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RESEARCH ARTICLE

ACUTE AORTIC DISSECTION TYPE A EXTENDED, COMPLICATED EXTENSIVE STROKE ABOUT A CASE, AND LITERATURE REVIEW

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Abstract

Patients with acute type A aortic dissection presenting with stroke: should we operate on them? Stroke is a severe complication in the early stages of acute type A aortic dissection and is associated with a dismal prognosis. The surgical treatment of these patients remains controversial. We herein report a challenging clinical presentation of an acute type A aortic dissection revealed by a major brain injury. We further review the literature related to the management of this dreaded association. Recent clinical series show favorable outcomes of early surgical repair in selected patients.

Introduction: -

Acute aortic syndromes (AAS) refer to a group of conditions affecting the thoracic aorta, posing an immediate threat to life and requiring early and urgent medical-surgical management. They include aortic dissection (AD), intramural hematoma, penetrating atherosclerotic ulcer, and aortic rupture.

Acute aortic dissection is a diagnostic and therapeutic emergency that jeopardizes the patient's life, with possible and dreaded complications, especially cardiac (tamponade) or neurological, making the prognosis unfavorable without prompt and tailored treatment. The use of imaging in diagnosis, advances in surgery, and resuscitation have improved the outlook for this condition [2], as potential complications, particularly cardiac (tamponade) or neurological, can be addressed more effectively with early and rapid intervention

According to the International Registry for Acute Dissection (IRAD), acute Stanford type A aortic dissections (DAAA) often complicate with an initial phase of ischemic stroke, presenting as an inaugural coma. Several mechanisms, either isolated or combined, explain this symptomatology, primarily affecting the central nervous system [1]. However, the initial management during the onset of neurological deficits remains controversial, balancing the risk of aggravating neurological injuries during early surgery under extracorporeal circulation with high-dose anticoagulation against the risk of spontaneous mortality in case of delayed intervention [2]. The prognosis of this lesion association is grim, with hospital mortality being 2 to 3 times higher than in patients without neurological signs [1,2]. Nevertheless, medical treatment is always recommended, while surgical or endovascular treatment is indicated immediately for DAAA with an inaugural stroke, which is associated with operative mortality below 10% and neurological deficit recovery in over 50% of cases, according to recent studies [4,5].

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Based on the observation of a case of DAAA complicated by ischemic stroke and postoperative vigilance disorders, we present a literature review on the management of this severe lesion association.

Observation: -

During my time as an associate intern at Joigny Hospital Center (CHJ), while on duty in the emergency department at CHJ, I received a 62-year-old man with a history of untreated hypertension, paroxysmal atrial fibrillation without anticoagulation due to low thromboembolic risk, and a penicillin allergy. He was admitted for acute chest pain accompanied by a sudden onset of malaise, with no prodromal symptoms, and complicated by a non-traumatic fall.

Clinically, the patient was conscious but in pain, with generalized pallor. He presented with acute, intense retrosternal chest pain radiating to the back, suggestive of a tearing type of pain. There was also an asymmetrical blood pressure with the left upper limb showing 64/35 mmHg and the right upper limb showing 127/51 mmHg. Signs of peripheral hypoperfusion were evident, such as cold extremities with a capillary refill time of more than 3 seconds. There were no sensory or motor deficits, and the Glasgow Coma Scale score was 15 (E4 V5 M6).

Given the acute presentation of tearing-type chest pain with dorsal radiation, asymmetrical blood pressure, and malaise, an emergency cerebral CT scan was performed, which showed no abnormalities [figure 1]. Further evaluation with an angioscan of the thoracic aorta revealed an extensive acute aortic dissection of Stanford type A (DAAA) involving the brachiocephalic trunk (TABC) from its origin to the descending thoracic aorta, the right common carotid artery, the left subclavian artery, and the ostium of the left vertebral artery [figure 2]. A thoracoabdominal-pelvic angioscan showed an acute type A aortic dissection with the entry point on the ascending aorta, at the base of the TABC. The dissection extended anterogradely to the aortic arch and the descending thoracic aorta. The abdominal aorta was dissected without signs of visceral malperfusion, extending to the right external iliac artery, without thrombus or abdominal-pelvic collection [figure 3]. Trans-thoracic echocardiography showed dilation of the aortic root with the ascending aorta measuring 40 mm and moderate aortic insufficiency, with no pericardial effusion [figure 4].

After hemodynamic stabilization with low-dose norepinephrine, the patient was urgently transferred to the cardiovascular surgery center at Dijon University Hospital (CHU Dijon) for a type A aortic dissection repair. The procedure involved replacing the ascending aorta by placing an extended supra-coronary tube in the hemiarch of the aorta and suturing a tear at the left coronary cusp under cardiopulmonary bypass (CPB) with a cerebral protection time of 20 minutes, a CPB time of 109 minutes, and an aortic clamping time of 67 minutes. The end of the surgery was marked by diffuse bleeding at the operative site, necessitating the administration of fibrinogen and 2 units of packed red blood cells (CGR) before transferring the patient to the cardiovascular intensive care unit..

Postoperatively, the patient remained stable hemodynamically, with an average mean arterial pressure of 65 mmHg after volume replacement with macromolecules and discontinuation of norepinephrine. However, an examination revealed left hemiplegia with a moderate NISHH score. A cerebral perfusion CT scan confirmed the occurrence of an extensive ischemic stroke affecting the superficial sylvian branch, with a thrombus present within the right M1-M2 branches, resulting in an estimated infarct volume of 130 ml [figure 5]. A follow-up angioscan of the thoracic aorta showed the persistence of an extensive intimal flap in the brachiocephalic trunk, the proximal end of the right common carotid artery, the left subclavian artery, and the ostium of the left vertebral artery [figure 6]. Due to the advanced time since the event and the established appearance on the scan, there was no indication for thrombolysis or mechanical thrombectomy. Similarly, prophylactic decompressive craniotomy was not recommended considering the risk of malignant middle cerebral artery infarction.

Cardiac echocardiography indicated a preserved left ventricular ejection fraction (FEVG) with no kinetic abnormalities, a non-dilated and slightly hypertrophied left ventricle, an average cardiac output of 6-6.5 l/min, no significant aortic or mitral valve pathology, normal right ventricle size and function with no pulmonary arterial hypertension (HTAP), and a non-respiratory inferior vena cava measuring less than 18 mm. There was no pericardial effusion.

On the rhythm front, the ECG remained in regular sinus rhythm around 75 bpm until March 30th when it switched to atrial fibrillation with a ventricular rate of 140-150 bpm, which was hemodynamically well-tolerated. The patient was started on antiarrhythmic treatment with amiodarone, with an initial loading dose of 3 tablets per day for 10 days, successfully reduced to 5 mg of bisoprolol daily at day 3. During the hospital stay, the patient experienced an

episode of transient ST-segment elevation, concave upwards, and without systemic pattern, likely related to concomitant neurological distress.

Regarding laboratory results, the patient's hemoglobin stabilized at 9.9 g/dl, troponin levels normalized after peaking at 3769 ng/l, and N-terminal pro-brain natriuretic peptide (Nt pro-BNP) levels increased to 1080 pg/ml. The patient also presented with a persistent fever of 38.5°C refractory to paracetamol. Repeated infectious samples were taken, with negative blood cultures except for one flask testing positive for coagulase-positive staphylococci and a fibroscopy test positive for methicillin-sensitive Staphylococcus aureus (MSSA). Due to the patient's penicillin allergy, the patient was put on cefotaxime for treatment.

During the course of the patient's stay, there was a sudden onset of circulatory collapse with exanthema on the limbs and trunk, and a granular appearance of the skin, indicative of an anaphylactic shock. The condition responded well to adrenaline, and after clinical and hemodynamic stabilization, the patient was transferred to the Intensive Care Unit (ICU) at Joigny Hospital Center. He remained hospitalized for one month before being transferred to the rehabilitation center for further psychomotor rehabilitation.

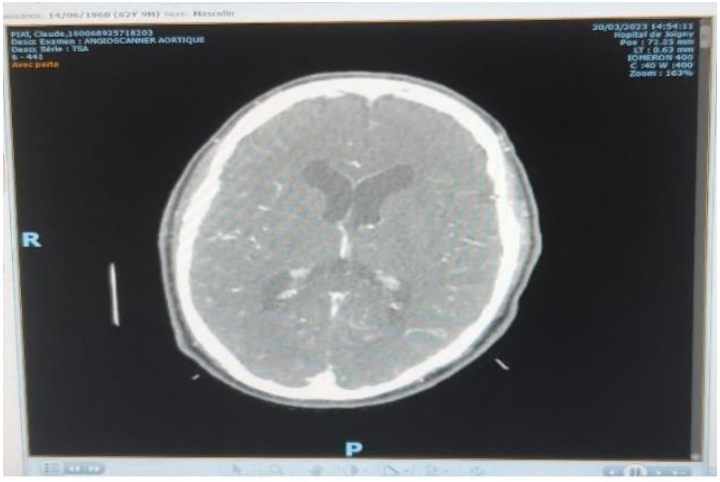


Figure 1:- Cerebral TDM emergency CH the Joigny , absence of ACVI sign.

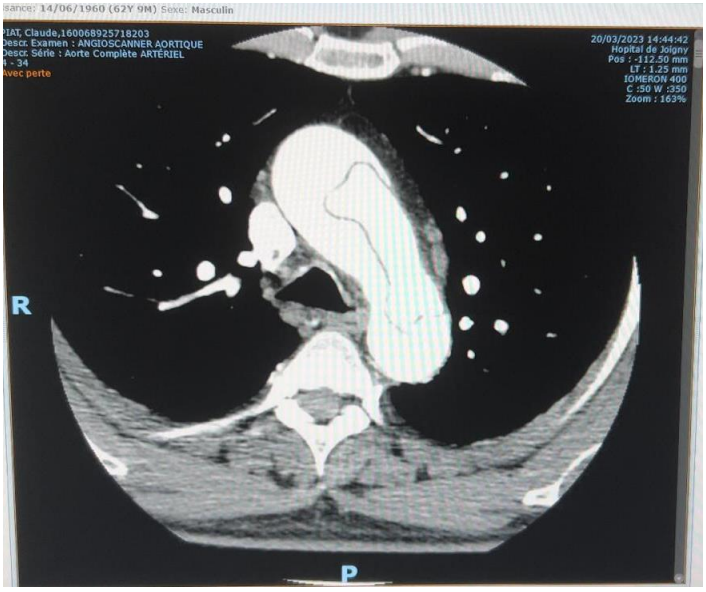


Figure 2:- CT angiography of AST in the emergency room of Joigny: shows an acute dissection extended from the brachiocephalic truncus arteriosus (TABC) from its birth to the descending thoracic aorta, the right common carotid artery, and left subclavian artery, and to the ostium of the left vertebral artery.

