

# Aortic dissection in cardiac ICU : Retrospective analysis of outcome and postoperative complications

---

**Kuric, Nicole Ana**

**Master's thesis / Diplomski rad**

**2022**

*Degree Grantor / Ustanova koja je dodijelila akademski / stručni stupanj:* **University of Split, School of Medicine / Sveučilište u Splitu, Medicinski fakultet**

*Permanent link / Trajna poveznica:* <https://um.nsk.hr/um:nbn:hr:171:161421>

*Rights / Prava:* [In copyright](#) / [Zaštićeno autorskim pravom.](#)

*Download date / Datum preuzimanja:* **2024-07-10**



*Repository / Repozitorij:*

[MEFST Repository](#)



**UNIVERSITY OF SPLIT  
SCHOOL OF MEDICINE**

**NICOLE ANA KURIC**

**AORTIC DISSECTION IN CARDIAC ICU: RETROSPECTIVE ANALYSIS OF  
OUTCOME AND POSTOPERATIVE COMPLICATIONS**

**DIPLOMA THESIS**

**Academic year:**

**2021/2022**

**Mentor:**

**Assoc. Prof. MLADEN CAREV, MD, PHD**

**Split, July 2022**

## TABLE OF CONTENTS:

<b>1. INTRODUCTION</b> .....	1
1.1 Aortic Dissection Definition.....	2
1.2 Pathogenesis of Aortic Dissection.....	2
1.3 Etiology of Aortic Dissection.....	3
1.3.1 Gender Related Risk Factors.....	4
1.3.2 Seasonal Correlation.....	4
1.3.3 Marfan Syndrome.....	5
1.3.4 Ehlers-Danlos Syndrome.....	5
1.3.5 Loeys-Dietz Syndrome.....	6
1.3.6 Bicuspid Aortic Valve.....	6
1.3.7 Familial Aortic Dissection.....	7
1.3.8 Intramural Hematoma.....	8
1.3.9 Penetrating Aortic Ulcers.....	8
1.4 Classification of Aortic Dissection.....	8
1.5 Clinical Presentation of Aortic Dissection.....	10
1.6 Physical Examination of Aortic Dissection.....	11
1.7 Imaging and Diagnostics of Aortic Dissection.....	12
1.8 Surgical Treatment.....	13
1.8.1 Surgical Indications.....	13
1.8.2 Surgical Treatment for Type A Dissections.....	14
1.8.3 Surgical Treatment for Type B Dissections.....	15
1.9 Postoperative Complications.....	16
1.10 Chronic Aortic Dissection.....	17
<b>2. OBJECTIVES</b> .....	19
2.1 Aim of Study.....	20
2.2 Hypotheses.....	20
<b>3. MATERIALS AND METHODS</b> .....	21
3.1 Study Design.....	22
3.2 Data Extraction.....	22
3.3 Data Analysis.....	22
3.4 Statistical Analysis.....	22
<b>4. RESULTS</b> .....	23
<b>5. DISSCUSION</b> .....	28
<b>6. CONCLUSION</b> .....	31

<b>7. REFERENCES</b> .....	33
<b>8. SUMMARY</b> .....	36
<b>9. CROATIAN SUMMARY</b> .....	38
<b>10. CURRICULUM VITAE</b> .....	40

## ACKNOWLEDGMENT

*First and foremost, I'd like to thank my mentor Assoc. Prof. Mladen Carev, MD, PhD for providing me with guidance and help with the writing process. I am grateful for your time and patience during this whole process.*

*A huge thank you to my parents for providing me with this opportunity to study medicine and for their love and support through all these years. I greatly appreciate the sacrifices they have made to help make this possible. I also thank all my family for their continuous love and support which has helped me overcome many obstacles along the way.*

*I would also like to thank my friends in Split and back home for all the support and amazing memories that have been made along the way. Our time together has been unforgettable. Thank you for always being there.*

*Last but not least, I would like to thank my partner Tristan Frembling for being by my side and providing me with the support I needed during the best and worst times over the last couple of years.*

## **LIST OF ABBREVIATIONS**

**ECG- Electrocardiogram**

**TEE- Transesophageal echocardiography**

**CT- Computed tomography**

**MRI- Magnetic resonance imaging**

**AR- Aortic regurgitation**

**CABG- Coronary artery bypass graft**

**DHCA- Deep hypothermic circulatory arrest**

**TEVAR- Thoracic endovascular aortic repair**

**AKI- Acute kidney injury**

**FL- False lumen**

**MDCT- Multidetector computed tomography**

**FBN1- Fibrillin-1**

**TAAD- Thoracic aortic ascending aortic aneurysms and dissections**

**TGFBR2- Transforming growth factor beta receptor type II**

**TGFb- Transforming growth factor beta**

**EDS- Ehlers-Danlos syndrome**

**vEDS- Vascular Ehlers-Danlos Syndrome**

**LDS- Loeys-Dietz Syndrome**

**BAV- Bicuspid Aortic Valve**

**MSCT- Multislice computed tomography**

## **1. INTRODUCTION**

## **1.1 Aortic Dissection Definition**

Aortic dissection is an acute life-threatening cardiac emergency. It results from an intimal tear that exposes the media to the pulsatile force of blood within the aortic lumen. Blood may then exit the true aortic lumen and dissect the aortic wall to create a false lumen (1). With each contraction, the portion of the lumen that is dissected may lengthen more proximally or distally, which may endanger branch arteries or cause rupture as the outer wall of the artery weakens (2). Branch occlusion or shearing of the intima may occur when the dissection propagates into aortic branch vessels which would result in intimal fenestrations (1).

## **1.2 Pathogenesis of Aortic Dissection**

The wall of the aorta is three-layered. The innermost layer, named the intima, is a metabolically demanding single endothelial layer that is supported by a relatively loose connective tissue sublayer. The sublayer allows for the motion of the intima with the media when the aorta contracts and expands during the cardiac cycle. The middle layer is named the media, which is comprised of about fifty layers of fenestrated, lamellar elastic fibers as well as interposed collagenous fibers and smooth-muscle cells. The aorta is impressively distensible and elastic due to elastin, which is highly stretchable; the fibers are able to stretch two to three times without rupturing. Collagenous fibers have the opposite characteristics of elastin, it is estimated that the stiffness is five thousand times the stiffness of elastin. Their main function is to maintain the strength of the aorta and to withstand shearing forces from the blood that is pumped by the left ventricle. The outermost layer is the adventitia, which is a strong layer of collagen and connective tissue which provides the strength to the aorta. The vasa vasorum provides the nutritional circulation to the rest of the wall of the aorta due to the inability of the aorta to solely rely on the diffusion of nutrients from the lumen.

As stated above, aortic dissection occurs when an entry tear, or an intimomedial tear, allows blood flow to pass into the aortic wall allowing for a new secondary channel, named the false lumen (FL). The FL propagates distally most often in a spiral, or in a straight manner. The FL may also spread proximally to the aortic valve. It also occurs that the true lumen may become



constricted by the pressurized FL, in some cases the true lumen may collapse which can lead to ischemia of the tissue below the point of collapse. The proximal aorta is typically the site of a primary entry tear or a secondary tear, also known as fenestrations, these may occur either in the thoracic aorta, abdominal aorta or the iliac arteries.

It is understood that a diseased or weakened vessel wall is an instigating factor that renders the aorta vulnerable when exposed to severe or uncontrolled hypertension. The conditions that lead to a weakened vessel wall includes Marfan syndrome and other connective tissue disorders, prior cardiac surgery, male sex, age between sixties and seventies, hypertension and many other conditions (3).

### **1.3 Etiology of Aortic Dissection**

There are many predisposing conditions that may lead to the formation of an intimal tear and consequently dissection. These conditions cause the weakening of the aortic wall and lamina media which may leads to a higher wall stress, this in turn induces dilation and aneurysm development, ultimately causing rupture or dissection. These conditions include inherited diseases, trauma, and iatrogenic causes. Inherited diseases include Marfan's syndrome, which is a defect in collagen and elastin which are main components of the walls of arteries. Ehler-Danlos syndrome is due to a structural anomaly in collagen type III which like Marfan's syndrome, is a major component of the walls of arteries (4). Iatrogenic causes of dissection may include cardiac catheterization, aortic cannulation, and manipulation of the heart during surgery (5).

