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Abdominal Aortic Aneurysm and its Rupture: An Overview

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ASBTRACT

The word aneurysm is originated from the Greek word "Aneurysma" ascribing the permanent cardiac or vessel dilatation. Abdominal Aortic Aneurysm **(AAA)** can be defined as an abdominal aortic diameter of 3.0 cm or more in size. The reported average growth rate of AAA ranges form 0.2-0.4 cm per annum. Risk modifications include antibiotic therapy, normal glycemic control, statins, anti-inflammatory medication, selenium, vitamin D (25-hydroxyvitamin D), Ace-inhibitors, smoking cessation and well-controlled chronic obstructive pulmonary disease. Screening for AAA is advocated in men aged 65-79 although this does not apply to the female population. Screening is performed by ultrasonography that remains cheap and radiation free. Upon detection of AAA depending on size, patients are entered to various surveillance clinics with variable follow up period defined by their size. Once a AAA, reached an interventional size (>5.5 cm), open repair and/or Endovascular Aneurysm Repair **(EVAR)** depends on patient anatomy, comorbidities and overall quality of life. Its elective repair has a mortality of 1-5% in specialized vascular units and up to 10 % in district general hospitals. The path to rupture is proportionate to an increase in the aneurysm size, multiple pathophysiological factors at cellular, molecular and systemic levels. Once the AAA ruptures, the mortality rises to 50-80% amongst those that make it to the hospital.

Currently, in United States **(USA)**, ruptured AAA **(rAAA)** claims 15,000 lives every year, and it is the 13th leading cause of death in USA. In United Kingdom **(UK)** this accounts for 8,000 deaths annually. The repair of ruptured AAA should not be delayed by any investigative modality unless patient exhibits a stable hemodynamic state. Computed tomography angiography remains the gold standard investigative modality of the choice. The current resuscitation of rAAA is subjected to various debates but the most common practice is permissive hypotension.

Keyword: Abdominal aortic aneurysm; Ruptured abdominal aortic aneurysm; History; Terminology; Anatomy; Pathophysiology; Epidemiology; Presentation; Screening; Surveillance; Risk factors; Novel Therapies: Mechanism of rupture

BRIEF HISTORY

The word aneurysm is originated from the Greek word "Aneurysma" ascribing the permanent cardiac or vessel dilatation. The most common site of arterial aneurysm is the infra-renal abdominal aorta [1,2]. The first description of the aortic aneurysm or so-called the "tumor of the artery" was found in one of the oldest medical documents called "Eber Papyrus" around 1500 BC. Since then various unsuccessful suggestions and attempts have been made by different physicians to repair the Abdominal Aortic Aneurysm **(AAA)** [3]. Amongst them, the most famous attempt was the wrapping of "Albert Einstein" aneurysm with "cellophone" that never prevailed. It was not until 1951, when two surgeons (Freeman & Dobust) within the time frame of one month in different countries managed to successfully repair an abdominal aortic aneurysm by a synthetic tube graft [4]. This technique referred to as an open repair remained the only method of abdominal aortic aneurysm treatment until the introduction of Endovascular Aneurysm Repair **(EVAR)** and/or stenting by Volodos and its later modification by an Argentinian surgeon Parodi in 1999 [5,6]. Currently, both techniques remain the only method of treatment for AAAs. In elective settings Endovascular Aneurysm Repair **(EVAR)** has now become the commonest approach in the repair of abdominal aortic aneurysms. Despite advances in the field of EVAR and it is associated technology; open technique remains the commonest surgical approach in the repair of ruptured abdominal aortic aneurysms.

EPIDEMIOLOGY

The current reported incidence of AAA is around 4.9-9.9%. Its elective repair has a mortality of 1-5% in tertiary vascular centers and up to 10 % in district general hospitals. Once the AAA ruptures, the mortality can rise to 50-80%. This is amongst the patients that make it to the hospital. Currently, in United States **(US)**, ruptured AAA **(rAAA)** claims 15,000 lives every year, and it is the 13th leading cause of death in USA. In United Kingdom **(UK)** this accounts for 8,000 deaths annually [7,8]. There also appears to be a clear familial (genetic) link for AAA. Estimated risk of AAA in first-degree relatives is 11.6 times higher than the rest of non-affected population. Screening process of siblings of aneurysm patients has shown 6 % of sisters and 29% of brothers to have aneurysm too [9]. The AAA possesses a 2:1 male to female ratio at any given age. In addition, there appears to be higher incidence of AAA in Caucasians population in comparison to Afro-Caribbean ethnic backgrounds [10].