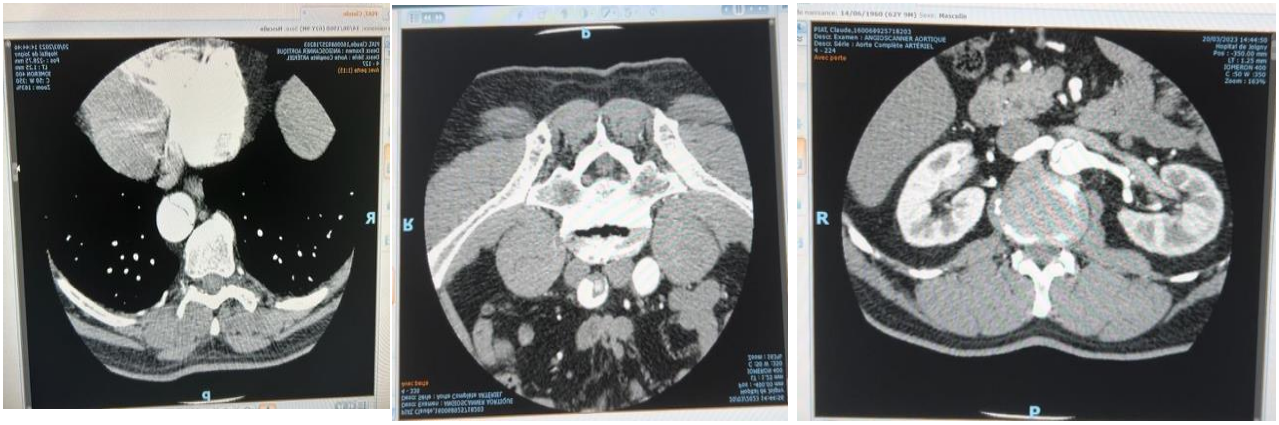


Figure 3:- CT angiography of the abdominal aorta in the Joigny emergency room: The dissection extends anterogradely to the descending thoracic aorta, the abdominal aorta is dissected without poor visceral perfusion, up to the right external iliac artery, without thrombus or abdominopelvic collection constituted.

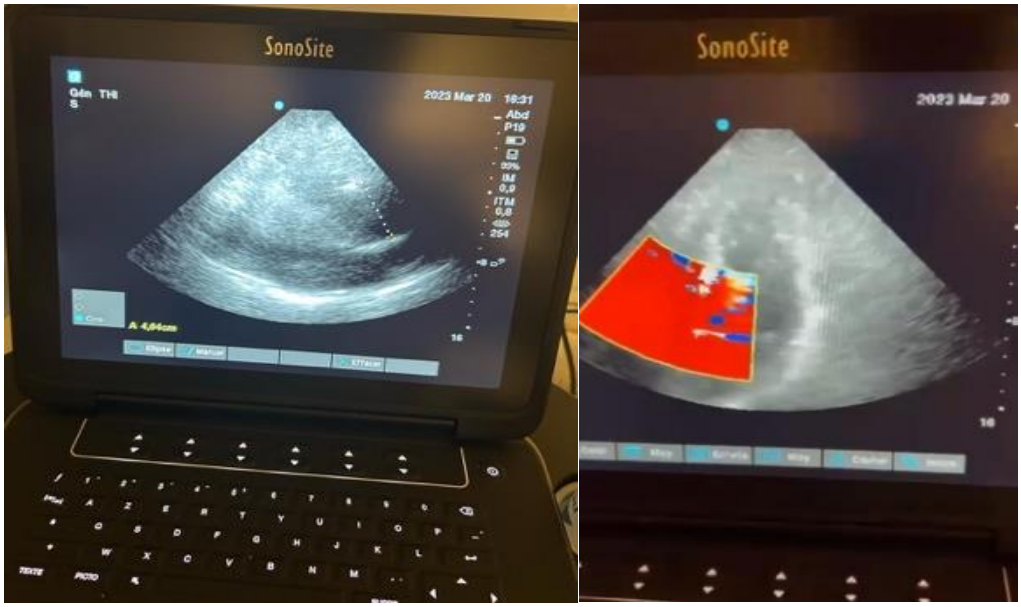


Figure 4:- Emergency ETT flash: reveals AA dilatation with aortic regurgitation.

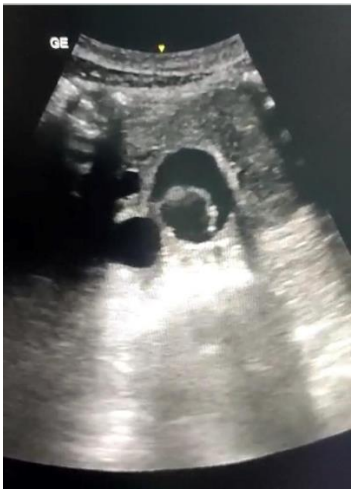


Figure 5:- Emergency ETT flash: intima flap.

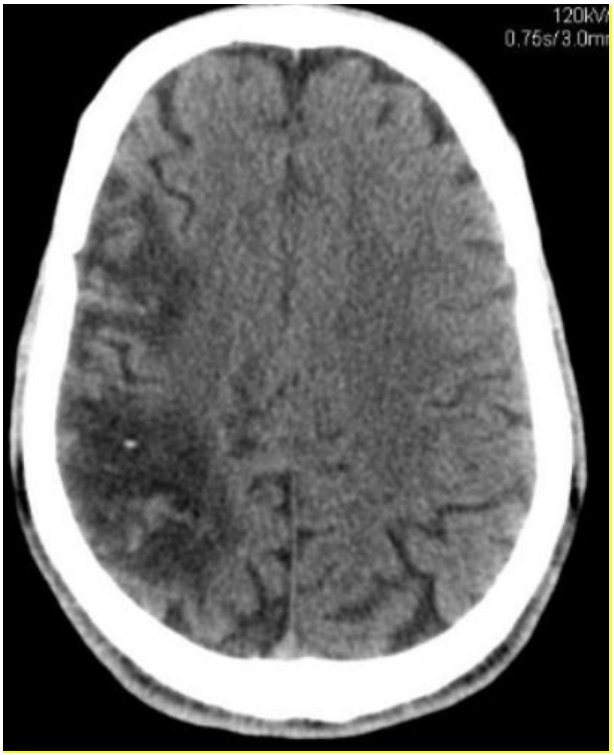


Figure 6:- TDM postoperative cerebral vascular accident, shows an extensive superficial right sylvian ischemic stroke consisting of.

Discussion:-

Definitions:

Aortic dissection is defined as acute within the first two weeks after symptom onset and becomes chronic beyond the second week. The International Registry of Acute Aortic Dissection (IRAD) proposed a classification of aortic dissection into four temporal types: hyperacute (<24 hours), acute (2-7 days), subacute (8-30 days), and chronic (>30 days) [1,6]. The most contemporary temporal classification system, proposed by the Society for Vascular Surgery (SVS) and the Society of Thoracic Surgeons (STS), similarly divides aortic dissection into four temporal types, as shown in Table 1, to improve prognosis and guide decision-making regarding the timing and types of potential interventions.

Aortic dissection results from a tear in the aortic intima, exposing the medial layer to pulsatile blood flow. The progressive separation of the layers of the aortic wall leads to the formation of a false lumen (false channel) (Figure 5).

Chronocite	Time elapsed since symptoms appeared
Hyperacute	<24H
Acute	1-14 D
Subacute	15-90 D
Chronic	>90 days

Tableau 1:- Classification of aortic dissection chronicity according to SVS/STS 2020 reporting standards.

Pathophysiology and Classification :

The aorta is the largest artery in the body, with high pressure, and it distributes blood throughout the entire organism via its various branches and collaterals. Its wall is similar to all the arteries in the body, composed of three tunics: the innermost tunic called "intima," which is in direct contact with the circulating blood, the middle tunic called "media," which is the thickest layer of the three and provides elasticity to the arteries, and the outer tunic called "adventitia," which protects the artery and anchors it to the surrounding structures [Figure 7].

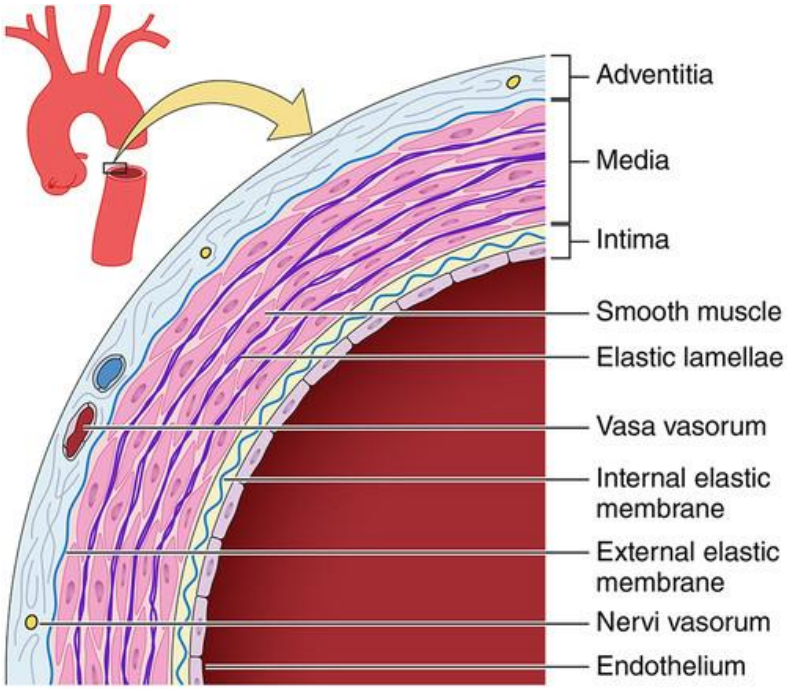
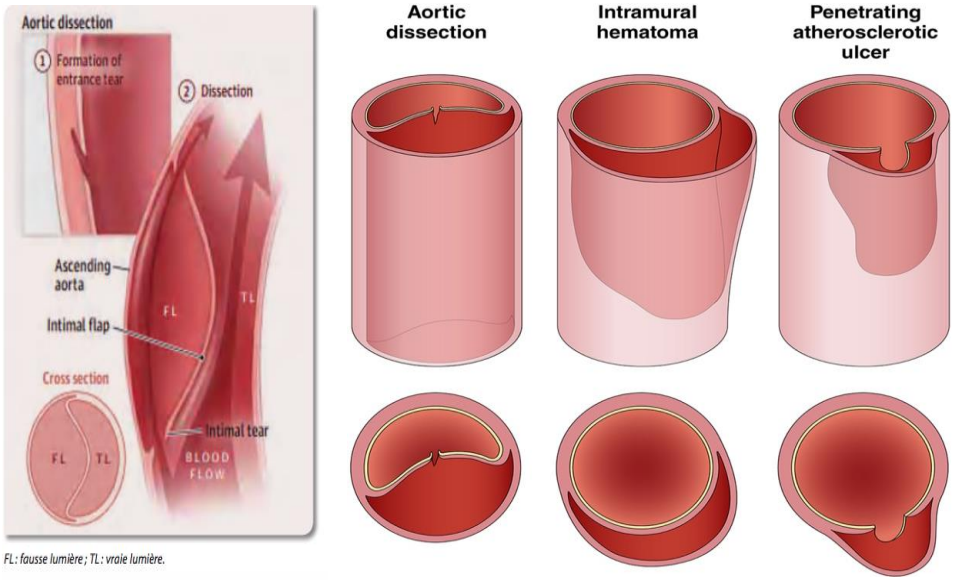


Figure 7:- Schematic illustrating the main histological components of the aortic wall.