Two other main conditions that are the main causes of degenerative mechanisms are atherosclerosis and arterial hypertension. Atherosclerosis leads to the thickening of the intima, reduced cellularity and collagen fiber hyalinization, and reduced nutritional supply dues to fibrosis which leads to necrosis. These changes to the aorta contribute to a higher susceptibility to shear stress as well as increased vessel stiffness, potentially causing aneurysms and dissections (4). Arterial hypertension is the main causative factor which acts as a parietal stressor and a proinflammatory trigger directly and indirectly, respectfully. Hypertensive patients have elevated proinflammatory molecules suggesting that this elevated state may lead to excessive extracellular matrix degeneration ultimately leading to aortic dissection. Hypertension the most significant risk factor which is present in 80% of patients with aortic dissection. Hypertension consists of 54% of

the population-attributable risk for acute aortic dissection in the general population, the incidence in person-years in this population is 21 per 100 000 unlike in normotensive individuals where the incidence is 5 per 100 000. Ultimately for those, who for five years before the manifestation of dissection had a higher blood pressure, are more likely to be pronounced deceased before making it to the hospital than those with better blood pressure control or with a general lower blood pressure (6).

### **1.3.1 Gender Related Risk Factors**

A study based on observational analysis from the International Registry of Acute Aortic Dissections found that 67% of patients were diagnosed with type A, and the remaining as type B. Male patients predominated both groups however female patients had a worse outcome (7). Female patients were found to be older when first diagnosed, about 10 years later, and there was no significant difference found between outcomes of either in-hospital or all-cause mortality of both type A and type B stent surgery populations (7,8). The later stage of onset is consistent with the incidence of cardiovascular disease in general. Literature suggests that premenopausal women have a lower risk of cardiovascular disease than postmenopausal women due to the protective effects of estrogen. Estrogen decreases the proportion of collagen and stimulates the formation of fibrillin in the aortic wall, decreasing the wall stiffness of the aorta and other arteries. Female patients were diagnosed more frequently with more proximally located dissection, such as DeBakey type II. However, it was found that with increasing age, healthy females have a greater increase in the ascending aorta and the aortic arch diameters than healthy males.

In males, the mean wall thickness in both the ascending and the descending aorta is higher, and the peak wave velocity is lower in healthy females. Lastly, the blood flow dynamics in the aorta are significantly different between males and females. During follow-up period, male patients were at a higher risk of redo aortic surgery than female patients in the type A open surgery group (8).

### **1.3.2 Seasonal Correlation**

In a study done to find the correlation between seasonal weather change and the occurrence of aortic dissection, the analysis revealed that there is a significantly increased number of events during certain months of the year, March, October, and December. Atmospheric temperature showed an impact whereas air pressure and humidity did not. A subgroup analysis of hypertensive

vs. non-hypertensive revealed no impact on aortic dissection through air pressure and humidity but a significantly monthly variation of dissection occurrence in hypertensive population. In hypertensive patients, a significantly increased risk of thirty-four percent was present in correlation with rising atmospheric temperature and this was not seen in non-hypertensive patients. In current literature several different correlations between weather condition and the incidence of dissection have been described as having and increased incidence during winter and lower frequency in summer (9).

### **1.3.3 Marfan Syndrome**

Marfan syndrome is a somewhat common disorder of connective tissue with autosomal dominant inheritance. This disorder's prominent manifestations are shown in defects of the cardiovascular, ocular, and skeletal systems which are due from mutations located in the gene for fibrillin-1, FBN1. Those with Marfan syndrome have a characteristic physical appearance of scoliosis, tall stature, arachnodactyly, long slender limbs, and pectus excavatum or carinatum. A common ocular manifestation is ectopia lentis which affects eighty percent of individuals and is usually bilateral. Aortic dissection, which typically follows a phase of gradual dilation of the ascending aorta, is the main cause of premature death in those who remain untreated (10).

### **1.3.4 Ehlers-Danlos Syndrome**

Ehlers-Danlos Syndrome is also a connective tissue disorder that is genetically, as well as clinically, heterogeneous and the phenotype is shown by abnormalities of skin, joints, ligaments, internal organs and blood vessels. Roughly one quarter of EDS patients develop an aortic aneurysm with a prevalence of 1:5,000 to 1:25,000. Classic as well as hypermobile cases of EDS account for ninety percent whereas vascular EDS accounts for five percent of cases. This syndrome is due to mutations in the genes which code for the formation of collagen fibrils or the proteins for processing of the collagens. There are 13 recognized subtypes of EDS.

One of the 13 subtypes is vascular EDS or vEDS is autosomal dominant and is due to mutations in COL3A1. vEDS is found in nearly five percent of all EDS cases and the prevalence ranges from 1:50,000 to 1:100,000. This subtype is characterized by abnormalities of the blood vessels, skin, hollow organs and joints. This subtype has the worst prognosis due to the possible rupture of arteries and hollow organs. Specific for this subtype is, arterial rupture, with or without the development of an aneurysm at a young age. Rupture most often affects the abdominal vessels

including the renal, mesenteric, hepatic, iliac, or femoral arteries. Involvement of the aorta may also be involved and the branches most often affected are the carotid, subclavian, ulnar, popliteal, and tibial arteries. Due to the fragility of the tissue, surgical interventions commonly have high complication rates (11).

### **1.3.5 Loeys-Dietz Syndrome**

Loeys-Dietz syndrome was first defined by Bart Loeys and Harry Dietz in 2005. Its characteristic appearance can be differentiated from Marfan syndrome by the presence of aortic and arterial aneurysm and tortuosity, hypertelorism, as well as abnormal uvula or cleft palate. Compared to Marfan syndrome, cardiovascular manifestations are more severe and aneurysms may dissect or rupture at a smaller diameter and at a younger age than in individuals with Marfan syndrome. Additionally, aortic aneurysms are not limited to the root or ascending aorta, and commonly affect aortic side branches as well as cerebral vessels. Cardiovascular features typically appear first in LDS patients, which differs from Marfan syndrome (11).

### **1.3.6 Bicuspid Aortic Valve**

The most common congenital cardiac defect is bicuspid aortic valve disease and is typically created by 2 unequal-sized leaflets. The morphologic appearance varies with which commissures have fused, the most prevalent is the fusion of the left and right cusps. BAV disease is often related to other congenital cardiac lesions, the most common being the dilation of the ascending aorta which is derived from the anomalies of the aortic media. Symptoms may manifest in patients during adulthood however, there is a broad range of presentation from severe disease which may be noticed in utero or asymptomatic disease that developed later in life. The process by which alterations occur may be secondary to flow dynamics however, current studies have demonstrated that structural abnormalities arise at the cellular level and separate of the hemodynamic lesion. There is a decreased amount of fibrillin, and increased elastin fragmentation and apoptosis in the thoracic aorta. Deficiency of fibrillin-1 effects smooth muscle cell detachment, matrix disruption, and cell death.

Dilation of the aortic root has been recognized during youth, implying that it occurs earlier in life therefore, children with BAV have larger increases in aortic proportions than those of the same age with trileaflet valves. In BAV disease, the aortic annulus, sinus, and proximal ascending aorta are larger than those found in adults with trileaflet valves. Aortic dissection has been

associated with BAV in only four percent of cases. However, due to the increased incidence of BAV compared to Marfan syndrome, dissections due to BAV are therefore more frequent. Dissections that occur in BAV patients typically involve the ascending aorta however, involvement of the descending aorta has been reported in older patients. Dissection is more common in patients with dilated aortas, but there are reports of dissection in normal-sized aortic roots and after valve replacement. Risk factors for dissection include aortic size, aortic stiffness, male sex, family history and the presence of coarctation of the aorta and Turner syndrome (12).

### **1.3.7 Familial Aortic Dissection**

Thoracic aortic aneurysms that may lead to type A dissection (TAAD) may be also cause from familial inheritance, which is of autosomal dominant inheritance however, affected individuals do not exhibit external features of connective tissue disorders or any other recurrent phenotype. Studies have shown that first degree relatives of those with TAAD, without syndrome, have a greater risk of aneurysms in the thoracic aorta and sudden death compared to individuals without affected first degree relatives. This supports the idea that genetic factors have a part in the etiology of TAADs in those who do not have a linked syndrome which may be causing aortic dissection.