TERMINOLOGY AND ANATOMY

The native and the normal caliber of infra-renal aortic diameter in healthy male population at its largest measures at about 24 mm and this value is 22 mm in female cohort [11]. Overall, the female population exhibits a smaller aortic diameter but the relative aortic diameter based on the predicted size in relation to overall body surface area, exhibits a much larger native aorta than their male counterparts [12]. Once the infra-renal aorta reaches 1.5 times of its mentioned size, the term aneurysm is applied, if the enlargement remains below this threshold, the term "Ectasia" is used. If this affects the entire circumference of the artery the term "Fusiform" and if partially, the term "Saccular" is applied. Each artery including the abdominal aorta is comprised of three major layers; The Tunica intima (inner most layer), Tunica media and Tunica adventitia. The aneurysmal degeneration of any artery involves all the three layers. Tunica intima is composed of multi-functional single layer endothelial cells where as tunica media contains smooth muscle cells, elastin (12%) and collagen fibers. Tunica adventitia contains connective tissue and vasa vasorum [13]. The current cut off size for the repair of AAA is based on two major prospective randomized trials. Both trials suggest that AAA measuring 5-5.5 cm should be repaired as the rupture ratio at this level remains at 1% per annum. If the AAA reaches 5.5-5.9 cm in size, the incidence of rupture increases to about 9.4%. This trend continues as the AAA continues to grow in size [14,15].

PATHOPHYSIOLOGY

The most common aetiology (90%) behind infra-renal abdominal aortic aneurysm formation (Figure 1) is atherosclerotic degeneration. The second common aetiology is an inflammatory process (5%) where as infectious (mycotic) and autoimmune disease plays a less attributing role. The details of all accepted and suggested aetiologies are tabulated in (Table 1) [16]. The current evidence suggests that the path to arterial wall remodeling (aneurysm formation) is the sequence of multiple pathological factors that result in endothelial dysfunction, proteolytic degradation, destruction of the elastic media, depletion of vascular smooth muscle cells and inflammation of the aortic wall components both at macro and micro level. The initiating trigger is believed to be the consequence of alterations in Wall Shear Stress **(WSS)** due to flow changes (high versus low) [17]. This process creates a unique hemodynamic and mechanical insult to the aorta and ignites the aneurysmal pathogenesis. The cascades of events occur mostly in the tunica media and intima. In this process the endothelial cell damage along with smooth muscle cell, elastic and collagen destruction is accelerated by enhanced Metalloproteinase **(MMP)** and other proteinases expressions. In such circumstances, elastin reduces from 12% in a healthy aorta to 1% only. The commonest MMPs associated with AAA are MMP 2 (Gelatinase A) and MMP 9 (Gelatinase B) that exhibit elastolytic and proteolytic features [18]. In spite of the cellular pathogenesis being widely understood, the exact mechanism of aneurysm formation remains undetermined to this date (Table 2).

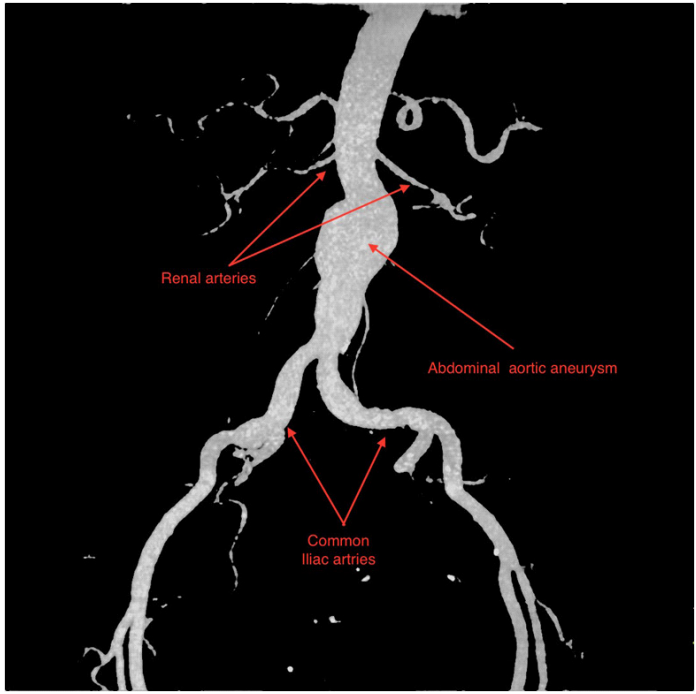


Figure 1: Abdominal Aortic aneurysm and anatomical relation to other arteries.

Table 1: Accepted and postulated aetiology of Abdominal aortic aneurysms.