Aortic dissection results from a tear in the aortic intima, which exposes the medial layer to pulsatile blood flow. The progressive separation of the layers of the aortic wall leads to the formation of a false lumen (false channel), and its further propagation can result in either an aortic rupture, which is rapidly fatal, or a re-entry into the true lumen through another intimal tear, creating a natural fenestration [Figure 8].

The false lumen may become a cul-de-sac at risk of thrombosis. In rare cases, when thrombosis occurs very early, the thrombosed false lumen is smaller than the true lumen. When thrombosis occurs later, the false lumen is usually larger than the true lumen, causing compression and leading to dynamic systemic malperfusion. The dissection can also extend to the aortic branches, resulting in systemic malperfusion and increasing the risk of mortality, particularly if the coronary arteries are involved [7].

Figure 8:- Acute aortic syndromes and pathogenesis of aortic dissection.



The classification of acute aortic dissection (AAD) is based on the anatomical location of the initial tear. There are two commonly used anatomical classification systems for aortic dissection (Figure 9): the De Bakey system and the Stanford system.

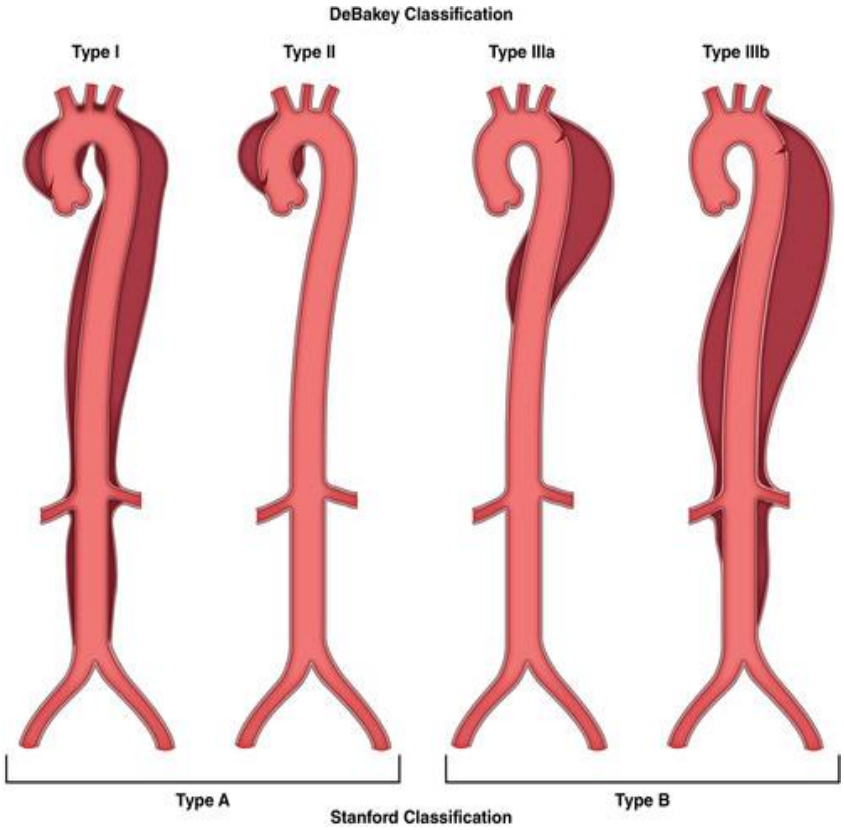


Figure 9:- Classification of acute aortic dissection.

The DeBakey system classifies dissections into types I, II, and III, based on the origin of the intimal tear and the extent of the dissection:

●	**Type I : dissection tear starts in the ascending aorta and propagates distally to include the aortic arch and generally the descending aorta**
●	**Type II : dissection tear is limited only to the ascending aorta**
●	**Type III : dissection tear starts in the descending thoracic aorta and most often spreads distally** ○ **Type IIIa : la déchirure de dissection se limite uniquement à l'aorte thoracique descendante** ○ **Type IIIb : dissection tear starts in the descending thoracic aorta and extends below the diaphragm**

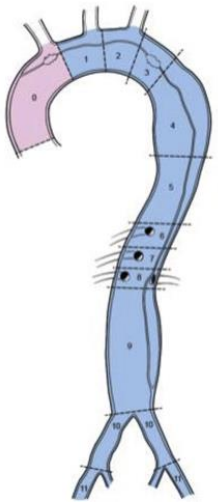
The Stanford classification system divides dissections into 2 categories depending on whether the ascending aorta is reached or not, regardless of the site of origin:

●	**Type A : Toutes les dissections impliquant l'aorte ascendante, quel que soit le site de la déchirure intimale**
●	**Type B : All dissections that do not involve the ascending aorta (including dissections that involve the aortic arch but spare the ascending aorta)**

In 2019, the European Association for Cardio-Thoracic Surgery and the European Society for Vascular Surgery published a consensus document by experts [8] for the treatment of thoracic aortic pathologies. In this document,

they introduced a third category called "non-A-non-B dissection," to be used in patients whose proximal dissection flap begins in the aortic arch.

More recently, in 2020, the Society for Vascular Surgery (SVS) and the Society of Thoracic Surgeons (STS) proposed an entirely new classification scheme that defines the anatomy of aortic dissection in a more detailed manner [9]. Dissections are anatomically defined based on the location of intimal tears and the proximal and distal extent of the dissection process (Figure 10).



Type	Proximal Extent	Distal Extent
A_D Entry tear: Zone 0	0	0
	1	1
	2	2
	3	3
B_{PD} Entry tear: ≥Zone 1	4	4
	5	5
	6	6
	7	7
	8	8
I_D Unidentified entry tear involving Zone 0	9	9
	10	10
	11	11
	12	12

Figure 10:- Anatomical aortic dissection reporting based on the 2020 SVS/STS reporting standards [9].

The A-DD classification indicates that Type A is used for any dissection with an entry tear in zone 0 and extends distally to the area designated by the index D. Type B is used for any dissection with an entry tear in zone 1 or beyond, and the proximal and distal extents of the dissection are designated by the indices P and D, respectively (e.g., B39). I-DD is used when a dissection starts in zone 0, but the location of the entry tear is not identified; it will be considered "Indeterminate" and denoted by an I, with its distal extent noted by the index D (e.g., I 399) [9].

Pathophysiology of Mal perfusion syndrome

The malperfusion syndrome is defined as the presence of ischemia in end organs due to inadequate perfusion from the aortic branches. The relationship between the true and false lumens in an aortic dissection plays a crucial role in maintaining stable perfusion to the end organs. Initially, the true lumen collapses due to the loss of transmural pressure across the dissection flap and subsequent elastic recoil of the medial smooth muscle. At the same time, the false lumen immediately dilates due to reduced elastic recoil, the depth of the dissection plane within the media, and the percentage of the wall circumference involved. All aortic branches are at risk of malperfusion as the false lumen expands and compresses the true lumen, which can occur simultaneously in multiple vascular beds as the dissection propagates distally [Figure 9]. When the dissection tear extends into the vessel itself and creates stenosis or thrombosis in the artery, static obstruction occurs [Figure 11] [10].

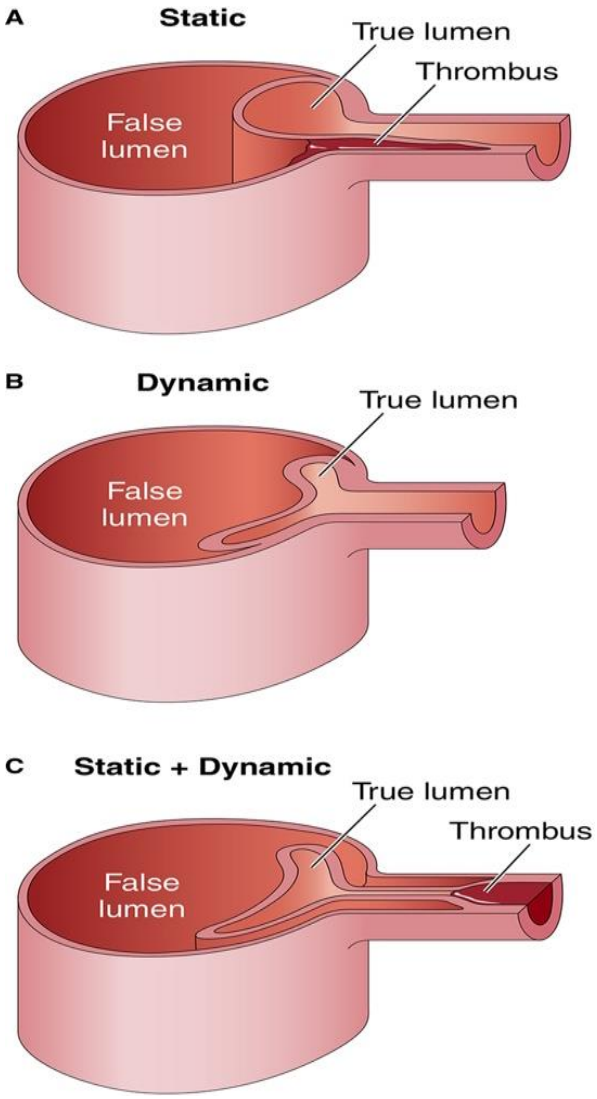


Figure 11:- Mechanisms of dynamic and static obstruction in aortic dissection [10].