Studies have shown that in the majority of families with aortic disease, it is not from a mutation in the FBN1 gene or from other genes coding for vascular proteins, such as COL3A1. Several loci for non-syndromic TAAD have been mapped in recent studies. The first locus is TAAD1, which is found on chromosome 5, and was proved by a study done on a Finnish population where roughly one-half of the families demonstrated linkage to TAAD1. Another locus for familial inherited aortic aneurysms and dissections is found on the long arm of chromosome 11 which was done using a single family. The phenotype of the family who was discovered to have the FAA1 locus showed a diffuse vascular etiology. Separately from the dilation of the sinuses of Valsalva, dilation of the abdominal aorta and left subclavian artery was also involved. The FAA1 locus is a rare source of a vascular disorder due to no other family having had demonstrated the association of the phenotype to this locus.

A third locus for TAAD was found on chromosome 3 by using an additional sizeable family with multiple people having been diagnosed with thoracic aortic aneurysms and dissections. This relation of the disease within the family was found to be autosomal dominant with decreased

penetrance with a variable age of disease onset. Mutations in the transforming growth factor beta receptor type II gene (TGFB2) was newly found to be the reason for the disorder at the TAAD2 locus. The TGFB2 gene was assessed for exon splicing errors, nonsense, and missense mutations, in four of the 80 unrelated families mutations were observed, suggesting that TGFB2 mutations are a somewhat rare source of familial TAAD. Therefore, TAAD is a genetically diverse disease which may be inherited with a syndrome or without a syndromic tendency for TAAD. Studies have also been done to assess the impact of TGFB2 mutations, and have emphasized that the dysregulation of the TGFb pathway may lead to aneurysm formation (10).

### **1.3.8 Intramural Hematoma**

Intramural hematoma results from a hemorrhage within the wall of the aorta without an intimal tear or flap. It has been considered a precursor of aortic dissection, and in fact does evolve into dissection, with a double-barrel aorta, in approximately 20% of cases (3). Intramural hematoma is caused by spontaneous bleeding from the vasa vasorum into the aortic media which is different from aortic dissection that is arising from an intimal tear. Intimal tear is considered a risk factor for a worse prognosis in these patients and the relationship between intimal tear and intramural hematoma remains unclear. However, multidetector computed tomography imaging showed the presence of small intimal tears in seventy-one to seventy eight percent of patients diagnosed with intramural hematoma. Surgical studies confirmed intimal disruption in seventy-three percent of patients, including those without evidence of the tear in preoperative MDCT in 51.9% of patients (13).

### **1.3.9 Penetrating Aortic Ulcers**

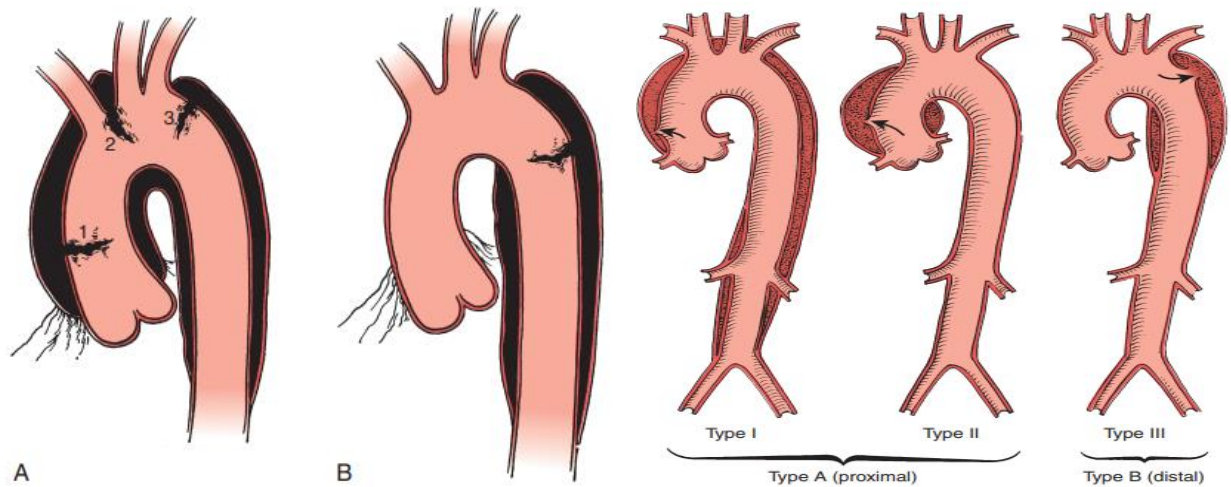
Penetrating aortic ulcers typically develop in the descending thoracic aorta however they may form anywhere along the aorta. Patients are more likely to be elderly with considerable atherosclerosis. The ulcer may precede aortic dissection as well as be associated with intramural hematoma. Early intervention and repair are warranted when there is both a penetrating ulcer and an intramural hematoma. Penetrating aortic ulcers tend to be unpredictable and may lead to rupture and hemorrhage. TEVAR has emerged as a treatment option due to the ulcer's development in areas of anatomical suitability for endovascular repair and engrafting. Intervention is justified for ulcers larger than 3cm in diameter, as well as for all symptomatic aortic ulcers regardless of size (3).

## 1.4 Classification of Aortic dissection

Aortic dissection is classified by two main classifications, Stanford and DeBakey. Stanford A dissection includes the ascending thoracic aorta independent of entry site, extent, or origin. Stanford B dissection includes only the descending thoracic aorta distal to the origin of the left subclavian artery irrespective of the level or entry site (1,5). DeBakey is classified into three groups (Figure 1), type I, type II, and type III. DeBakey type I dissections derive from an entry site in the ascending aorta which may expand to include the rest of the aorta. Type II originates from, and is limited to, a primary entry site in the ascending aorta. DeBakey type III dissections derive from an entry site in the descending thoracic aorta and are limited distal to the beginning of the left subclavian artery. Type III classification is further subdivided into IIIA, which is restricted to the thoracic aorta above the diaphragm, differing from subtype IIIB which spreads below the diaphragm to the abdominal aorta (1). Thus, Stanford A dissections include DeBakey types I and II, and Stanford B dissections include DeBakey type III (5).

Recent studies have shown that aortic ulcers and intramural hemorrhage and hematomas may be early indications of developing dissections, leading to a new classification with five classes. Class 1 is defined as classical aortic dissection that includes an intimal flap between the true and false lumen, the intimal flap tears characterize communicating dissections (4). The following class, Class 2, is medial disruption with formation of intramural hematoma/hemorrhage, which may be the result of rupture vasa vasorum and the initial lesion in cases of cystic medial degeneration (4,5). Class 2 dissections are found to be present in ten to fifteen percent of patients with suspected aortic dissection (5). Class 3 is defined as a subtle dissection without hematoma, with an eccentric bulge at the tear site (4). This type of dissection is characterized by a stellate or linear intimal tear with the exposure of the underlying media and adventitia but without progression to separation of medial layers (5). The next class, class 4, is described as a plaque rupture leading to aortic ulceration, penetrating aortic atherosclerotic ulcer with surrounding hematoma, usually subadventitial, and this ulceration of the aortic plaques can lead to dissection (4,5). The final class is class 5, which is a traumatic or iatrogenic cause of aortic dissection (5). These classes, 1-5, represent a subdivision to Stanford or DeBakey classification (4). If the mechanism is blunt trauma, the dissection may be found to be at the level of the aortic isthmus.

However, iatrogenic dissections may follow aortic angioplasty for aortic coarctation or after cross-clamp of the aorta during heart surgery (5).



**Figure 1.** Stanford and De Bakey classifications. Source: Kaplan JA, Reich DL, Savino JS. Kaplan's Cardiac Anesthesia: The Echo Era. 6th ed. St. Louis, Missouri: ELSEVIER; 2011. P. 658.

