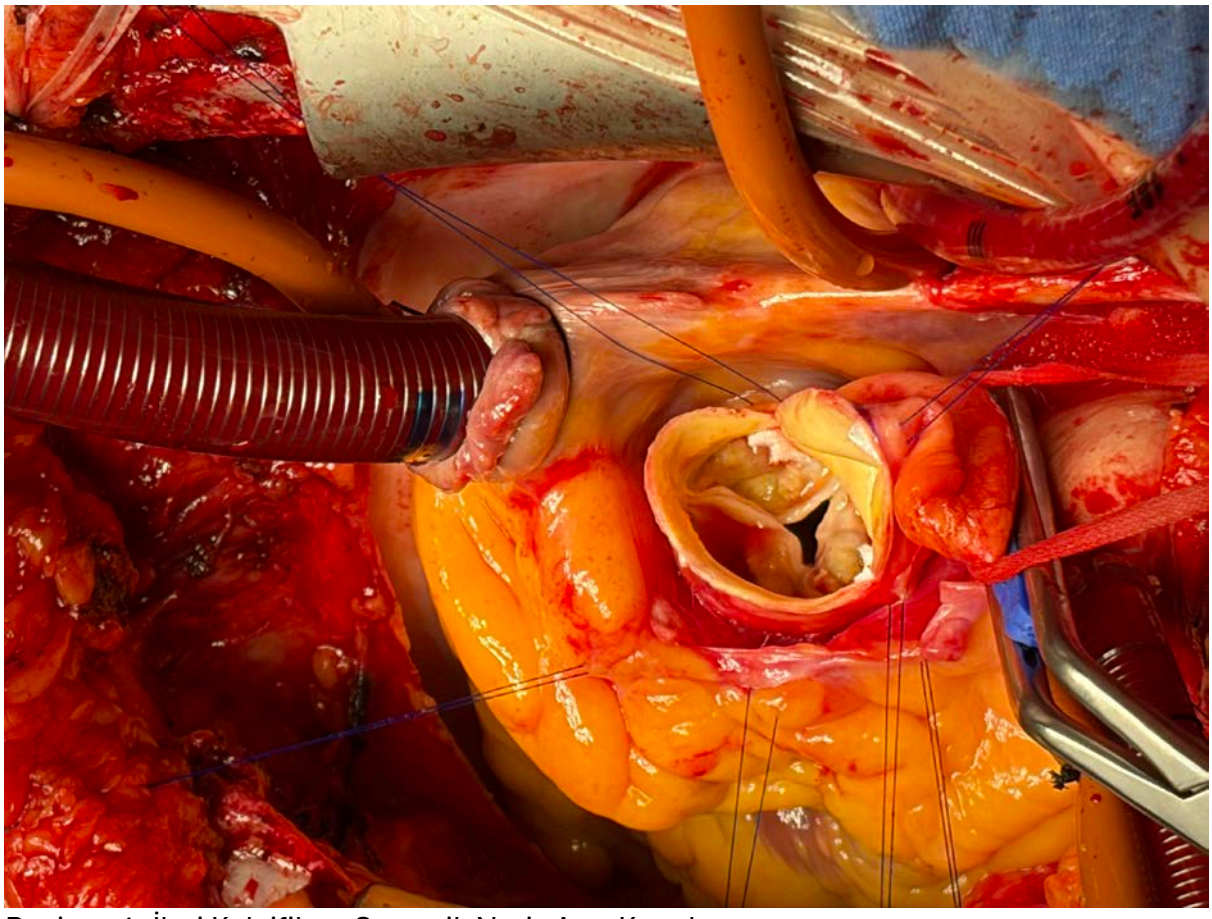
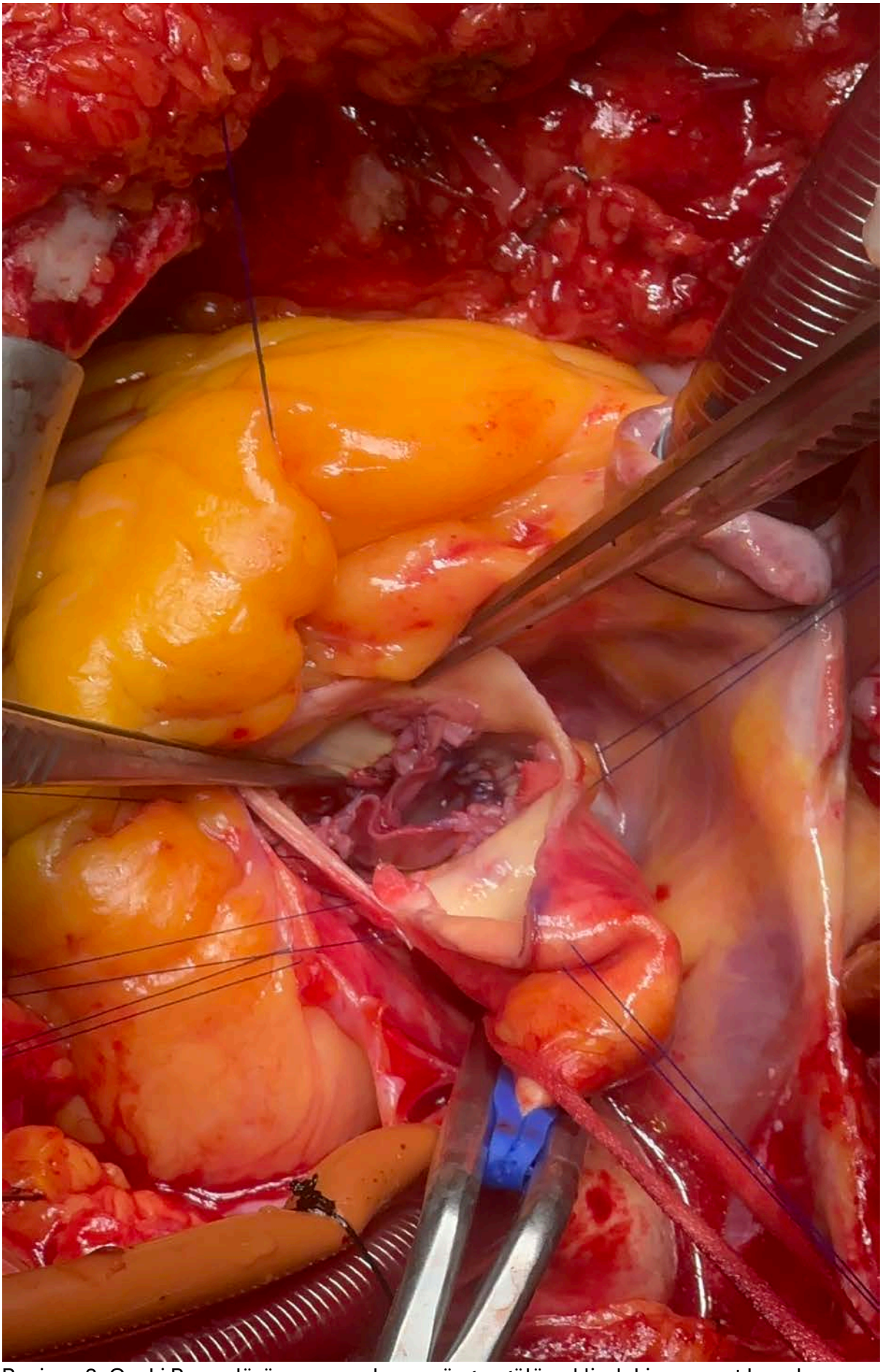


Resim – 1: İleri Kalsifik ve Stenotik Nativ Aort Kapak



Resim – 2: Ozaki Prosedürü sonucu oluşan rüzgar gülü şeklindeki neo-aort kapak

TÜRKİYE VE DÜNYADA

KALP VE DAMAR CERRAHİSİ

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