(A) Static obstruction occurs when the dissection flap extends from the aortic lumen into the ostium of the affected branch vessel, leading to localized thrombosis of the false lumen within the branch. This thrombosis narrows or compresses the true lumen of the branch, thereby impairing perfusion to the distal branch.

(**B**) Dynamic obstruction occurs when the false lumen becomes persistently pressurized and compresses the true lumen, pushing the dissection flap against the ostium of the affected branch vessel, significantly reducing or obstructing its flow.

(**C**) Sometimes, a secondary vessel may suffer from both static and dynamic obstruction.

Facteurs de risque de DAA :

Several risk factors are involved in the development of aortic dissection (Table II). Men have a higher risk of developing aortic dissection than women, with an age-adjusted incidence of 5 versus 2 per 100,000 person-years [11]. Although women are less frequently affected, their prognosis is worse. The gender distribution is consistent in both type A and type B dissections. Women with type A dissection also have a higher in-hospital mortality rate, which may be attributed to either their older age, delayed presentation to the hospital, or late diagnosis due to less typical symptoms [12]

Male Gender
Age > 65 years
High blood pressure

Smoking
Aneurysm
Congenital disorders
Marfan syndrome
Loeys-Dietz syndrome
Vascular Ehlers-Dqnlos syndrome
Bicuspid aortic valve
Inflammatory disease
Aortitis
Giant cell arteritis
Takaysu arteritis
Systemic lupus erythematosus

Tableau II:- Risk factors for aortic dissection.

The incidence of aortic dissection is correlated with age, with the average age of onset being around 65 years. Generally, patients with type A dissection are younger than those with type B dissection [12,13]. However, in patients with connective tissue disorders or bicuspid aortic valve, aortic dissection often occurs before the age of 40 [12].

Arterial hypertension is considered a major risk factor for aortic dissection, affecting approximately 80% of patients. The incidence rate of aortic dissection in hypertensive patients is 21 per 100,000 person-years, compared to 5 in normotensive individuals [13,14]. Patients with high blood pressure during the five years before the onset of aortic dissection (either undiagnosed or poorly controlled) have a higher mortality rate compared to well-controlled normotensive or hypertensive patients [14].

Smoking is another risk factor for developing aortic dissection and/or aortic aneurysm. Smokers are twice as likely to develop thoracic aortic dissection and five times more likely to develop abdominal aortic aneurysm compared to non-smokers [14].

The annual rate of aortic dissection increases progressively with aortic diameter, and the incidence of aortic complications reaches 30% when the aortic diameter reaches 60 mm [15]. Although aortic dilatation significantly increases the risk of aortic dissection, it is not mandatory for a dissection to occur; approximately 60% of acute type A aortic dissections occur at aortic diameters < 55 mm [16].

Marfan syndrome and other hereditary connective tissue disorders such as Ehlers-Danlos syndrome or Loeys-Dietz syndrome are associated with an increased risk of developing aortic dissection, especially in young patients [17]. The prevalence of type A dissection in patients with Marfan syndrome at the age of 60 is approximately 50% [18]. Patients with Loeys-Dietz syndrome have a high risk of aortic dissection or rupture at an early age and with aortic diameters that are not typically predictive of these events [19].

Bicuspid aortic valve is the most common congenital malformation, occurring in 1 to 2% of the general population, and is a risk factor for aortic dissection and aortic aneurysm. In this population, the prevalence of aortic dilatation and aneurysm increases with age, and the risk of acute aortic complications, such as dissection and rupture, is eight times higher in these patients compared to the general population. In the emergency setting, patients with genetic diseases and vasculitis are rare. Since these patients are underrepresented in diagnostic studies, they should be treated as highly suspected cases, as the applicability of general exclusion protocols is largely unknown [20].

Atherosclerosis constitutes a major cardiovascular risk factor, but its role in the development of aortic dissection remains unclear. It is more common in patients with type B dissection than in those with type A dissection [21]. In general, the prevalence and extent of atherosclerosis increase with age, both in the general population and in patients with aortic dissection [22].

There is no association between diabetes mellitus or obesity and aortic dissection or aneurysm [14]. The prevalence of aortic dissection in individuals with known atherosclerotic disease, diabetes, and smoking is relatively lower compared to patients with aortic dissection, as this condition typically develops in non-atherosclerotic tissues. Therefore, the presence of these pathologies may relatively reduce the pre-test probability for acute coronary syndromes [20].

Conduct to diagnose:

Clinical Diagnosis:

The acute clinical manifestations of aortic dissection are related to the following pathological mechanisms: 1) aortic dilatation, 2) imminent rupture with external bleeding, 3) organ malperfusion, 4) inflammation, and 5) congestive heart failure.

According to practical data from the literature, "red flag" symptoms that should prompt the physician to consider the diagnosis of aortic dissection include trunk pain (including the neck), syncope, neurological deficits, and limb ischemia. However, the overall accuracy of these signs/symptoms remains limited [13, 23].

In diagnostic studies, the most sensitive symptom is sudden or severe chest pain described as tearing with dorsal radiation and accompanied by blood pressure asymmetry. However, chest pain is one of the most frequent complaints in the emergency department (approximately 6% of emergency visits), and the absence of specific features only modestly reduces the pre-test probability of aortic dissection. Specificity is higher for less common signs, such as blood pressure asymmetry, bounding pulses, acute-onset neurological deficit, and hypotension/shock, which should be considered stronger predictors [24] (Table III).

In the emergency setting, the variety of symptoms of aortic dissection that overlap with other clinical presentations makes the diagnostic approach challenging, leading to delayed diagnosis. In 38% of cases, the diagnosis was initially missed, resulting in a poorer prognosis and a significant increase in mortality rates. Therefore, there is an interest in standardizing the diagnostic approach for appropriate patient selection (Table IV).

Tableau III: Signs and symptoms of DAAA.

Clinical Signs and Symptoms	Cause
Asymmetric blood pressure (>20mm Hg) between limbs	Branch artery flow compromise
Intestinal ischemia or gastrointestinal bleeding	Malperfusion of the superior coellic or mesenteric artery
Dysphagia	Compression of the eosophage
Dyspnea	Compression of the trachea or bracnhas, congestive heart failure due to aortic regurgitation or cardiac tamponade
Hemoptysis	Vascular rupture in the pulmonary parenchyma
Hoarseness	Recurrent laryngeal nerve compression
Horner's syndrome	Nice chain compression
Myocardial ischemia or myocardial infarction	Coronary involvement by dissection or compression by aneurysm
New breath of aortic regurgitation	Incomplete closure of secondary aortic valve has leaf attachment through dilated aorta or prolapsed cusp due to dissection in aortic root
Oliguria or hematuria (coarse)	Malperfusion of one or more renal arteries
Paraplegia	Attributable spinal malperfusion to intercostal artery
Ischemia of the lower limbs	Malperfusion of the iliac artery
Shock	Cardiac tamponade, hemothorax, frank aortic rupture, acute severe aortic regurgitation, severe miocardial ischemia
Shortness of breath	...
Stroke symptoms	Cause
Superior vena cava syndrome	Branch artery flow compromise
Syncope	Malperfusion of the superior coellic or mesenteric artery
AAS indicates acute aortic syndrome	

The clinical presentation of aortic dissection in the emergency department can be classified into three main clinical scenarios.:

Scenario 1 : hemodynamic instability :
It includes critically ill patients presenting with cardiac arrest, hemodynamic instability, or shock. According to data from the International Registry of Acute Aortic Dissections (IRAD), a multicenter research organization founded in 1996 to continuously assess the management and prognosis of acute aortic dissection, 29% of patients have a systolic blood pressure < 90 mm Hg. Most of these patients are affected by complicated acute aortic dissection, including rupture (28-26% type A/B), pericardial tamponade (8%), severe myocardial ischemia (15%), or acute aortic regurgitation (12%) [25]. For these patients, stabilization and advanced resuscitation care should be accompanied by a rapid bedside evaluation, including a 12-lead ECG and Point of Care Ultra Sound (POCUS) examination.

Scénario 2 : Critical ischemia of an organ, summarizes the case of our figure:
These patients present to the emergency department with critical and time-dependent organ ischemia (myocardium or central nervous system). Overlap with scenario 1 is common, as hemodynamic instability and critical ischemia can occur simultaneously. The ECG may reveal signs suggesting myocardial ischemia, and ST-segment elevation should be interpreted as a warning sign indicating the possibility of direct involvement of the coronary ostia [33]. However, patients with ST-segment elevation should undergo a rapid and targeted evaluation of medical history, searching for signs/symptoms of acute aortic dissection, and a quick POCUS (Point of Care Ultra Sound) examination to identify patients requiring urgent advanced imaging before proceeding with medical treatment and transfer to the catheterization room.

An acute alteration in neurological status suggesting a stroke is observed in 16% of patients with SAA/DAA [32]. However, acute aortic dissection is only the cause of stroke in 1% of patients [33]. Considering the strict timeframes for reperfusion treatment in ischemic stroke, systematic evaluation of the thoracic aorta (via POCUS/thoracic radiography) in all patients is not recommended and may even be harmful. A pragmatic approach to limit diagnostic errors in cases of suspected ischemic stroke should be based on systematic search for risk factors for aortic dissection and a thorough physical examination. In patients with one or more risk factors and clinical suspicion of aortic dissection as the cause of the stroke (e.g., bounding pulses or truncal pain), thoracic angiography should be considered simultaneously with head-neck angiography.

Scénario 3 : Non-critical presentation, patient stable in instability :
This scenario involves patients without hemodynamic instability or critical organ ischemia. In the emergency department, this scenario is by far the most common, and the decision to use advanced imaging can be discussed after three steps: 1) pretest probability evaluation and clinical judgment, 2) initial imaging/POCUS, and 3) blood analysis with d-dimers (for low probability patients) (Figure 3).

Diagnostic evaluation of the clinical risk of DAAA
To standardize the pretest probability assessment of acute aortic dissection, the 2010 AHA/ACC guidelines adopted a risk score (Aortic Dissection Detection Risk Score, ADD-RS) developed based on the IRAD registry, incorporating risk markers organized into three categories: predisposing conditions, type of pain, and physical examination [31, 32] (Table II). The ADD-RS has been validated by several studies [33]. Therefore, even in cases of lower pretest probability, the ADD-RS alone is insufficient to safely rule out the disease.