### 1.5 Clinical Presentation of Aortic Dissection

Aortic dissection is an emergency, such the swift recognition of symptoms and clinical presentation is imperative to diagnosing and treating the condition as quickly as possible. The typical presentation is a male with a history of hypertension in his sixties who presents with sudden chest pain (4). Those with Marfan syndrome, bicuspid aortic valve, Loeys-Dietz syndrome, aortic coarctation Ehlers-Danlos syndrome, and familial aortic dissection usually have an earlier onset of the disease. As stated previously, aortic dissection is most associated with hypertension and less due to atherosclerosis. Other associated risk factors include pregnancy, aortic trauma, cocaine abuse, and arteritis (1).

The common clinical manifestations that are present in ninety percent of patients presenting with aortic dissection include, severe abrupt pain that is maximum at the time of onset, usually sharp, stabbing or tearing, the pain may be located retrosternally, for proximal dissections; this is contrasted from more distal dissections which are interscapular or include



back pain (4). Upon further clinical evaluation of the patient, we may find: a highly suggestive finding of a pulse deficit, with systolic blood pressure with a limb differential greater than 20 mm Hg, new murmur that is indicative of aortic regurgitation and a focal neurologic deficit (1). In addition to the more common presenting symptoms, there are also fewer common symptoms, one of which is absent chest pain, which is generally suggestive of chronic aortic dissection. In roughly twenty percent of patients who present with acute aortic dissection, the typical presentation is syncope devoid of a history of pain or neurological findings. Following a period of pain, cardiac failure may become the main symptom and is usually related to severe aortic regurgitation. Another complication is cardiac tamponade which may cause hypotension and syncope. Syncope, which can be caused by cardiac tamponade, may also be caused by severe pain, activation of baroreceptors or the obstruction of cerebral vessels.

As previously stated, we may see pulse deficits in the clinical presentation. Pulse deficits are caused from the destruction of the peripheral vessels due to the involvement of the vessel's origin within the dissection, or the elimination of the true lumen by the enlarging false lumen (4). However, the patient may present as relatively stable with sufficient perfusion in the event of blood redirection into the true lumen, which creates natural fenestration (6). Multiple branches of the aorta may be involved during aortic dissection. Involvement of the intercostal arteries may lead to the sudden development of paraplegia, oliguria or anuria may result with the extension to the renal arteries. Persistent abdominal pain, and the increase of acute phase proteins, as well as an increase in lactate dehydrogenase are indicators of celiac artery involvement, which is found in about eight percent and mesenteric artery involvement in eight to thirteen percent. If there is further dissemination occurring at a later time, it may typically cause the same acute pain to occur once again and is often linked to a worsening outcome (4).

## **1.6 Physical Examination of Aortic Dissection**

The physical examination, as well as the patients presenting symptoms, may help us to better assess the location of the dissection. It was found that of the patients in their seventies who had proximal aortic dissections, fifty percent had pulse deficits, however a current registry consisting of many patients with acute aortic dissection, found the presence of pulse deficits in less than twenty percent of patients. This suggests that due to the intimal flap's altering position, a

pulse deficit may be transitory. Neurological deficits, such as ischemic paresis or loss of consciousness, occur in forty percent of patients with proximal aortic dissection. Rarely, we may observe vocal cord paralysis, hemoptysis or hematemesis, superior vena cava syndrome, Horner's syndrome, upper airway obstruction due to compression, pulmonary embolism or signs of mesenteric or renal ischemia.

Findings also include diastolic murmur which would indicate aortic regurgitation which is observed in 50% of individuals with aortic dissection that is more proximal. The murmur may be weak, and a wide pulse pressure as well as other peripheral signs may be absent. There may also be pericardial involvement, which are: pericardial friction rub, paradoxical pulse or jugular venous distention are signs that warn for rapid surgical intervention.

Up to thirty percent of patients who were later found to have aortic dissection were initially suspected to have other conditions such as, acute coronary syndromes, non-dissecting aneurysms, pulmonary embolism, or aortic stenosis. Therefore, when patients present with acute onset of congestive heart failure, unexplained syncope, stroke, and acute ischemia of the extremities or viscera, the differential diagnoses should also be considered (4).

### **1.7 Imaging and Diagnostics of Aortic Dissection**

All patients presenting with sudden chest pain with suspicion of aortic dissection should first receive an ECG. It is found that nearly twenty percent of patients with Stanford A dissections have ECG findings consistent with acute ischemia or myocardial infarction. In one third of patients, a normal ECG is present, and most will have non-specific ST-T segment changes (4). Along with ECG it is also recommended to order a chest radiograph along with complete aortic imaging such as with TEE, MRI, or CT. The most common imaging method is contrast-enhanced spiral CT or CTA due to its high availability. The typical findings include a luminal displacement of intimal calcifications, intimal flap, and aortic dilation.

High-resolution aortic images can be given by TEE with the similar specificity and sensitivity to CT and MRI. Aortic dissection appears as an undulating intimal flap, dividing a true and false lumen. We can also look for many associated complications as well as evaluating ventricular function as well as regional wall motion for coronary dissection, the aortic valve, degree of AR severity, and for diagnosing pericardial effusion and cardiac tamponade. TEE also may differentiate clinical imitators of aortic dissection, as well as assessing the anatomy of thoracic

aorta to better guide surgical decision making (1). Published studies of the diagnosis of aortic dissection by TEE, helical CT, and MRI showed that the tests yield clinically equally reliable diagnostic values. TEE had a 99% sensitivity and 95% specificity, and MRI had a 98% sensitivity and 98% specificity.

Helical CT is the most widely used method for diagnosis, with a very high sensitivity and specificity. It was found to have the highest negative predictive value, making it the best test to rule out aortic dissection. MRI has the highest accuracy and sensitivity for detection of all types of dissection, except for class 3, which is only diagnosed with aortography (5).

MSCT is non-invasive, has a short imaging time, high accuracy and is relatively practical for aortic dissection diagnosis. The limitations of MSCT are that it shows poor performance in dissection of aortic root as well as some small dissections. Recent studies have confirmed that MSCT improves the accuracy of rupture site detection from fifty to ninety percent. The limitations, as mentioned earlier, are due to excessive heart rate, aortic valve regurgitation, coronary artery involvement, and motion artifacts interfering with MSCT imaging, particularly of an aortic root dissection (14).

## **1.8 Surgical Treatment**

### **1.8.1 Surgical Indications**

The purpose of surgical management in Stanford A (type I, II) aortic dissection, is the avoidance of rupture and pericardial effusion, which may evolve into cardiac tamponade; as well as to reduce aortic regurgitation and the avoidance of myocardial ischemia. In Stanford B (type III) dissection, the intent is to thwart aortic rupture. In class I dissection, the goal is to resect the intimal tear. Although in dissections limited to the ascending aorta, either including or excluding the aortic arch, it is possible to resection the entirety of the intimal flap. Complete repair of types A and B (type I-III) dissections are rarely achieved (4).

Surgery is suggested for all patients with acute and chronic type A dissections, unless there is a prohibited risk such as age, stroke, overall medical condition, development of renal, bowel, myocardial infarction, and comorbidities. Fenestration and stenting may be indicated in cases of mesenteric malperfusion prior to the surgical repair at the site of the dissection (2).

The surgical indications in patients with acute type B (type III) are aimed at the avoidance of life-threatening complications such as a quickly enlarging aortic diameter, intractable pain, and mediastinal or periaortic hematoma which are indications of aortic rupture. Dissections that occur in a previously detected aortic aneurysm is also deemed an emergency. The development of complications such as ischemia of limbs, intestines or kidneys may be indicative for catheter guided fenestration of the membrane consequently opening the true lumen of the segment of the abdominal aorta (4).

Patients with uncomplicated type B are typically treated medically, those with complicated dissections are treated with interventional or surgical procedures. Complicated dissections consist of those with: persistent pain, uncontrollable hypertension, evidence of aneurysmal expansion or rupture, or visceral, renal, or lower-extremity vascular compromise. Type B dissections that are treated medically are not ideal since long-term prognosis is affected by the possibility of an expanding aortic aneurysm. This is more probable if the patient's blood pressure and heart rate are not well controlled, or if the initial false lumen diameter is more than 40-45mm, as well as if the false lumen remains patent. Therefore, surgery or endovascular stenting may prove beneficial for uncomplicated type B dissections in order to inhibit expansion as well as improve the patient's long-term survival (2).