Aetiology	Percentage
• *Degenerative (atherosclerosis)*	**90%**
• *Inflammatory (Vasculitis).*	**5%**
• *Infective (Mycotic).*	**2%**
Fungal.	
Bacterial *(Staphylococcus Species, Streptococcus Species,*	
Non-typhoidal Salmonellae Species,	
Sexual transmitted disease).	
Viral.	
Poly-microbial.	
• **Autoimmune/ Genetic Disorders.**	**1%**
Marfan Syndrome.	
Loeys-Dietz syndrome.	
Ehlers-Danlos syndrome.	
Turner syndrome.	
Genetic Mutation.	
• ***Traumatic***	**1%**

Table 2: Risk factors associated with AAA expansion and their respective targeted therapies.

Risk Factor	Impact	Targeted Therapy
Smoking	• 2.5 fold increase in expansion & rupture.	• Smoking cessation (Varenicline)
COPD	• Enhances degenerative changes & expansion.	• Control of COPD
Hypertension	• Increase aortic wall stiffness, reduction in elastin fibers.	• Regular monitoring and medication (ACE Inhibitors & Ang II receptor blockers).
Low Vitamin D	• Enhances expansion	• Multivitamins & Sun exposure
Low Selenium	• Enhances expansion	• Multivitamins
Cholesterol &LDL	• Enhances expansion through matrix degeneration	• Statins (HMG-CoA reductase)
Renal Failure	• No impact on large vessels.	• None
Diabetes Mellitus	• Inhibitor of Growth	• Normo-glycemic control
Female Gender and old Age	• Worse outcome	• Informed consent.
Low atmospheric pressure	• Higher rupture incidence	• Prioritization of AAA in such season.

PRECIPITATING RISK FACTORS

Advancement in vascular biology has lead to better understanding of ongoing degeneration of AAA at molecular, cellular and systemic level. This in conjunction with recent development in pharmacological therapies aimed at associated risk factors could serve as an important strategic tool for control or limitation of AAA expansion. Hence, in this review, each associated risk factor will be discussed with their respective management strategy (Table 2). In addition, the role of novel therapies in inhibition of AAA growth will be discussed in further detail (Table 3).

Table 3: Novel therapies in inhibition of abdominal aneurysm growth and their eventual rupture.

Therapy	Impact	Medication Types
Antibiotic therapy	• Atherosclerotic Plaque. • 40% reduction (MMP-9).	• Doxycycline 150mgs. • Roxithromycin 300mgs. • Rapamycin
Anti-inflammatory Therapy	• Prostaglandins. • Induces MMP-9	• Celecoxib (COX-2 inhibitor).
Mast cell Inhibitor	• Reduces MM expression. • Reduction of AAA expansion by 40%.	• Cromolyn Sodium
Kinase Inhibitor (c-Jun N-terminal kinase) (JNK)	• Molecular protein in apoptosis. • Cell differentiation & Proliferation.	• JNK inhibitors.
Genome Therapy	• Genome regulation & expression.	• Inhibition of genome transactivation.

Diabetes Mellitus

Diabetes mellitus **(DM)** is a well-established risk factor for atherosclerotic arterial disease but interestingly DM plays a protective role in AAAs. This has been attributed to increase synthesis

and decrease degradation of the Matrix Metalloproteinase **(MMP)** resulting in thicker aortic wall. Hence reducing growth and rupture incidence. This is believed to be the direct result of hyperglycemia in this cohort of patients [19].

Smoking

Smoking remains as one of the most relevant risk factors in AAAs growth and subsequent rupture. There exists a 2.5 fold increase in smokers versus non-smokers [20]. It appears that Tobacco and one of its contents Nicotine can induce MMP-2 through activation of protein kinase alpha-2 resulting in enhanced arterial degeneration and remodeling [21]. Therefore, an aggressive approach in smoking cessation plays a crucial role in early inhibition of aneurysm growth and its subsequent rupture.

Chronic Obstructive Pulmonary Disease (COPD)

Although majority of COPD in AAAs cohort is smoking related but evidence suggests that COPD remains an important risk factor well beyond the smoking point [22]. This might be related to the impact of significant reduction in forced expiratory volume 1 to that of Forced Expiratory Volume **(FEV1/FEV)** in AAA patients compared to non COPD group. This reduction has shown to enhance MMP 2 and neutrophil elastase (proteolytic enzymes) expression thus enhancing aneurysmal degeneration [22].