The AHA/ACC guidelines identify three patient groups: ADD-RS = 0 or low risk, ADD-RS = 1 or intermediate risk, ADD-RS $\geq$ 2 or high risk. ESC recommendations suggest a dual classification: ADD-RS $\leq$ 1 or low probability, and ADD-RS $\geq$ 2 or high probability. The presence of an ADD-RS $\geq$ 2 justifies advanced aortic imaging, regardless of other results [32]. On the contrary, ADD-RS $\leq$ 1 identifies patients who can be subjected to integrated clinical exclusion, in whom the decision to perform thoracic angiography should be carefully weighed. These patients represent approximately 80% of all suspected AD patients and, therefore, significantly impact emergency department practices and resources (Figure 12).

High risk conditions	Characteristics of high-risk pain	High Risk Review Function
Marfan syndrome or other connective tissue disease		
Family history of aortic disease	Chest, back or abdominal pain described as Sudden Onset	Pulse deficit or systolic blood pressure differential
Known aortic valve disease	Sévère en intensité	Focal neurological deficit (with pain)
Recent aortic manipulation	Déchirure ou déchirure de qualité	Aortic regurgitant breath (new, with pain)
Known Thoracic Aortic Aneurysm		High blood pressure or shock

Tableau IV: Clinical risk assessment of acute aortic syndrome according to ACC/AHA criteria [7]Score : 0 criterion: low risk, 1 criterion: intermediate risk 2 or 3 criteria: high risk.

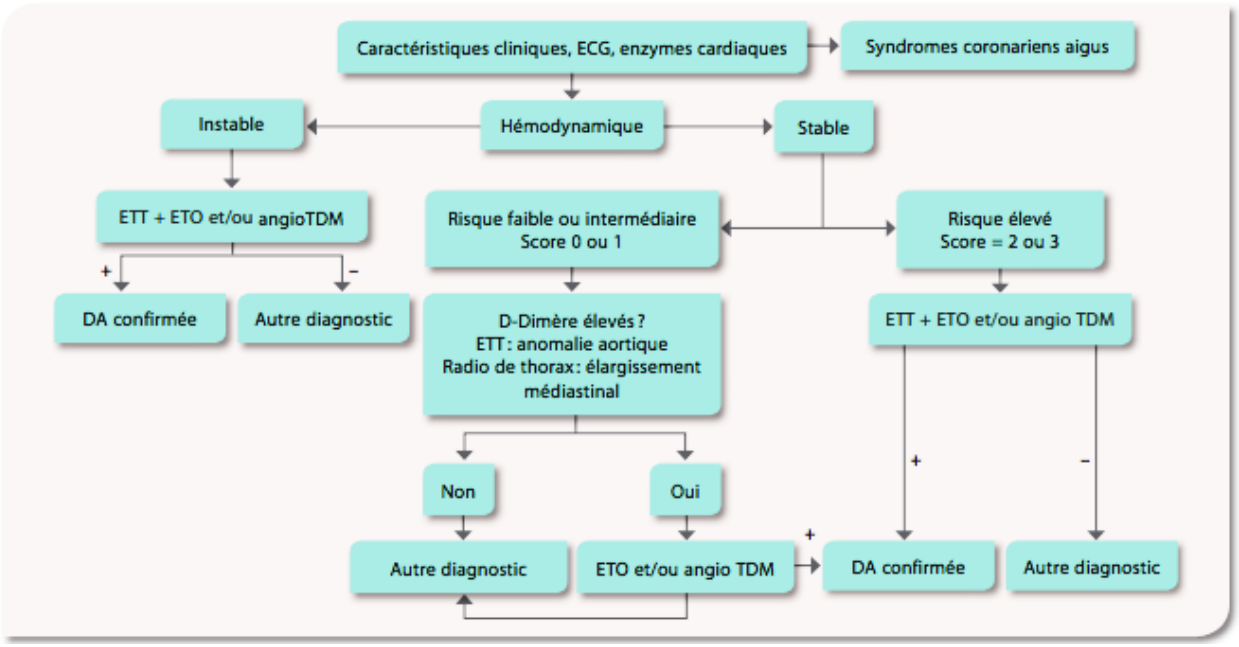


Figure 12:- Diagnostic approach to chest pain compatible with acute aortic dissection.

AD: aortic dissection; ETT: transthoracic cardiac ultrasound; TEE: transesophageal cardiac ultrasound; angioCT: angioscanner.

Diagnosis of malperfusion syndrome.

Evidence of malperfusion is present in up to 25% of patients with acute type A aortic dissection, but it must be distinguished from clinical evidence of end-organ ischemia, often referred to as the malperfusion syndrome (Table 26). Malperfusion syndrome is associated with an unfavorable prognosis, with a high mortality rate of 30.5%, compared to a mortality rate of only 6.2% in individuals without malperfusion syndrome [57, 58]. The combination

of pulse deficits (a marker of malperfusion) and hypotension should prompt rapid intervention to restore perfusion to vital organs.

Final organ	Clinical Discoveries
Cardiac	Electrocardiographic changes of ischemia or infarction, troponin elevation, myocardial dysfunction
Cerebral	Stroke and neurological deficit, coma and altered mental state
Spinal	Paraplegia
Mesenteric	Abdominal pain, intestinal ischemia, lactic acidosis, elevated liver function test results
Renal	Acute renal failure, oliguria
End	Loss of pulse in > 1 extremity, sensory or motor malfunction

Tableau V:- Clinical evidence of malperfusion "malperfusion syndrome"

The paraclinical diagnosis:

POCUS: Point Of Care Ultra Sound

It plays a significant role in the evaluation of patients in whom AAD is suspected [25, 34]. The most typical direct echocardiographic sign of AAD is the presence of an intimal flap. Indirect signs include thoracic aorta dilatation (diameter ≥ 40 mm at any level), pericardial effusion/tamponade, and at least moderate aortic valve regurgitation [27, 34]. In low-risk patients, the identification of direct signs justifies an urgent thoracic CT angiography, while the isolated presence of indirect signs on POCUS requires a case-by-case approach. POCUS is also useful for differential diagnosis with suffocating pneumothorax (absence of pleural sliding), massive pulmonary embolism (right ventricular overload), severe left ventricular dysfunction (dilated/hypokinetic left ventricle) [26, 27].

Portable TEE is useful in emergency situations with limited time and in patients with hemodynamic instability or alternatively once the patient is in the operating room. However, TEE remains operator-dependent.

ECG:

Systematic in the presence of chest pain, it can reveal signs suggesting myocardial ischemia, and ST-segment elevation should be interpreted as a warning sign indicating the possibility of direct involvement of coronary ostia. In patients with type A DA, ST-segment elevation in aVR has been specifically identified as a predictor of in-hospital death [31, 32]. However, ST-segment elevation is only found in 15% of cases [33]. Patients with ST-segment elevation should undergo a rapid and targeted evaluation of medical history, search for signs/symptoms of AAD, and rapid POCUS, which can help identify a small minority of patients requiring urgent aortic imaging before proceeding with medical treatment and transfer to the catheterization room or aortography before evaluating and treating the culprit artery, given the risk of diagnostic error and thus the probability of inappropriate management, such as coronary angiography or intense antithrombotic treatment. Nevertheless, in the context of AAD, the ECG is frequently abnormal (42% in the IRAD registry). Most patients have non-ST-segment elevation abnormalities and nonspecific repolarization disorders. The presence of myocardial ischemia is associated with an unfavorable prognosis, and ECG alterations increase the risks of misdiagnosis and diagnostic and therapeutic errors.

Chest X-ray.

A chest X-ray is routinely performed in the presence of chest pain, and in addition to providing differential diagnoses for AAD (e.g., pneumonia, pneumothorax, subphrenic free air, rib or vertebral fracture), it can partially visualize the thoracic aorta and detect pathological findings, such as mediastinal widening (≥ 80 mm at the level of the aortic knob or mediastinum-to-thorax ratio > 0.25), or rarer signs. However, chest X-ray is associated with low sensitivity (60%) and specificity (85%) for AAD and only marginally impacts diagnostic decisions [35].

Signs of aortic dissection on the results of chest radiography
Mediastinal enlargement
Contour disturbance normally distinct from aortic button
Calcium sign, which appears as a separation of the aortic wall intimal calcification of >5 mm
Double density appearance in the aorta
Right Tracheal Deviation
Deviation of the nasogastric probe to the right

Table VI:- Simple chest radiograph suggesting AAD[35].

biological test:

No biomarker is considered diagnostic, and blood tests have only a limited role in the evaluation of patients suspected of having AAD. D-dimers are commonly used in the diagnosis of pulmonary embolism, but they can also contribute to the diagnosis of AAD. The widely used threshold of 500 ng/mL to exclude pulmonary embolism can also be used for the diagnosis of AAD [36]. A meta-analysis that investigated the diagnostic performance of D-dimers in AAD showed a very high sensitivity of 97% and a rather low specificity of 56%. However, they have an excellent negative predictive value, reliably ruling out AAD in low-risk patients who would probably not benefit from aortic imaging [37]. The Diagnostic Strategy for AAD Exclusion, integrating ADD-RS = 0 plus D-dimer < 500 ng/mL or ADD-RS ≤ 1 plus D-dimer < 500 ng/mL, was found to miss approximately 1 case out of 300 AAD cases [36].

Approximately 50% of patients with AAD present with positive high-sensitivity troponin T levels, along with an ECG suggesting myocardial ischemia. Positive troponin T results delayed the diagnosis of AAD, leading to diagnostic errors and the possibility of inappropriate management, such as coronary angiography or intense antithrombotic treatment [38].

AAD is often associated with an increase in white blood cell count, neutrophil/lymphocyte ratio, a decrease in platelet count, an increase in platelet/lymphocyte ratio, and a decrease in fibrinogen level. The diagnostic accuracy of these results is modest, even when combined. However, in low-clinical probability patients, they could be used to refine the pre-test probability evaluation [39].

Despite their negligible diagnostic accuracy, lactate dehydrogenase, troponin, and C-reactive protein are markers of poor organ perfusion, poor myocardial perfusion, and secondary inflammation [41].