### **1.8.2 Surgical Treatment for Type A Dissections**

The goal of surgery in acute type A (type I and type II) dissection, is to prevent aortic rupture, pericardial tamponade and to relieve aortic regurgitation (4). Surgical therapy involves excision of the intimal tear, removal of the most diseased segment of aorta, obliteration of the false channel, reconstitution of the aorta directly or with interposition of a synthetic graft, and, if necessary, restoration of aortic valve competence (15). For the repair of an incompletely dissected aortic root, aortic valve resuspension can be used (1). A composite graft or a valve-sparing root replacement, known as a Bentall procedure, is to be performed for a destroyed aortic root (1,2). The entire dissected aorta is replaced in DeBakey type II dissections. For dissections that include the coronary ostia, CABG may sometimes be necessary during surgery (1). Deep hypothermic circulatory arrest, DHCA, should be used during any repair of type A dissection (2).

A small subset of type A dissections are when the entry tear is in the descending aorta and propagation occurs up to the ascending aorta in a retrograde fashion, also known as retrograde type

A. This subset occurs in ten to twenty-seven percent among DeBakey type III and from four to twenty percent among Stanford A dissections. Emergency surgery is the treatment of this subtype however it remains challenging and controversial. Graft replacement of only the ascending aorta retains the primary entry tear and consequently the postoperative risk of rupture. However, excision of the entry tear and replacement of both the ascending aorta and aortic arch is associated with high mortality and morbidity (15).

A different approach is required for acute type A dissections (type I, type II) in which the proximal aorta was previously dilated. This is including a significant number of individuals with Marfan's syndrome where the recommendation is the insertion of a composite graft, or an aortic valve plus ascending aortic tube graft. Bentall and De Bono previously described the original method, which stated that the coronary ostia are to stay in connection with the aorta and are then to be anastomosed to the ascending portion of the graft after valve implantation. However, the coronary ostia may be excised, in button form, before anastomosis if they are close to the aortic annulus or if the stiff aortic wall produces strain on the ostia when they are transferred to the graft. In the emergency situation, valve sparing operations or aortic root remodeling have, more recently, become recommended however, they are time consuming and more complicated than composite grafting (4).

In DeBakey type 1 dissections, the thoracic aorta may frequently undergo aneurysmal changes and is therefore responsible for considerable long-term mortality. The prevention of distal aortic degeneration would significantly improve the prolonged effects after vast type A dissection. For open aortic arch repair of DeBakey type 1 dissections, current clinical studies have shown the safety and efficacy of treating the thoracic aorta with antegrade stenting. This method is known as the endovascular stented elephant-trunk technique or the frozen elephant-trunk technique. For the prevention of prolonged aneurysmal worsening, urgent stenting during the acute dissection phase facilitates proper aortic remodeling. Due to some patients having a residual dissection after repair, all patients after should be treated with antihypertensive therapy (1).

### **1.8.3 Surgical Treatment for Type B Dissections**

It has been reported that for type B dissections, medical therapy is superior to surgical treatment (16). When treated medically, uncomplicated type B dissections presently have the greatest outcome (1). However, twenty to fifty percent of those patients who survive the acute

stage, develop aneurysms within one to five years after onset (16). The medical therapy is targeted for management of systemic hypertension in order to thwart aortic rupture and aortic aneurysm formation and extension. Medical therapy consists of mainly of combination therapy to maintain a blood pressure below 130/80 mm Hg this is achieved with a B-blocker, diuretic, angiotensin-converting enzyme inhibitor, or other antihypertensive medications (1).

The standard surgical interventions for complicated type B dissection, involve the resection of the intimal tear as well as interposition graft replacement in order to reapproximate the wall of the aorta (2). For both acute and chronic dissections of the descending aorta, the surgical management includes the replacement of affected sections with the proper tubular graft. The standard approach for placement of the graft is through a fifth rib posterolateral chest incision, this permits the replacement of the descending aorta to the point of the eighth intercostal space. Most of these operations are performed utilizing extracorporeal circulation by way of left heart bypass (4). In patients who require surgery due to life-threatening complication, TEVAR is the preferred alternative treatment to surgery for uncomplicated type B and is associated with lower risk of short-term mortality (1,17). It has lower major in-hospital complication rate, ranging from 3.4 % to 11.1 %, compared to the complication rate of forty percent with surgery.

TEVAR uses a stent graft which covers the primary entry tear, discontinues blood flow into the false lumen, expands the true lumen and depressurizes the false lumen preventing extension of dissection, and ideally leading to false lumen thrombosis with aortic remodeling. TEVAR also stabilizes the patient hemodynamically and reverses end-organ ischemia (17). TEVAR has been widely used for the last decade due to the lower mortality and morbidity compared with open chest surgical repair however, conservative methods still play an important role due to the increased risk of postoperative adverse events with TEVAR (18). High surgical mortality is due to rupture of the aorta and splanchnic ischemia, which are particularly serious in patients older than seventy, with a mortality rate of seventy percent (19).

## **1.9 Postoperative Complications**

It has been shown that seventy percent of postoperative death was due to cardiac failure, postoperative stroke, and hemorrhage (20). One of the main complications after aortic surgical procedures is acute kidney injury (21). Postoperative AKI is associated with an increased risk of

long-term mortality, as well as an association with thirty-day postoperative death in patients with type A dissection (20,21). Those who developed stage 3 AKI markedly increased both early and long-term all-cause mortality, an 11-fold and 4.46-fold higher risk. Preexisting renal impairment as well as renal malperfusion are also a factor for survival post type A dissection repair. Impaired renal function at baseline has been identified as a risk factor for AKI. Other factors that contribute the development of AKI are, hypertension, renal malperfusion, long bypass duration and sepsis (20).

Cerebral malperfusion is a major risk factor for the worsening of the early results observed in patients after surgical repair (22). Postoperative outcomes are worse in the presence of neurological deficits and malperfusion (20). Early cerebral revascularization may appear advisable however, patients with type A aortic dissection and neurological deficit will often present with severe hemodynamic instability. Antegrade or retrograde cerebral perfusion did not influence postoperative mortality or the incidence of neurological deficits. Therefore, early antegrade arterial reperfusion of the true lumen may reduce the duration of brain ischemia and limit the neurological deficits (22).

Most late deaths following primary surgery has been caused by rupture of the aorta which prevented by reoperation. Reoperation may be performed for two reasons the first, is due to aneurysmal formation (five to six cm in diameter) distant from the site of the earlier repair. An additional reason for reoperation may due to the improper repair during the first operation. The reoperation rates for type I and II dissections are around ten percent at five years and about forty percent at ten years after the first surgery for patients with Marfan's syndrome the risk is even higher. The most common sites which require reoperation are the aortic root and the ascending portion of the aorta. The primary causes are typically iatrogenic where either the dissection remained after the primary procedure, or it had returned after the surgical intervention. Other conditions that affect further dissection consist of congenitally deformed valves, prior aortic valve replacement, or an overlooked annuloaortic ectasia. Other causes may include root aneurysms, composite replacement or redissection after glue repair (4).

### **1.10 Chronic Aortic Dissection**

Chronic aortic dissection is deemed when a patient survives fourteen days following the initial inception of an acute aortic dissection. This is centered on autopsy findings which found

that seventy-four percent of patients who succumb to the aortic dissection do so within the first 2 weeks. Patients with chronic dissections who comprise the majority are those surviving emergency surgery as well as those who were treated with only medical therapy. A smaller group of individuals who either never sought medical care or during the acute phase went undiagnosed and untreated comprise the remainder of patients.

Patients whose dissections were restricted to the ascending aorta (DeBakey type II) may have been fully treated after emergency operation alone however, this only characterizes one-third of cases, the majority of the remaining cases extend distally to the subclavian artery. In a study of 208 patients who had undergone the repair of a proximal dissection between 1978 and 1995 at the Cleveland Clinic, had shown the thirty-day, five-year and ten-year survival to be eighty-seven percent, sixty-eight percent, and fifty-two percent respectively. In sixty-three percent of patients, a residual distal dissection was found with flow in the false lumen. However, once the proximal segment of the aorta is repaired, those who have a remaining distal dissection have a comparable survival to patients with type B dissection therefore, the management of these patients is equivalent to those for chronic type B dissections.