Renal Failure

In recent years there has been an accumulating level of evidence to support the independent role of Renal Insufficiency **(RI)** and raised estimated Glomerular Filtration Rate **(eGFR)** in prediction of vascular events in peripheral vascular disease [23]. Despite known impacts of RI and raised eGFR on small to medium arteries, such impact on AAA (large artery) remains contentious. In addition, no direct impact between MMPs and renal failure has yet been established.

Hypertension

Hypertension is a well established and an independent risk factor for rupture of AAAs [24]. Each cardiac cycle produces discrete quanta of energy, which result in oscillation, loading and unloading of elastin and collagen fibers permitting a considerable expandability of the normal aorta within normal blood pressure [25]. But due to significant reduction of such fibers (12% to 1%) in a deformed and aneurysmal aorta, exceeding blood pressure (≥ 100mmHg) results in stiffening of aorta and further recruitment of non-distensible fibers precipitating rupture [26]. Therefore, a well-controlled blood pressure in conjunction to life style modifications in achieving so is of vital importance.

Atmospheric Pressure

Current literature suggests that periods of low Atmospheric Pressure **(AP)** (meteorological parameter) irrespective of the day, season and month have a direct correlation to higher incidence

of sudden rupture. This has been attributed to alteration in partial Pressure of Oxygen **(Po2)** and Carbon Dioxide **(Pco2)** along with changes in main arterial blood pressure. The combination of hypoxia, hypercarbia and drop in blood pressure activates the sympathetic stimulation, enhancing the intra and extra luminal arterial pressure and subsequent rupture [27]. Despite various postulated hypothesis the exact mechanism of rupture in correlation to AP remains unknown [27].

Vitamin D (25-hydroxyvitamin D)

Another recently identified risk factor has been low levels of circulating plasma Vitamin D (25-hydroxyvitamin D). It appears that in a population-based study of three hundred and eleven older men, low levels of Vitamin D remained an independent risk factor in enlargement of AAAs. In addition, this study also demonstrated dose-response relationship between AAA diameter and vitamin D supplementation. The only set back of this study was the lack of calcium and parathyroid circulatory levels that are known to have a direct relationship to Vitamin D levels [28].

Selenium

Another recently recognized serum marker associated with AAA degeneration and enlargement has been low levels of Selenium **(Se)**. In this study seventy-three individuals (n=73) based on their aneurysm size were categorized to three groups and their respective serum Se were obtained. The higher AAA size was associated with lower levels of circulatory Se. In Addition, an inverse relationship was observed between serum Se and AAA diameter. Hence, suggesting Se might have an independent impact on molecular degeneration of AAAs [29].

Cholesterol

High levels of cholesterol remain an independent risk factor for atherosclerotic change of abdominal aorta and aneurysmal degeneration. Literature suggests Low-Density Lipoprotein **(LDL)** cholesterol to act via inflammatory mediated pathway in matrix degeneration of the abdominal aorta and contribute further to their expansion. Therefore, initiation of statins (HMG-CoA reductase) upon diagnosis of AAA remains essential to inhibit their further growth [30,31].

Gender and Age

The male population tends to present at a younger age and demonstrate a better outcome (mortality & morbidity) in comparison to female cohort. Overall, there is insidious difference in diagnosis, intervention and outcome of rAAA in woman compared to male individuals. The most widespread accepted explanation to date has been the protective impact of endogenous estrogen on cardiovascular disease in female patients. It has been demonstrated that estrogen inhibits the aneurysmal degeneration and negative remodeling of the aorta. This effect eventually wears off at the menopausal stage and hormone replacement therapy has failed to achieve same result *in vitro* studies [32].

NOVEL THERAPIES INHIBITING AAA GROWTH

Antibiotic Therapy

In the last decade there has been a suggestion that atherosclerotic plaque in the wall of abdominal aortic aneurysm often contains bacterial pathogens and early initiation of antibiotics may aid in regress or inhibition of the aneurysm growth. Two randomized clinical trials, demonstrated doxycycline (150 mg daily) and roxithromycin (300mg daily) to inhibit aneurysm expansion but this was only effective in the first year of treatment and beyond that time frame no further effect was noted [33,34]. Rapamycin an immunosuppressive agent in a recent trial has shown 40% reduction in aneurysm expansion through reduction of Matrix Metalloproteinase **(MMP-9)** expression [35].

Anti-Inflammatory Agents

Increase production of prostaglandins in the process of inflammation induces Matrix metalloproteinase production, which plays a significant role in expansion of the aneurysm. Recent study by Walton et al. demonstrated that Celecoxib, a COX-2 inhibitor (non-steroidal anti inflammatory, cyclooxygenase type 2) significantly decreases the incidence and severity of AAA formation. This effect was not similar in COX-1 inhibitors [36,37].