Advanced Imaging

A literature review on the diagnostic accuracy of thoracic angioscanner and magnetic resonance imaging (MRI) in cases of suspected AAD showed an overall sensitivity of 98 to 100% and specificity of 95 to 98% for both imaging techniques [41].

1. Thoracic angioscanner is the most commonly used imaging technique for the diagnosis of AAD. It allows for rapid image acquisition and processing with the possibility of obtaining complete 3D reconstruction of the entire aorta. Additionally, important findings provided by this examination, such as the extent of dissection and regurgitation, the size of the false and true lumen, involvement of arterial branches, or the presence of hematoma, can facilitate patient management.
2. MRI is more sensitive and precise in evaluating AAD than angioscanner, but it is rarely used as a first-line imaging modality due to its non-availability, lengthy acquisition time, difficulty in monitoring the patient during the examination, and incompatibility with implanted metallic devices. MRI is highly suitable for follow-up of patients with known aortic disease [42]. Table VII summarizes the advantages and disadvantages of each

diagnostic tool, specifying their place in the diagnostic approach for AAD according to the recommendations of the ESC (European Society of Cardiology).

Diagnostic method	**Benefits**	**Disadvantages**	CFS Recommendations [5]	**Classe de recommandation**	**Niveau d'évidence**
Measurements of D-dimers	Accessible, fast Very high sensitivity (97%)	Low specificity (56%)	Only patients with a priori low risk of aortic dissection	Iia	B
Chest radiogrqphie	Fast and non-invasive	Low sensitivity and specificity	Only patients with a priori low risk of aortic dissection	Iib	C
Transthoracic echocardiography	Fast and non-invasive	Limited to patients with abnormal chest wall, obesity, pulmonary emphysema and mechanical ventilation, Cannot visualize all aortic segments	Initial Imaging Exam	I	C
Transesophageal echocardiography	Overcomes the limitations of transthoracic echocardiography, Suitable for hemodynamically unstable patients, Excellent sensitivity (95%) and specifite (95%)	Semi-invasive, it requires sedation and control of blood pressure, Not feasible in patients with esophageal diseases, Dependent operator, Cannot visualize all aortic segments	Unstable patients with suspected aortic dissection	I	C
			Stable patients with suspected aortic dissection	IIa	C
Thoracic angioscanner	Fast image acquisition, Ability to obtain 3D images of the entire aorta, Excellent 100% sensitivity and specifite 98%	Exposure to ionizing radiation and contrast agents	Unstable patients with suspected aortic dissection	I	C
			Verification of an initially negative result of persistent suspicion	I	C
Magnetic resonance imaging	Excellent sensitivity of 98% and specifite of 98%, Visualization of the entire aorta	Indomitability with metallic devices implants and pacemakers, Problematic monitoring of the patient during the examination, Lack of large-scale availability in emergency situations	Unstable patients with suspected aortic dissection	I	C
			Verification of an initially negative result of persistent suspicion	I	C

Table VII:- Comparison of diagnostic tools in aortic dissection.

Therapeutic management: (figure 13)

Acute aortic dissection of the ascending aorta is highly lethal in untreated symptomatic patients, requiring prompt and adequate early management. The treatment depends on its location and associated complications and can be medical and/or surgical (conventional or endovascular). Initial medical management is recommended for all patients to control pain, blood pressure, and heart rate.

Management of serious patients:

In the presence of a state of shock, treatment is based on the management of the underlying cause: hemorrhage, cardiac tamponade or myocardial ischemia, restore blood volume by crystalloids, and transfusion support can be used to increase cardiac preload and output. Severe patients receiving anticoagulants should have appropriate antidotes. In case of tamponade, an emergency pericardiocentesis is indicated (AHA Recommendations 2010) in case of severe hemodynamic instability incompatible with surgery (cardiac arrest or peri-arrest), and in case of refractory shock at filling, Vasoactive drugs can be used with caution. Ideally, practitioners should aim for proper infusion of "noble organs" while minimizing stress on the damaged aortic wall [43].

Medical treatment

Optimal medical treatment is recommended for all patients (Class I/C) Figure 13 [42]. In a systematic way, the control of pain by opioids (Table VI) is the first therapeutic line, these Tier III analgesics have beneficial effects on agitation, dyspnea, respiratory distress and hemodynamic status, because of sedation and reduction of the adrenergic component [42, 43]. Pain control should be rapid, while avoiding excessive sedation, respiratory depression and vomiting. In case of shock, opioids should be avoided, but a case-by-case assessment will be required in very severe patients for whom advanced treatments are not considered.

Blood pressure control is recommended in hemodynamically stable patients for simultaneous reduction of heart rate and blood pressure to prevent aortic lesions (delay tearing process and prevent rupture) and malperfusion of organs, also perfusion of the myocardium by decreasing postcharge, and oxygen consumption. The goals of the anti-impulse therapy are a heart rate of 55-66 bpm and a systolic blood pressure of 100-120 mmHg, within minutes. Treatment should continue until the patient is transferred to a specialized unit or operating room [44]. First-line drugs are intravenous beta blockers and remain the pillar of medical treatment (Table VIII) [45]. esmolol and labétalol are preferred thanks to their easy management... other alternatives are calcium inhibitors, urapidil or clonidine [46]. If the target blood pressure is not obtained after the titration of a beta blocker and after ensuring adequate control of the heart rate, a vasodilator must be added (nitroprussiate or nitroglycerin). The single use of a vasodilator is not recommended, to avoid reflex tachycardia aggravating wall stress Figure [46].

Outside of the acute phase, the studies report long-term benefits and highlight the importance of continuing beta-blockers at the exit to improve clinical outcomes Figure 14 ,They also showed that angiotensin conversion enzyme inhibitors (ACIAs) and ARAs are beneficial in the long-term management of hypertension in patients with aortic dissection [46]. Statins are routinely used in patients after aortic dissection, although the evidence is not very strong [47].

Table VIII :- Medical treatment of aortic dissection (a) First-line drug. (b) Limited data in acute aortic syndromes.

Médicaments	Doses
Analgesia	
Morphine	1-4 mg/kg bolus (up to 10 mg every 4 h)
Fentanyl	25-100 µg every 30-60 minutes
Anti-impulse	
Beta blockers	
Esmolol (β1-blocking) (a)	0.5-1 mg/kg bolus followed by 0.05-0.3 mg/kg/min infusion (titrate with 0.1 mg/kg/min)
Labetalol (β1/2, α 1-blocking) (a)	5-20 mg bolus (can repeat after 15 min) 5-15 mg/h infusion
Metoprolol (β1-blocking) (b)	5 mg bolus (can be repeated after 5 min, up to 15 mg)
Propranolol (β1/2-blocking) (b)	1-3 mg bolus (can be repeated after 5min, up to 5mg)
Calcium inhibitors	
Verapamilb	5-10 mg bolus (can be repeated after 5-10min)
Diltizemb	5-20 mg bolus (can be repeated after 15min), 5-15 mg/h infusion
Sympatholytic with central action	
Clonidine (central a2-presynaptic agonist) (b)	0.15-0.3 mg bolus (can be repeated after 40min)
Vasodilators	
Nitroglycerine (b)	0.25-0.5 µg/kg/min infusion (titrate to
Clonidine (central α 2-presynaptic agonist) (b)	10 µg/min)
Clonidine (central α 2-presynaptic agonist) (b)	5-200ug/min infusion

Figure 13 :- The recommendations of the AD Medical Management.

Recommendations for Acute Medical Management of AAS
Referenced studies that support the recommendations are summarized in the Online Data Supplement.

COR	LOE	Recommendations
1	B-NR	1. In patients presenting to the hospital with AAS, prompt treatment with anti-impulse therapy with invasive monitoring of BP with an arterial line in an ICU setting is recommended as initial treatment to decrease aortic wall stress.[1-5]
1	C-LD	2. Patients with AAS should be treated to an SBP <120 mm Hg or to lowest BP that maintains adequate end-organ perfusion, as well as to a target heart rate of 60 to 80 bpm.[3,6]
1	B-NR	3. In patients with AAS, initial management should include intravenous beta blockers, except in patients with contraindications.[2,5,7]
2a	B-NR	In those with contraindications or intolerance to beta blockers, initial management with an intravenous non-dihydropyridine calcium channel blocker is reasonable for heart rate control.[1,2,5]
1	C-LD	4. In patients with AAS, initial management should include intravenous vasodilators if the BP is not well controlled after initiation of intravenous beta-blocker therapy.[8]
1	C-EO	5. Patients with AAS should be treated with pain control, as needed, to help with hemodynamic management.

Recommendation for Subsequent Medical Management of AAS Referenced studies that support the recommendation are summarized in the Online Data Supplement.		
COR	**LOE**	**Recommendation**
1	**B-NR**	1. In patients with AAS, it is recommended to treat with long-term beta blockers (unless contraindicated) to control heart rate and BP to reduce late aortic-related adverse events.[1-7] Additional antihypertensive agents (particularly ARBs and ACEIs) should be added, as necessary, to adequately control BP.

Figure 14 : The place of BB, IEC and ARAII in the long-term management of AD.

Traitement chirurgical et interventionnel :

DAA Type A

DAAA is a life-threatening condition due to potential sequelae, and can affect the life-threatening prognosis once suspected or diagnosed warrants urgent surgical assessment [5], surgical repair is the primary therapeutic option (Class I/B) Figure 15 , an assessment of the benefits of surgery must be balanced against the risks of the surgery itself (a demanding and complex operation in patients often physiologically compromised). Universally recognized risk factors that increase surgical mortality include shock and tamponade, neurological or visceral malperfusion, and preoperative myocardial ischemia [45,49].

Surgical repair will consist of resection of the tear site, any aneurysm aorta and the most proximal extent of dissection. Primary tearing without resection is a risk factor for re-intervention[50] by replacing the ascending aorta with a vascular graft. This replacement may require a coronary ostia re-implantation or repair/replacement of the aortic valve. If the aortic root is damaged or dilated, the surgeon can use a technique to replace the aortic bulb, while preserving the valve (technique to spare the valve: reimplantation according to Tirone David V or reconstruction according to Yacoub) or complete replacement of the aortic bulb and valve with a tube-valve conduit (Bentall technique) [51]. An individualized approach to aortic root management is based on pathology and general condition[48].