Hospital survival for uncomplicated type B dissection may be improved by primary medical therapy however, it has not improved long-term survival. The mortality is associated with comorbidity however, late complications from distal aortic dissection is expected to occur in twenty to fifty percent of patients. The complications include, most commonly, saccular or fusiform aneurysmal changes of the false channel, a new dissection that is related to new complications, and rupture of a weak false channel which may lead to rupture and exsanguination (23).



## **2. OBJECTIVES**

## **2.1 Aim of Study**

The aim of this study is to evaluate the most common postoperative complications of aortic dissection. In addition, this study also will evaluate the risk factors and predispositions associated with the development of aortic dissection.

## **2.2 Hypotheses**

- Male gender is a main risk factor.
- Hypertension is the main predisposing condition for aortic dissection.
- There is a seasonal (temporal) correlation with the development of an aortic dissection.
- Acute kidney injury is the most common postoperative complication.

### **3. MATERIALS AND METHODS**

### **3.1 Study Design**

This is a retrospective study which analyzed patient discharge records from the Department of Anesthesiology and Intensive Care Unit at the University Hospital Split, Croatia.

### **3.2 Data Extraction**

Discharge records were extracted from patient discharge records from the Department of Anesthesiology and Intensive Care Unit. There was a total of 42 discharge records, regarding patients with aortic dissection, however due to some patients having been readmitted to the department there are 38 patients in total. The present study was approved by the Hospital Ethics Committee of the University Hospital Centre in Split, Croatia (approval no. 2181-147/01/06/M.S.-22-02) and was performed in accordance with the ethical standards described in the 1964 Declaration of Helsinki and its later amendments.

### **3.3 Data Analysis**

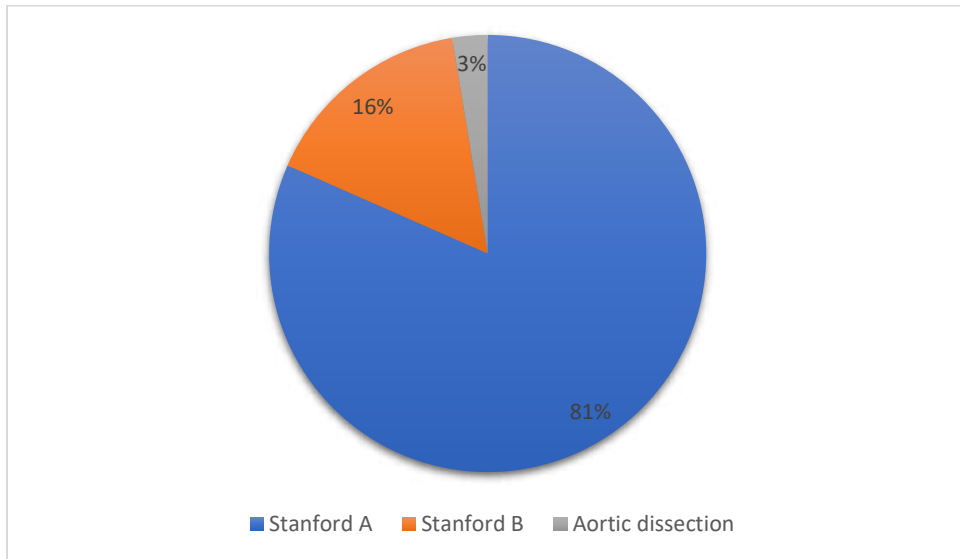
The data from the discharge records were categorized specifying classification of dissection, gender, age, comorbidities, postoperative complications, length of stay in ICU, time of the year in which the dissection occurred and finally outcome defined as released or death.

### **3.4 Statistical Analysis**

For the descriptive statistics we used Microsoft Excel, and for the statistical analysis we used MedCalc Statistical Software version 20.013 (MedCalc Software Ltd, Ostend, Belgium). Data has been tested for normality by D'Agostino-Pearson test. For the analysis of categorical variables, we used an independent t-test.

## **4. RESULTS**

The total number of patients admitted to the Department of Anesthesiology and Intensive care unit at the University Hospital of Split during the period of our study, from January 2019 to December 2021, was 38 patients. The total number of patients diagnosed with Stanford A aortic dissection was 31 (81.6%), those with Stanford B was 6 (15.8%) and one patient with aortic dissection unknown of which classification (Figure 2). The median age of patients presenting with aortic dissection was 66 with an interquartile range of 54-72. Patients were predominantly male with 31 (81.6%) whereas for females there were 7 (18.4%) patients.



**Figure 2.** Distribution of the aortic dissection classifications.

Table 1 embodies the descriptive statistics for patients presenting with aortic dissection in the postoperative period. As well as the statistical significance ( $P < 0.0001$ ) between age and the length of stay in the department.

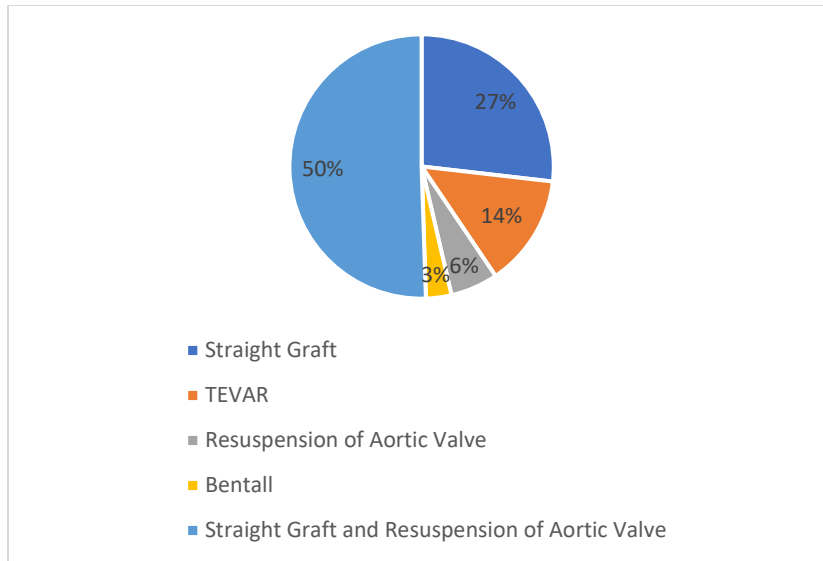
**Table 1.** Descriptive statistics for aortic dissection

<b>Parameter</b>	<b>Measurements</b>	<b>P-value*</b>
Male gender N (%)	31 (81.6)	
Female Gender N (%)	7 (18.4)	
Age in years (Md,IQR)*	66 (54-72)	<0.0001
Length of stay in days (Md,IQR)*	5 (3-15)	<0.0001
Chest pain N (%)	25 (65.8)	
Other symptoms N (%)	16 (42.1)	
Comorbidities of any kind N (%)	35 (92.1)	
Obesity N (%)	19 (50)	
Arterial Hypertension N (%)	32 (84.2)	
Released N (%)	30 (81.1)	
Deceased N (%)	7 (18.9)	

Data is presented as whole number (percentage)

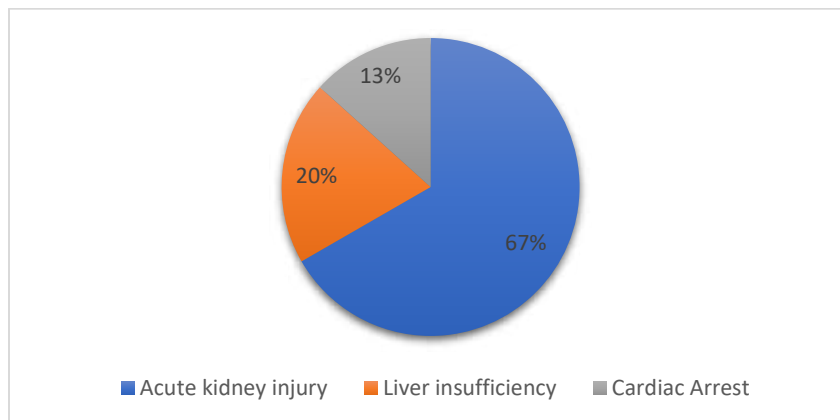
\*t-test for independent samples

We also evaluated the most common surgical procedures and found the one that was performed the most was placement of a straight graft with resuspension of the aortic valve, and the second most common was placement of a straight graft without resuspension of aortic valve (Figure 3).