Mast Cell Inhibitor

Mast cell stabilizers or inhibitors traditionally used for control of allergic disorders and bronchial asthma have shown promising outcome in reduction of aortic expansion by 40%. Amongst suggested medication is Cromolyn Sodium that inhibits histamine and macrophages in the process of the inflammation that in turn reduces MMP expression [38].

Kinase Inhibitors

Kinase or so called c-Jun N-terminal Kinases **(JNK)**, a molecular protein that play a crucial part in apoptosis, cell differentiation and proliferation reacts to inflammatory changes as a result of stress stimuli, shock and cytokines. Presence of AAA has shown elevated levels of JNK that contribute to pro-inflammatory and MMP-9 activation. The JNK-Inhibitors not only reduce the inflammatory changes in AAA patients but also aid in restoration of aortic tissue [39].

Genome Therapy

In recent years there has been a significant emphasis on gene regulation and expression therapies. The transcription of a defined gene is up-regulated by specific proteins known as transcription factor that are found in families with AAA aneurysms. Targeted therapies aiming to avoid transactivation of such factors are still in early stages but their inhibition could contribute in complete resolution of inflammatory and matrix degradation associated with AAA expansion [40].

MECHANISM & HEMODYNAMICS OF RUPTURE

In general, it is accepted that the natural history of AAA is further growth and eventual rupture (Figure 2). This might have direct correlation to the aneurysm diameter. The expansion rate of an AAA is at around 0.2-0.4 cm per annum [41,42]. This remains subjective to the individual risk factors and genetic predisposition explained in detail in later section of this chapter. In theory, the "Laplace law" has been traditionally used to demonstrate why an AAA deforms and ruptures [43]. According to this law there is a direct correlation between tension, pressure and the radius of each cylindrical structure. This aids in calculating the circumferential tension and the possibility of the rupture [43]. However this law might not be applicable to infra-renal AAAs of all shapes (Saccular versus Fusiform) as they might present more of a spherical structure then cylinder in practice. In such cases "vessel wall stress" defined as the restoring force per unit area in circumferential and longitudinal direction is more useful in defining the rupture (Table 4). Glagov et al. [44] in 1983 demonstrated a linear relationship between the thickness of the aorta (tunica media) and that of the aortic radius at a mean given blood pressure. So a larger aorta that has a thicker wall is more resistant to rupture than a smaller aorta. Therefore, if two aortas of unequal size develop an aneurysm of a same size, the aneurysm formed from a smaller aorta is at higher risk of rupture. This theory was reinforced by the work of String fellow and colleagues [45]. In this study, they exhibited that the gradient wall stress in a six (6)cm aneurysm arising from three (3) cm and of one (1) cm aortas exhibit significant differences in longitudinal and circumferential directions. Therefore, a 6cm aneurysm arising from a 1cm native aorta has much higher chance of rupture than that of a 3 cm native aorta due to significant gradient wall stress differences [45]. Overall, the evidence suggests that the path of rupture is multifactorial and its exact nature remains elusive to this date.