In AD type IA, the primary entry breach is in the ascending aorta in about 65% of patients. In these cases, the surgical treatment will be the same as in AD type IIA. This will restore the anterograde flow in the true aortic light and decompress/close the false light. In about 35% of cases, the entrance breach is located in the arch of the aorta, which requires more important interventions such as the replacement of the hemoglobin or the total stick with re-implantation of the supraaortic trunks (Carrell patch island technique or trifurated prostheses) or disconnecting the surgical graft in the ascending aorta. To reduce the flow in the false distal light and facilitate subsequent interventions, an additional aortic endograft can be distally positioned in the descending aorta (elephant trunk or frozen elephant trunk) [49,53].

Various interventional approaches, such as replacement of the extended open arch (with or without frozen elephant trunk), hybrid techniques or endovascular stenting have been described [52]. Aortic arch exclusion with emerging endovascular stent graft devices is an evolving area[53].

Figure 15: Recommendation in the surgical management of DAAA.

Recommendations for Surgical Repair Strategies in Acute Type A Aortic Dissection
Referenced studies that support the recommendations are summarized in the Online Data Supplement.

COR	LOE	Recommendations
Aortic Repair Strategies		
1	B-NR	1. In patients with acute type A aortic dissection and a partially dissected aortic root but no significant aortic valve leaflet pathology, aortic valve resuspension is recommended over valve replacement.[1-5]
1	B-NR	2. In patients with acute type A aortic dissection who have extensive destruction of the aortic root, a root aneurysm, or a known genetic aortic disorder, aortic root replacement is recommended with a mechanical or biological valved conduit.[6-9]
2b	C-LD	In selected patients who are stable, valve-sparing root repair may be reasonable, when performed by experienced surgeons in a Multidisciplinary Aortic Team.[10,11]
1	B-NR	3. In patients with acute type A aortic dissection undergoing aortic repair, an open distal anastomosis is recommended to improve survival and increase false-lumen thrombosis rates.[12-15]
1	B-NR	4. In patients with acute type A aortic dissection without an intimal tear in the arch or a significant arch aneurysm, hemiarch repair is recommended over more extensive arch replacement.[16-18]
2b	C-LD	5. In patients with acute type A aortic dissection and a dissection flap extending through the arch into the descending thoracic aorta, an extended aortic repair with antegrade stenting of the proximal descending thoracic aorta may be considered to treat malperfusion and reduce late distal aortic complications.[19,20]
Perfusion and Cannulation Strategies		
2a	B-NR	6. In patients with acute type A aortic dissection undergoing surgical repair, axillary cannulation, when feasible, is reasonable over femoral cannulation to reduce the risk of stroke or retrograde malperfusion.[21,22]
2a	B-NR	7. In patients with acute type A aortic dissection undergoing surgical repair who require circulatory arrest, cerebral perfusion is reasonable to improve neurologic outcomes.[23-25]
2a	B-NR	8. In patients with acute type A aortic dissection undergoing surgical repair, direct aortic[26,27] or innominate artery[28] cannulation with imaging guidance is reasonable as an alternative to femoral or axillary cannulation.[29-31]

DAA Type B :

Patients with type B aortic dissection generally have better survival than those with type A aortic dissection. In uncomplicated type B acute dissection Table IX, medical management is the first mode of treatment for DAAB (Class I/C) figure, for immediate and long-term pain control, hemodynamic stabilization and organ perfusion [54].

Patients with complicated ADAB (table X) or developing such characteristics, have an increased risk of morbidity and death need urgent interventional treatment [5,6,55].

Endovascular or surgical procedures should be considered in patients with severe aortic dilation, signs of imminent rupture, aortic rupture, or malperfusion of organs. These situations, in addition to the persistence of severe pain and refractory hypertension, define the status of «DA type B complicated» and justify endovascular thoracic aortic repair: TEVAR (thoracic endovascular aortic repair), class I/C, compared to IIb/C for surgery).

In “uncomplicated” AD type B cases, TEVAR is not indicated as a front-line strategy, and the priority will be the transfer of the patient to an ICU for medical treatment optimization and ongoing monitoring.

A subsequent TEVAR may be considered based on the long-term risk/benefit assessment (Class IIa/B). TEVAR is intended to cover the entrance breach, induce false closure of light and thrombosis, stabilize the aorta and prevent further aortic dilation. Unfortunately, up to 30% of patients may develop aneurysm degeneration of the aortic segment dissected under the treated portion, which requires further intervention in the medium to long term. The

main complications associated with TEVAR are paraplegia (3%), retrograde AD-A (2%) and new endoprosthetic-induced entry (1-35%) [44, 46].

Figure 16: recommandations for the management of acute type B dissection

Recommendations for the Management of Acute Type B Aortic Dissection
Referenced studies that support the recommendations are summarized in the Online Data Supplement.

COR	LOE	Recommendations
1	B-NR	1. In all patients with uncomplicated acute type B aortic dissection, medical therapy is recommended as the initial management strategy.[1-3]
1	C-LD	2. In patients with acute type B aortic dissection and rupture or other complications (Table 27), intervention is recommended.[4-6]
1	C-EO	In patients with rupture, in the presence of suitable anatomy, endovascular stent grafting, rather than open surgical repair, is recommended.
2a	C-LD	In patients with other complications, in the presence of suitable anatomy, the use of endovascular approaches, rather than open surgical repair, is reasonable.[4-6,7]
2b	B-R	3. In patients with uncomplicated acute type B aortic dissection who have high-risk anatomic features (Table 28), endovascular management may be considered.[8,9]

Table IX:- Characteristics of complicated acute type B aortic dissection.1 change to 5, 2 to 6, 3 to 56, 15 to55 .

functionality	commentary
Aortic rupture 1	This can be free or contained (including hemothorax, increased periaortic hematoma or both: or mediastinal hematoma) and must be treated quickly
Branch artery occlusion and malperfusion 2	Partial complete occlusion of a main branch, with or without clinical signs of ischemia including visceral, renal and peripheral arterial branches
Extension of dissection 3	Extension of the dissection flap distally or proximally (i.e., retrograde dissection type A)
Aortic enlargement	The progressive widening of the true, false or both lights during the acute phase may require rapid intervention
Intractable pain 15	
IHT not checked	

Table X:- High Risk Characteristics in Uncomplicated Type B Acute Aortic Dissection.

High Risk Imaging Results
Maximum aortic diameter >40 mm
Diameter of false light . 20-22 mm
Inlet tear >10 mm
Inlet tear on small curvature
Total aortic diameter increase >5mm between series imaging studies
Bloody pleural effusion
Evidence of malperfusion by imaging only
High risk clinical outcomes
Refractory hypertension >3 different classes of anti-hypertension drugs at maximum recommended or tolerated doses
Persistent refractory pain >12 despite maximum recommended or tolerated doses
Readmission need

Support for Malperfusion:
In the presence of malperfusion, the rate of surgical mortality is correlated with the number of malperfused organs [57, 58] hence the interest of a therapeutic approach to restore the perfusion of the branched vessels consisted of a central aortic repair (i.e., at the site of proximal aortic tearing), early reperfusion predicts survival [59] , This strategy rapidly reduces the risk of aortic rupture and corrects for associated coronary malperfusion and tamponade sequelae. After repair of the central aorta, any residual malperfusion should be evaluated with secondary interventions, if necessary [60] Figure 16 and 17.

Malperfusion cérébrale :
In patients with cerebral malperfusion, the surgical indication in emergency remains controversial, in patients with DAAA with neurological disorders (stroke, coma) are operated less often than other patients. According to the IRAD registry, only 11% of patients with no brain damage do not have surgery, compared to 33% and 24% respectively in coma and stroke [61,62]. In coma patients, Pocar et al. Retain as surgical contraindications hemodynamic instability with systolic blood pressure below 100 mmHg, absence of pupillary reactivity, a delay of more than 12 hours since the onset of symptoms and signs of gravity on brain imaging (intraparenchymal hemorrhage, bilateral massive cerebral ischemia, major cerebral edema with mass effect) [63]. However, early surgery can cause cerebral reperfusion edema, or even hemorrhagic conversion of the initial ischemic lesions, exposing the patient to the risk of functional non-recovery or even worsening of inaugural neurological deficit [63,64]. Most recent series report no cases of intraoperative hemorrhagic transformation, despite early aortic repair in extracorporeal circulation with heparin at 3 mg/kg [64,65,67,68].

While many authors consider that early aortic repair can correct brain malperfusion and limit neurological sequelae in the right hemisphere. Nakamura et al. recommend emergency surgery for patients with right ischemic stroke or limited to less than one-third of the left hemisphere on the initial cerebral scanner [67].

The strategy of brain protection during aortic arch repair surgery in patients with initial brain malperfusion remains controversial [69]. In order to prevent decanulation and recanulation maneuvers when resuming CEC after distal aortic repair, cannulation of the right axillary artery appears to be the technique with the best compromise. Its exposure in the del-topectoral furrow can however be difficult in the obese subject and the need to interpose a vascular prosthesis is not conceivable in case of hemodynamic instability of the patient requiring the rapid implementation of CEC. Another limitation to the canu-lation of the right axillary artery is the presence of TABC dissection, a common situation in DAAA, When the brachiocephalic arterial trunk dissection extends to the subclavian artery or even to the common carotid artery, We cannulate the femoral artery by planning a Y-shaped assembly on the arterial line in order to be able to perform an endoluminal anterograde cereoplasty in the real channel of ASD.

In a recent development, Rylski et al. highlight the importance of cerebral pathology by reporting the advantages and disadvantages of different sites of arterial canulation [70]. No randomized study has so far demonstrated the

superiority of a technique, however anterograde cereoploplasty via right axillary canulation with moderate hypothermia provides safe brain protection in the absence of extensive dissection of TABC [71,72]. Retrograde cerebral perfusion through the superior vena cava would appear to be more protective in case of preoperative neurological challenge, with operative mortality and long-term survival equivalent to other techniques [66,73]. This strategy should be preferred in the case of thrombotic occlusion of a carotid artery by the dissecting process [67].

With an infusion rate of between 200 and 400 ml/min, it maintains high-quality cerebral hypothermia without providing metabolic support equivalent to selective anterograde cerebral plegy [70].

The principle of deferred surgery, on the other hand, must be privileged in patients with DAAA with inaugural stroke formed according to the same team. There is no consensus on the timeline. By analogy, carotid stenosis surgery with inaugural stroke should be deferred and considered only when the recovery of the initial neurological deficit reaches a plateau and the blood-brain barrier is healed [77].

La malperfusion cardiaque et viscérale :
it leaves extremely poor results given the high mortality rate associated with irreversible organic lesions . More recent series have shown potential for improved outcomes by establishing an endovascular terminal organ infusion prior to the open central aorta repair (with the timing of the subsequent open repair decided on a case-by-case basis). These procedures can be performed in a hybrid operating room if the necessary resources and personnel are available.After repair of the central aorta, any residual malperfusion must be evaluated with secondary interventions, if necessary [78 , 79].