**Figure 3.** Distribution of surgical procedures.

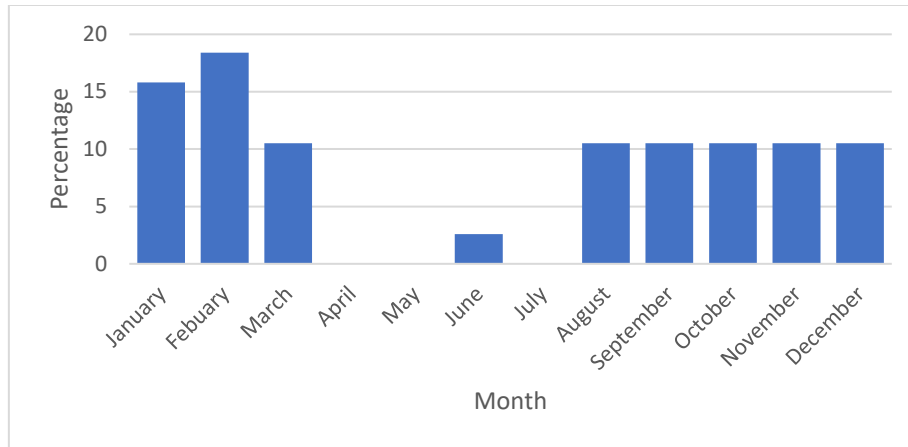
The total number of patients developing postoperative complications was 15. The most common complication is acute kidney injury and was present in 67% of patients whereas liver insufficiency was found in 20% (Figure 4).



**Figure 4.** Distribution of the postoperative complications.

The seasonal correlation between aortic dissection and the time of the year was analyzed and an uneven distribution was found. However, the winter months, as well as fall months, had the highest percentage of patients presenting with aortic dissection (Figure 5).





**Figure 5.** Distribution between the months of the year and the percentage of patients.

During further statistical analysis using Chi-squared test gender was found to have no statistical significance for complications ( $P=0.479$ ), diagnosis ( $P=0.543$ ), reoperation ( $P=0.211$ ), treatment ( $P=0.358$ ) or for the patients outcome ( $P=0.732$ ). The same statistical analysis was used to find the correlation between complications and diagnosis ( $P=0.130$ ), gender ( $P=0.479$ ) and outcome ( $P=0.233$ ) which were found to not be statistically significant however, reoperation ( $P=0.003$ ) and treatment ( $P=0.015$ ) were found to be statistically significant. Arterial hypertension was also analyzed to find the relationship between hypertension and the same observed elements. There was no statistical significance found between complications ( $P=0.896$ ), diagnosis ( $P=0.447$ ), gender ( $P=0.905$ ), outcome ( $P=0.879$ ), reoperation ( $P=0.950$ ) or treatment ( $P=0.129$ ) and arterial hypertension.

## **5. DISCUSSION**

Aortic dissection is a cardiac emergency which may come with many postoperative complications that may lead to life threatening conditions. Our study investigated the most common postoperative complications as well as the risk factors of the patients who were admitted to the Department of Anesthesiology and Intensive Care at the University Hospital center of Split during the year span from January 2019 to December 2021.

In our study the total number of patients diagnosed with aortic dissection was 38 with 31 of those being diagnosed with Stanford A dissection, 81.6%. Male gender had a larger predominance, 81%, over females, 18.4%, however, we found that gender did not carry any significance in terms of complications and postoperative outcomes. The median for the age at which patients presented was 66 and the interquartile range is (54-72). We also found that median for the length of stay was 5 days with an interquartile range of (3-15). After conducting an independent samples T-test a correlation was found between the age and length of stay at the department ( $P < 0.0001$ ).

The risk factors for the development of aortic dissection have been well defined to be male gender, age most commonly between 60s and 70s, history of hypertension as well as prior cardiac surgery, bicuspid aortic valve, and a history of Marfan syndrome (3). The postoperative complications have also been well investigated and understood, in a study done by Wenbin *et al*, they found that preoperative stage 3 AKI and perioperative acute renal failure was an independent predictor of in-hospital mortality in type A aortic dissection patients (21). The seasonal correlation for the development of aortic dissection has not been well investigated; however, studies would suggest that in hypertensive patients there is a 34% increase in the occurrence of aortic dissection in weather conditions with rising atmospheric temperatures (9).

Arterial hypertension was found in 84.2% of patients however, like gender, it did not show significance in further statistical analysis when finding the relationship to the observed elements. This differs from the study done by Kahlil *et al* which found that hypertension occurs in 72.1% of patients, and as stated in the current literature, is the most important risk factor for the development of aortic dissection (5).

When evaluating the seasonal correlation, we found that there is an uneven distribution in the occurrence of aortic dissection. However, February and January had the largest percentage of occurrences with 18.4% and 15.8% respectively. In the current literature it is described that there is an increase during winter, especially in January, and lower frequency in summer (9). Our study compares to the current literature as, aortic dissection was diagnosed more frequently in the winter

months and occurred only once in June, and 4 times in the late summer and fall months of August, September, October, November, and December.

As in previously conducted studies and, as in accordance with what is accepted in the current literature, we found acute kidney injury to be the most common postoperative complication. Acute kidney injury occurred in 26.32% of patients. The other complications that were also evaluated were liver insufficiency and reanimation occurring in 7.89% and 5.26% of patients respectively. The current literature states that there is a significant association between the development of postoperative acute kidney injury and long-term mortality (21).

Upon further statistical analysis using Chi-squared tests there was no correlation found between gender and the adverse outcomes of the patient, meaning that in this study both males and females are statistically likely to have the same observed elements. Outcome was also found to not have a correlation between the reoperation of the patient, diagnosis, gender, complications, or treatment of the dissection. Likewise, arterial hypertension was also found to not have correlations between the same observed elements, however as stated previously, arterial hypertension was found in most patients. Unlike, the correlation between complications, and reoperation and treatment were found to be statistically significant, which would mean the treatment and the reoperation of a patient have an association with the development of a postoperative complication.

The limitations of our study are due to the retrospective analysis of a small population at one center during a three-year time period. To better evaluate the risk factors and postoperative complications, a larger sample should be used. A larger sample would better evaluate the relationship between arterial hypertension, gender and outcome with the observed elements compared in this study. It would also ensure more accurate distribution of the gender as well as seasonal correlations with the development of aortic dissection, which we were not able to definitively show in our study. Despite the limitations we were able to describe the most important postoperative complication, acute kidney injury as well as the most common risk factor, arterial hypertension.

## **6. CONCLUSION**

We can conclude that males present more often with aortic dissection however, this is not statistically significant in the development of complications and outcome. Majority of patients have arterial hypertension however, like gender it does not show significance in other observed elements. Reoperation and the treatment of the aortic dissection has an impact on the development of postoperative complications. The study sample was too small to determine the seasonal correlation. And finally, we can conclude that acute kidney injury is the most common postoperative complication as well as a correlation between the age of the patient and the length of stay in the ICU.