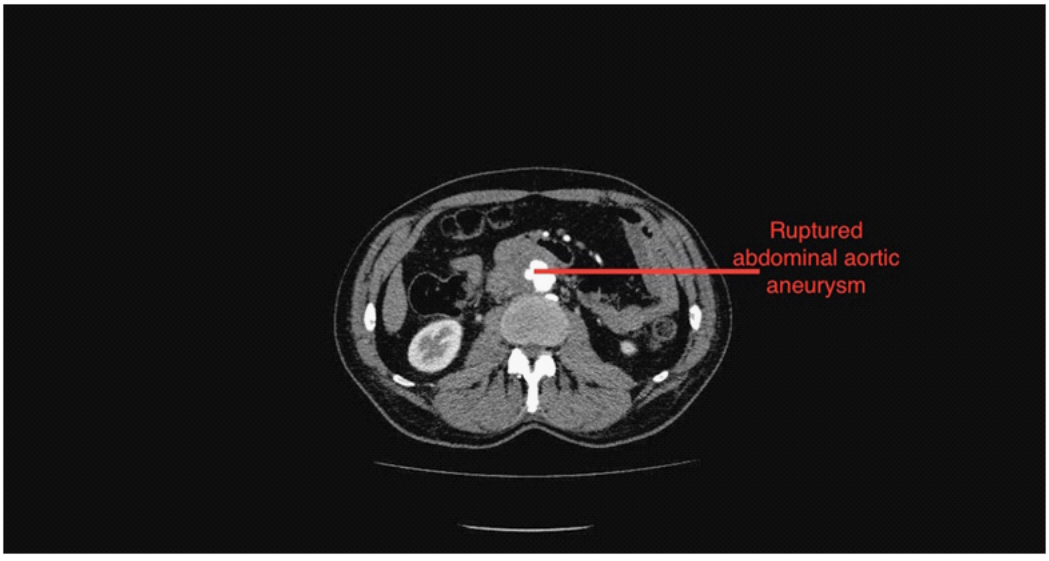
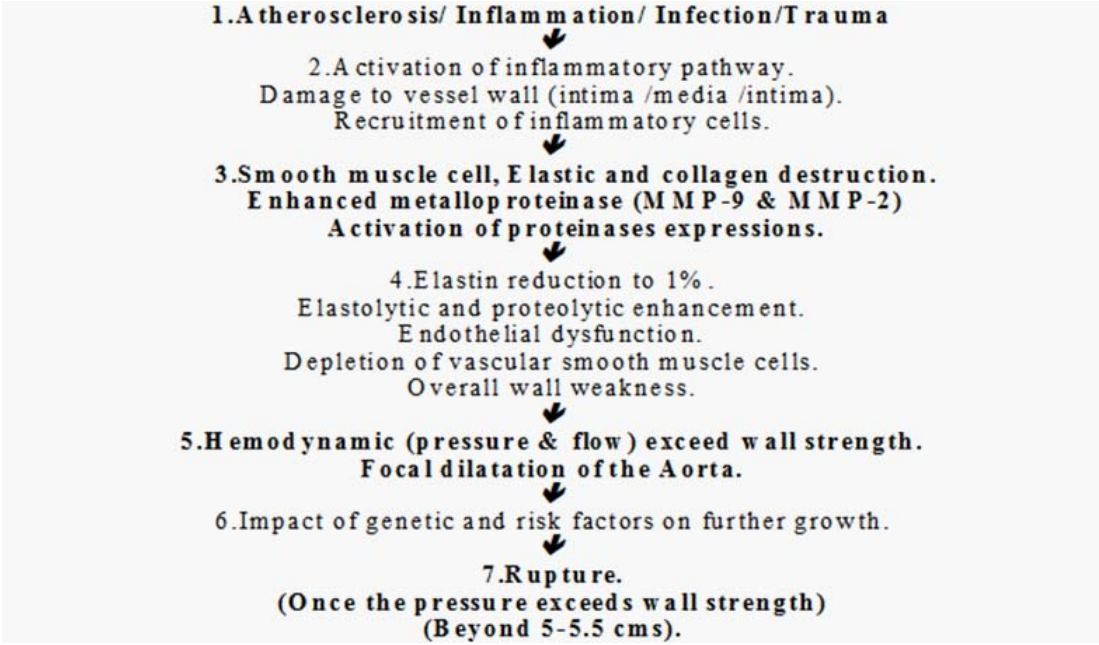


Figure 2: Ruptured Abdominal aortic aneurysm.

Table 4: Process and mechanism of rupture of abdominal aortic aneurysms.



PRESENTATION OF RUPTURE

According to the words of Sir William Osler "Aneurysm of the abdominal aorta is very often diagnosed when not present, and when present the symptoms may be so obscure that the nature of the trouble is overlooked" [46]. (Figure 2) This perhaps remains the best explanation of AAA and its clinical manifestation despite significant advancements in the field of diagnostic medicine. Almost all patients with infra-renal AAA are asymptomatic and their diagnosis is mainly incidental. This is mainly the result of an ongoing investigation for other abdominal presentation where an Ultrasound **(USS)** or Computed Tomography scan **(CT)** and detail clinical examination reveals a pulsatile mass. AAA becomes symptomatic when they rupture or cause complications [47]. The most common presentation of ruptured infra-renal AAA is the classical triad of: sudden and sever back pain with or without abdominal pain, hypotension and/or syncope and pulsatile abdominal mass. However, such presentation is only seen in 25-50 % of the population and some may present with entirely different presentation (Table 5), misleading clinicians towards other patho physiological conditions [48]. It appears that the anatomical site of AAA rupture dictates its clinician presentation and this factor perhaps explain why there is 30% misdiagnosis in practice [49].

Table 5: Common and uncommon presentation of ruptured AAA.