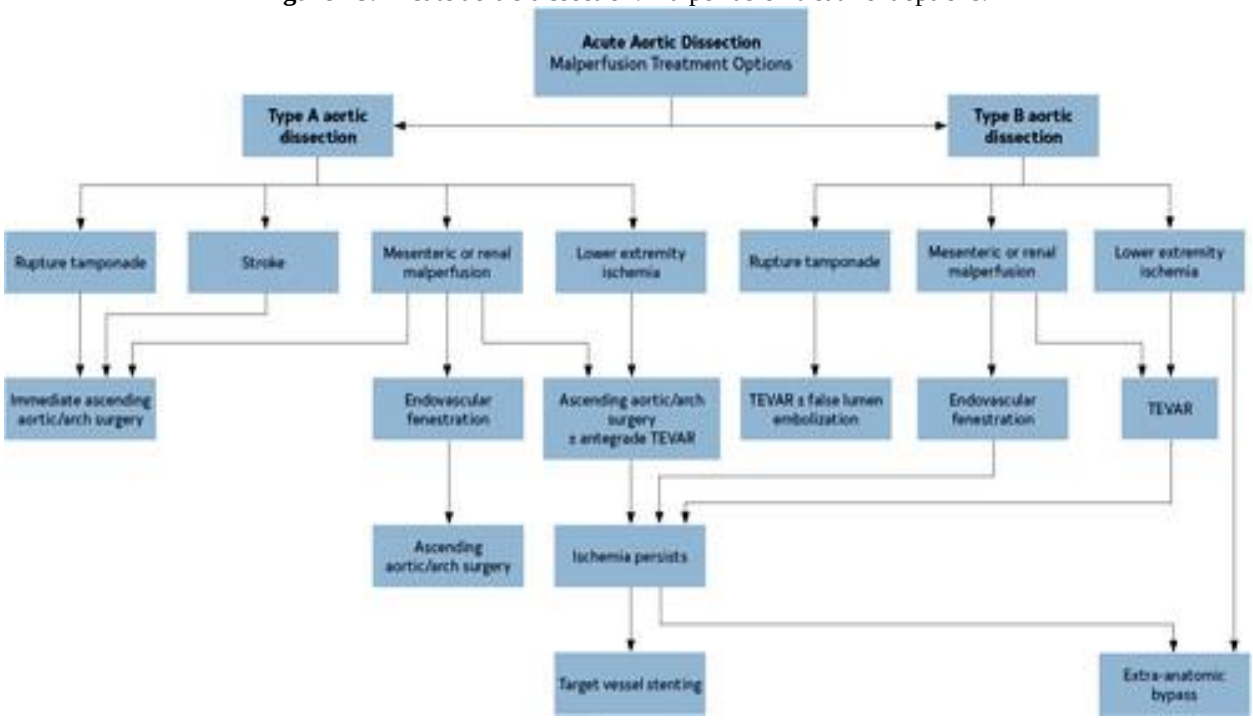
Mesenteric malperfusion
Mesenteric malperfusion is one of the most serious complications of DAAA, with an associated mortality rate of 63.2%[80]. As a result, these patients are often treated solely through medical treatment, yet at IRAD, nearly one-third of patients with mesenteric ischemia who were treated without intervention had a 95% hospital mortality rate. For DAAA patients with clinical signs of mesenteric ischemia, some centres have advocated strategies for early direct reperfusion (by either endovascular or open abdominal surgery before central aortic repair [82], other centres continue to advocate the traditional central aortic repair strategy first. Currently, data is limited to help define the best strategy. In IRAD [80,85], a surgical and hybrid strategy seems to have superior results to medical or endovascular therapy alone. An institutional series of endovascular therapy initially showed a low rate of aortic repair operative mortality of 2.1%; however, only 58% of the cohort had an open repair, 24% died of organ failure and 13% died of aortic rupture. In addition, an initial approach to endovascular therapy requires expertise in fenestration, to treat dynamic obstruction, and in branch stenting, to treat static malperfusion [85].

Figure 17: Recommendation for the management of poor perfusion syndrome

Recommendations for Management of Malperfusion
Referenced studies that support the recommendations are summarized in the Online Data Supplement.

COR	LOE	Recommendations
1	B-NR	1. In patients with acute type A aortic dissection presenting with renal, mesenteric, or lower extremity malperfusion, it is recommended to proceed to immediate operative repair of the ascending aorta.[1,2]
2a	C-LD	2. In patients with acute type A aortic dissection presenting with clinically significant mesenteric (celiac, SMA) malperfusion, either immediate operative repair of the ascending aorta or immediate mesenteric revascularization via endovascular or open surgical intervention by those with this expertise before ascending aortic repair is reasonable.[3-6]

Figure 18: Acute aortic dissection: malperfusion treatment options.



Prognosis and complication of acute aortic dissection:

AD Type A:

The potential sequelae of acute aortic dissection type A, including myocardial infarction, acute AI, cardiac tamponade, aortic rupture, and target organ malperfusion, are associated with high rates of morbidity and mortality. The reported short-term mortality rate (intrahospital and 30-day mortality) for type A AD was 13-17% (median, 14%) for open surgery and 0-16% (median, 7%) and 0-16% (median, 7%) for TEVAR (Table XI) [20].

	Medical treatment			**Open surgery**			**TEVAR**		
	Number of studies	**Number of patients**	**Mortality rate %**	**Number of studies**	**Number of patients**	**Mortality rate %**	**Number of studies**	**Number of patients**	**Mortality rate %**
DA-A et DA_B	6	1413	0-27	7	2530	13-17	20	38	0-16
Type A	1	17	7,6	3	2275	13-17	3	38	0-16
Type B	4	1126	0-27	5	255	13-17	20	1128	0-16

Table XI:- Results reported on in-hospital and 30-day mortality following aortic dissection [87].

AD Type B

Patients with type B aortic dissection generally have better survival than those with type A aortic dissection.

Patients with complicated type B acute aortic dissection have an increased risk of morbidity and death. The IRAD study showed an overall hospital mortality rate of 13%, with those requiring open repair having higher mortality rates than those managed with optimal medical management and percutaneous intervention (32.1% vs. 9.6% vs. 6.5%, respectively; P <0.0001) [88].

Medical management of uncomplicated B-type aortic dissection still having a 30-day mortality rate of 10% and a decrease in long-term survival. In the ADSORB trial, which compared optimal medical management to optimal medical management plus TEVAR, there were no early deaths in both groups and, at 1 year of follow-up, there was only one death in the TEVAR group [44,55]. TEVAR was superior to optimal medical management alone. 2 article Cambria et al reviewed the SAA results managed by TEVAR against historical controls and found a 1-year survival rate of 79% for endovascular-treated B-type acute aortic dissection [89].

A subsequent one-arm study of patients treated with TEVAR showed a 30-day mortality rate of 8% and a 1-year survival rate of 88%. VIRTUE investigators have found an advantage in early intervention, but with re-intervention rates ranging from 20% to 39%. The RESTORE patient registry showed similar results. When the intervention is an emergency, TEVAR has significantly lower morbidity and hospital mortality rates compared to open repair, with the greatest benefit in older Table IV patients [89].

Perfusion syndrome:

Malperfusion syndrome is associated with a mortality rate of 30.5%, compared to a mortality rate of only 6.2% in people without malperfusion syndrome [57]. Mortality is correlated with the number of branched arterial vessels involved and the number of malperfused organs [58].

Reference	N	Time before surgery (h)	Postoperative cerebral hemorrhage	Survival D30	Neurological recovery D30	Survival at 5 years
Nakamura, 2011 [9]	10	9,2 ± 8,7	NC	100%	100%	NC
Nakamura, 2006 [6]	5	6,8 ± 1,5	0	80%	80%	NC
Tsukube, 2014 [4]	24	3,7 ± 1,4	0	88%	58%	60%
Conzeimann, 2012 [8]	43	NC	NC	NC	13%	NC
Estrera, 2006 [5]	16	NC	0	81%	57%	58%
Morimoto, 2011 [7]	41	21,7 ± 40,5	0	85%	73%	65%

Tableau XII : Results of immediate surgical treatment of DAAA with central neurological lesions based on recent major published studies. 6 par 63,5.remplacer par 66, 9 par 67, 7 par 92, 8 par 93.

In patients with cerebral malperfusion, survival is higher with surgery, surgical management mortality rates compared to medical management are 25% to 27% versus 76%, respectively [94.95]. Even more striking, Estrera et al showed that patients with type A acute aortic dissection who had had a stroke had an operative mortality rate of only 7% and showed no worsening of the neurological condition after surgery [66], Although their study and others highlighted the speed of aortic repair in stroke patients, with a threshold of about 5-10 hours (after which neurological outcomes decreased), Fischbein et al found no association between post-operative neurological improvement and the time between onset of neurological symptoms and surgery [66]. IRAD data showed that stroke and coma resolved in 84% and 79% of patients, respectively, despite average wait times up to surgery of 12.3 and 13.8 hours, respectively [80].

Mesenteric malperfusion is one of the worst complications of DAAA, with an associated mortality rate of 63.2% [80]. As a result, these patients are often medically managed, yet at the IRAD, nearly one-third of the medical patients had a hospital mortality rate of 95%.

Conclusion:

AAS, although infrequent, are associated with life-threatening complications, and high mortality, misleading symptoms overlap with other more common ED complaints, makes the diagnosis at time difficult and often with delay, makes the prognosis darker , however , doctors must be vigilant when evaluating patients with acute chest pain, states of shock, or syndrome of cerebral or abdominal perfusion, also continuing medical training of primary care centers on recognition of aortic dissection, the introduction of simplified standardized protocols for the initial management of aortic dissection and improved communication with cardiovascular centres can halve the time between presentation and diagnosis confirmation and repair surgical.

Imaging is the cornerstone of the diagnosis and monitoring of aortic dissection, although most biological analyses in the diagnosis of aortic dissection is very low, D-dimers have very good negative predictive value and reliably rule out aortic dissection in low-risk patients who would likely not benefit from aortic imaging [36].

Patients with type A aortic dissection are generally candidates for evaluation and urgent surgery, so complicated type B aortic dissections require evaluation for urgent endovascular treatment, then for uncomplicated B-type aortic dissections, the optimal medical treatment is the current reference.

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