## **7. REFERENCES**

1. Kaplan JA, Reich DL, Savino JS. Kaplan's cardiac anesthesia: The Echo Era. 6th ed. St. Louis: ELSEVIER; 2011. 657-62 p.
2. Bojar RM. Manual of perioperative care in adult cardiac surgery. 5th ed. West Sussex: Wiley-Blackwell; 2011. 43-8 p.
3. Criado F J, Coselli J S. Aortic dissection: a 250-year perspective. *Tex Heart Inst J.* 2011;38:694-700.
4. Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A et al. Diagnosis and management of aortic dissection. *Eur. Heart J.* 2001;22:1642-81.
5. Khalil A, Tarik T, Porembka D. Aortic pathology: aortic trauma, debris, dissection, and aneurysm. *Crit Care Med.* 2007;35:392-400.
6. Gawinecka J, Schonrath F, von Eckardstein A. Acute aortic dissection: pathogenesis, risk factors and diagnosis. *Swiss Med Wkly.* 2017. doi: 10.4414/smw.2017.114489.
7. Chen FT, Chou AH, Chan YH, Wu VCC, Lin CP, Hung KC et al. Sex-related differences on the risks of in-hospital and late outcomes after acute aortic dissection: A nationwide population-based cohort study. *PLoS One.* 2022;17: doi: 10.1371/journal.pone.0263717.
8. Meccanici F, Gokalp AL, Thijssen CGE, Mokhles MM, Bekkers J, van Kimmenade R et al. Male-female differences in acute thoracic aortic dissection: a systematic review and meta-analysis. *Interact CardioVasc Thorac Surg.* 2022;34:616-27.
9. Henzi D, Platzmann A, Brtek J, Holubec T, Emmert MY, Vogt P et al. Increasing atmospheric temperature implicates increasing risk for acute type A dissection in hypertensive patients. *J Thorac Dis.* 2021;13:5799-806.
10. Robinson PN, Arteaga-Solis E, Baldock C, Collod-Beroud G, Booms P, De Paepe A et al. The molecular genetics of marfan syndrome and related disorders. *J Med Genet.* 2006;43:769-87.
11. Meester JAN, Verstraeten A, Schepers D, Alaerts, Van Laer L, Loeys BL. Differences in manifestations of marfan syndrome, ehlers-danlos syndrome, and loeys-dietz syndrome. *Ann. Cardiothorac. Surg.* 2017. doi: 10.21037/acs.2017.11.03.
12. Siu SC, Silversides CK. Bicuspid aortic valve disease. *J Am Coll Cardiol.* 2010;55:2789-800.
13. Li Y, Zhang N, Xu S, Fan Z, Zhu J, Huang L et al. Acute type A aortic intramural hematoma and type A aortic dissection: correlation between the intimal tear features and pathogenesis. *Quant Imaging Med Surg.* 2020;10:1504-214.



14. Huang F, Wu H, Lai QQ, Ke XT. Application value of preoperative dual-source computed tomography in assessing the rupture site of thoracic aortic dissection. *J Cardiothorac Surg.* 2021;16:346.
15. Dake M, Wang D. Will stent-graft repair emerge as treatment of choice for acute type B dissection. *Semin Vasc Surg.* 2006;19:40-7.
16. Czermak B, Waldenberger P, Fraedrich G, Dessi A, Roberts K, Bale R et al. Treatment of stanford type B aortic dissection with stent-grafts: preliminary results. *Radiology.* 2000;217:544-50.
17. Bedi V S, Swain P, Yadav A. Medical therapy versus TEVAR for uncomplicated type B aortic dissection. *Indian J Thorac Cardiovasc Surg.* 2019;35:174-8.
18. Liu J, Xia J, Yan G, Zhang Y, Ge J, Cao L. Thoracic endovascular aortic repair versus open chest surgical repair for patients with type B aortic dissection: a systemic review and meta-analysis. *Annals of Medicine.* 2019;51:360-70.
19. Bortone A, De Cillis E, D'Agostino D, Schinosa L. Endovascular treatment of thoracic aortic disease four years of experience. *Circulation.* 2004;110:262-7.
20. Toh S, Choon Men Yew D, Choong JJ, Chong TL, Harky A. Acute type A aortic dissection in-hours versus out-of-hours: A systematic review and meta-analysis. *J Card Surg.* 2020;35:3432-9.
21. Wenbin M, Rui L, Lihua E, Nashunbayaer Z. Postoperative acute kidney injury and early and long-term mortality in acute aortic dissection patients. *Medicine.* 2021;100:1-6.
22. Vendramin I, Isola M, Piani D, Onorati F, Salizzoni S, D'Onofrio A et al. Surgical management and outcomes in patients with acute type A aortic dissection and cerebral malperfusion. *JTCVS open.* 2022. doi: 10.1016/j.xjon.2022.03.001.
23. Bavaria J, Coselli J, Curi M, Eggbrecht H, Elefteriades J, Erbel R et al. Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts. *Ann Thorac Surg.* 2008;85:1-41.

## **8. SUMMARY**

**Objectives:** The main purpose of this study was to identify the risk factors, including gender, comorbidities, and seasonal correlation, with the development of aortic dissection. We also aimed at determining the most common postoperative complications.

**Materials and methods:** We conducted a retrospective study analyzing the records of 38 patients who had been admitted postoperatively to the Department of Anesthesiology and Intensive Care Unit at the University Hospital of Split during the period of January 2019 to December 2021. The collected data was analyzed using Microsoft Excel for the descriptive data, while for the statistical analysis, we have used MedCalc Statistical Software version 20.013 (MedCalc Software Ltd, Ostend, Belgium).

**Results:** Most of the patients were male (81.6%). The mean deviation for age was 66 years. The mean deviation for the length of stay in the ICU was 5 days. The correlation between the age of the patient and the length of stay in the ICU was statistically significant ( $p < 0.0001$ ). The majority of patients (84.2%) had a history of arterial hypertension. The seasonal correlation was found to have uneven distribution. The most common postoperative complication was found to be acute kidney injury occurring in 26.32% of patients.

**Conclusion:** We concluded that males present more often with aortic dissection. Patients more often have a history of comorbidity such as arterial hypertension. Acute kidney injury is the most common postoperative complication. The age of the patient correlates with the length of hospital stay. However, the sample size was too small to determine the statistical significance between the risk factors and the postoperative outcomes, a larger study is needed for further evaluation.

## **9. CROATIAN SUMMARY**

**Naslov:** Disekcija aorte na kardialnom JILu: Retrospektivna analiza ishoda i postoperativnih komplikacija.

**Cilj:** Glavna svrha ove studije bila je identificirati čimbenike rizika, uključujući spol, komorbiditete i sezonsku korelaciju, s razvojem disekcije aorte. Kao i utvrđivanje najčešćih postoperativnih komplikacija.

**Materijali i metode:** Proveli smo retrospektivnu studiju kojom smo analizirali povijest bolesti od 38 pacijenata koji su bili postoperativno primljeni na Odjel anesteziologije i intenzivnog liječenja KBC-a Split u razdoblju od siječnja 2019. do prosinca 2021. Prikupljeni podaci analizirani su pomoću statističkog softvera Microsoft Excel i MedCalc Statistical Software verzija 20.013 (MedCalc Software Ltd, Ostend, Belgium).

**Rezultati:** Većina bolesnika bili su muškarci (81,6%). Prosječna devijacija za dob bila je 66 godina. Prosječna devijacija za dužinu boravka na JIL-u bila je 5 dana. Korelacija između dobi bolesnika i duljine boravka na JIL-u bila je statistički značajna ( $p < 0,0001$ ). Većina bolesnika (84,2%) imala je anamnezu arterijske hipertenzije. Utvrđeno je da je sezonska korelacija neravnomjerno raspoređena. Utvrđena je najčešća postoperativna komplikacija akutne ozljede bubrega u 26,32% bolesnika.

**Zaključak:** Zaključili smo da se muškarci češće javljaju s disekcijom aorte. Bolesnici češće imaju anamnezu komorbiditeta poput arterijske hipertenzije. Akutna ozljeda bubrega najčešća je postoperativna komplikacija. Dob bolesnika korelira s duljinom boravka u bolnici. Međutim, veličina uzorka bila je premala da bi se odredila statistička značajnost između čimbenika rizika i postoperativnih ishoda, potrebno je veće istraživanje za daljnju procjenu.

## **10. CURRICULUM VITAE**

**Personal information**

Name: Nicole Ana Kuric

Date of Birth: 12.05.1996

Place of Birth: Long Beach, California

Nationality: American/Croatian

Address: Long Beach, California 90808

E-mail: [kuric.nicole@yahoo.com](mailto:kuric.nicole@yahoo.com)

**Education:**

10.2016 - DD: University of Split School of Medicine, Croatia

09.2014 – 05.2016: Biology, California State University, Long Beach

08. 2010 – 05.2014: Saint Joseph Highschool

**Languages:**

English (Native language)

Croatian