Common presentation of ruptured AAA (25-50%) **(Classic Triad)**
• Sudden onset sever back pain (± abdominal pain) • Hypotension (± syncope) • Pulsatile abdominal mass
Non-common presentation of ruptured AAA (30%)
• Right flank pain. • Groin pain. • Testicular pain or pressure. • Compression effect causing transient lower limb paralysis. • Testicular ecchymosis (blue scrotum sign of Bryant). • Hernia. • Urolithiasis.
Rare presentation of rupture AAA (5%).
• Aortocaval fistula (high output cardiac failure, dyspnea, tachycardia, wide pulse pressure, cyanosis, and lower limb edema) • Aortoenteric fistula (Sever Melena and death). • Aorto-left renal vein fistula (abdominal pain, haematuria, silent left kidney syndrome).

Rupture Site and Clinical Manifestation

Given the anatomical location of the abdominal aorta, rupture can occur anteriorly (peritoneal), posteriorly (retroperitoneal) or in to the adjusting anatomical structure such as bowel (Duodenum, Aorto-enteric fistula), Inferior vena cava (aorto-caval fistula), left renal vein (Aorto –left renal vein fistula) and in very rare cases contained chronic rupture. The retroperitoneal rupture (posterior) is the most common type of rupture (80%) [50]. This is the result of tear in the abdominal aortic wall that permits blood leak into the posterior compartment of abdominal cavity. This manifest itself clinically with the classical triad of presentation as described earlier. Such patients due to temporary sealing of the rupture site due to tamponade effect may be stable for the few hours of presentation and normally have better prognosis due to their early diagnosis [50].

In 15% to 20% of the cases, the AAA ruptures anteriorly and into the peritoneum. In such circumstances, given the amount of free embryological space in the anterior compartment of abdominal cavity, the extent of bleeding is so severe that patient goes into sudden syncope and loses consciousness. Due to the lack of tamponade effect and loss of entire circulatory volume, patients do not make it to the hospital [51]. In very rare scenarios (3-4%), the presence of AAA and constant chronic inflammation can cause erosion into the neighboring anatomical structures.

Amongst them, erosion to the inferior vena cava (aorto-caval) remains the most common of all. Given the containment of the circulatory volume (blood) in such circumstance, the size of the fistula (communication) between the aorta and inferior vena cava dictates the presenting symptoms. Abdominal bruit on auscultation remains the most common clinical finding but in extreme clinical situations, high output cardiac failure (dyspnea, tachycardia, wide pulse pressure, cyanosis) and lower limb edema or pulsation has also been reported. Majority of aortocaval fistulas are detected during elective surgery (50-90%) and they rarely present in extrermis [52-54].

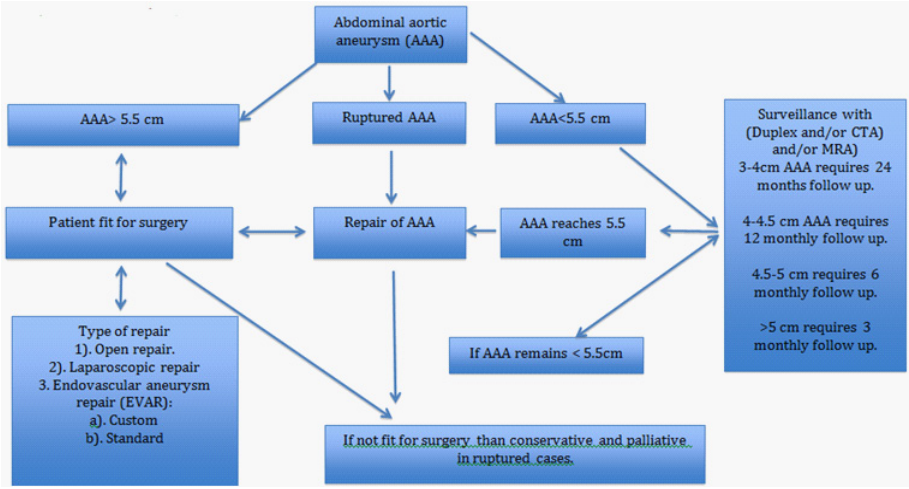
Aorto-left renal fistula remains a rare identity and following its first description by Lord et al. and its repair by Debakey et al. only 26 reported cases have been identified in the literature. In majority of cases, the AAA erodes to the left renal vein, which runs anteriorly (90%) and in rare anatomical variation posteriorly (1-4%). Almost all patients, present with abdominal pain, hematuria and silent left kidney syndrome and in rare scenarios, high output cardiac failure [55].

Due to the close proximity of third and fourth parts of duodenum to AAA, in extremely rare circumstances (0.5–2.3%) an aortoeneteric fistula is formed. This condition is fatal (95%) and very few patients make it to the hospital. The reported incidence of aortoeneteric fistula in general population as a result of AAA is 0.04–0.07% at autopsy [56]. In very rare clinical (4%) situations following posterior rupture of AAA, a large hematoma and its binding resistance due to peri-aortic tissue (tunica adventitia) can result in contained ruptured or sealed ruptured aneurysm. Some patients might escape detection for days and months due to their limiting clinical manifestation. This was first reported by Szilagyi et al. and majority of such patients presented with chronic pain and signs of compression (transient neuropathy). Such patients may rupture at any time and upon their detection they should be priorities like ruptured AAA cohort [57].

SCREENING AND SURVEILLANCE

The role of regular screening programme for AAAs has gained significant attention in the last decade. The rational is early detection of AAA in order is to avoid high mortality and morbidity from rupture. According to Cochrane review the odds ratio in favour of screening for male population in reduction of mortality was 0.60 (95%CI 0.47-0.78) [56]. This review recommends men aged between 65-79 will benefit significantly from routine screening for the detection of AAA. The method of screening is through ultrasonography and calculation of the aorta and/or AAA through Anterior-posterior measurement rather than transverse. The use of ultrasound is cheap and none invasive with no radiation and a minimal learning curve [56]. The Cochrane review did not report any reduction in mortality and morbidity associated with the use of routine screening for the detection of AAA in female population (OR 1.99; 95% CI 0.36 to 10.88) [56]. Once an abdominal aorta of >3 cm in size (termed as abdominal aortic aneurysm) is detected, the patient is than followed up by routine surveillance in specialized vascular and/or cardiovascular units. For AAA measuring 3-4 cm in size 12-24 monthly, for 4-4.5 cm AAA 12 monthly and above 4.6 cm 6 monthly surveillance is recommended [58] (Table 6).

Table 6: Algorithmic approach to AAA.



PREOPERATIVE INVESTIGATIONS

Imaging Modality

Computed Tomography Angiography **(CTA)** is the gold standard investigative modality and should be performed in any patient with an interventional size AAA. Upon suspicion of ruptured AAA, the only treatment is the repair of abdominal aortic aneurysm. However prior to surgery, if patient is stable, the gold standard and the investigative modality of the choice is Computed Tomography Angiography **(CTA)** [59]. This modality demonstrate, the size, shape, site, extent and possible other variations or complication of the ruptured AAA with 95 % specificity and sensitivity. In addition, CTA can also assist the operating surgeon in minimizing the amount of dissection and assist in defining a more precise surgical route. In unstable individuals an ultrasound abdomen [60] can be performed to confirm diagnosis although the information obtained from this modality is not as precise and informative as CTA. This is mainly due to the patient body habitus, bowel shadow and its lack of tolerance due to pain. In such circumstances, where time is of essence given the significant hemodynamic instability, no investigative modality should delay surgery (Table 7).

Table 7: Investigative modality and their relevance for ruptured abdominal aortic aneurysms.

Type of investigation	Information obtained
• **Computed tomography angiogram (CTA).**	
• **Ultrasonography (USS).**	Size, Site, Shape, Anatomy, rupture site, defining surgical approach.
• **Full Blood Count (FBC).**	Site, Size, Shape, Anatomy, No detail on site of rupture. Degree of blood loss (Hemoglobin). Platelet count, Inflammatory markers.
• **Renal Function Test (RFTs).**	Electrolytes (Sodium & Potassium), Kidney function status (Urea, Cr).
• **Coagulation Profile.**	Activated Partial Thromboplastin Time **(APTT)**, Prothrombin Time **(PT)** & International Normalized Ratio **(INR)**.
• **Group, Save and cross match**	Type and rhesus status of patient blood for blood and blood component transfusion.

Other Investigations

Once decision for surgery is made, all patients required detailed blood evaluation although these investigations should not delay surgery and must be parallel to final preparation for surgery. Full Blood Count **(FBC)**, coagulation profile, renal function test, group and save and cross match for blood and its component transfusion are of vital importance (Table 6).

RESUSCITATION STRATEGY

The management of a patient with rAAA (hemorrhagic shock) in the preoperative period plays a vital role in the final outcome of the patient. The ongoing loss in the circulatory volume (blood) causes compensatory and reduced perfusion to the vital organs. This explains why such patient present with syncope or loss of consciousness. In order to achieve adequate perfusion, the aim of resuscitation is to obtain a systolic blood pressure of 100 mmHg. This is referred to as "permissive arterial hypotension" [61] and remains the main strategy in the primary resuscitation of all type of ruptured aneurysms in the body. In permissive hypotension, the objective is to gain adequate perfusion to all vital organs but not to increase the systolic pressure to a level that can result in further tearing of a rAAA and loss of present tamponade effect [61].

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