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Emergency Embolectomy for Acute Vascular Occlusion in Al Sader Teaching Hospital /Al-Basrah City

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Abstract

Background Embolectomy can be consider a simple procedure that can be performs under local anesthesia which have a great influence on the outcome of patients and their limbs ,especially when it performs within the golden hours of ischemia.

Aim of Study This study was designed to discuss the effectiveness of embolectomy procedure in the management of acute vascular occlusion in our center and compare the outcomes with other regional and national studies.

Patients And Methods A retrospective study was carried out from October 2014 to October 2017 at AL Sadder teaching hospital in Al Basrah city. Traumatic vascular occlusions were excluded from this study. The collected data was reviewed retrospectively including age, sex, associated, comorbidities, time before initial symptoms and outcome after embolectomy.

Results Ninety seven patients were admitted to the vascular ward with signs and symptoms of ALI. The mean age was 56 years, 51.5% female and 48.5% male. All of them underwent emergency embolectomy. The cause of vascular occlusion was embolism in 72 patients and thrombosis in 12 patients. The limb salvage rate was 88.65%, Eleven patients ended with limb amputation. Complication occurred in 31 patients in the form of: ten patients hematoma, nine patients SSR, five patients re embolectomy, four patients fasciotomy, two patients foot drop and one patient AKI. Thirteen patients died with hospital mortality rate of 13.4%. There was a significant correlation between the time from the initial symptoms to the surgical intervention and limb amputation rate (P value = 0.0009).

Conclusions Embolectomy is an important procedure in the management of patients with ALI with a high limb salvage rate up to 88%. Time interval between the initial symptoms of vascular occlusion and surgical intervention is an important predictor to the limb's outcome after embolectomy; the earlier the treatment the better the results.

Introduction

History

In 1628 the concept of vascular occlusion was presented by William Harvey: "whenever the motion of the blood in the arteries is impeded, by compression, by infarction, or by interception, there is less pulsation distally, since the beat of the arteries is nothing else than the impulse of blood in these vessels''. Embolectomy procedure was proposed by John Hunter in 1768 as a method to restore blood flow. More than a century later Georges Labey carried out the first successful embolectomy. There were many devices proposed to facilitate clot removal, such as the corckscrew wire of Shaw, retrograde milking with Esmarch bandage and endovascular suction catheters of Dale and local removal through multiple and lengthy arteriotomies. However, none of these surgical techniques was particularly effective in reducing the high morbidity and mortality associated with acute arterial occlusion[(1)].

There were two main developments that occurred in the twentieth century which led to a rapid improvement in limb saving surgery for acute arterial iscahemia. Firstly, the anticoagulant heparin made it possible to limit the propagation of a clot distal to the point of occlusion and to minimize the occurrence of recurrent embolus and thrombosis. Secondly, the invention of the Fogarty balloon catheter in 1963 by Tom Fogarty make it possible for extraction of emboli and thrombi from occluded vessels[(2)].

Etiology

Risk Factors of Acute Limb Ischemia

The possibility that a patient has a vascular disease depends in part on their demographics and underlying clinical conditions[(3)].

Non modifiable risk factors: Age Gender Ethnicity Chronic Renal Insufficiency Family history of PAD Modifiable risk factors :Smoking Hypertension Diabetes Mellitus type 2Dyslipidemia Sedentary lifestyle Hyperhomocysteinemia_Hypercoagulable state.

Causes of Acute Limb Ischemia

Acute limb ischemia is the result of sudden occlusion in the arterial supply to the limb. Arterial embolism and thrombosis are the two main reasons for acute limb ischemia, excluding trauma and iatrogenic causes, table 1.1.

Embolism

Embolism (from the Greek *embolos*, or "plug") is the result of material passing through the arterial tree and occluding the distal artery. The embolus is usually of cardiac origin and its material is mural thrombus that has accumulated and detached.

The other main cause is atherosclerotic debris from a diseased proximal artery, often the thoracic aorta, in individuals with a heavy burden of atherosclerotic disease.

Thrombosis

Thrombotic occlusion is most commonly the result of progressive atherosclerotic narrowing in peripheral arteries. Once a stenosis becomes critical, platelet thrombus develops on the stenotic lesion, leading to an acute arterial occlusion[(4)].

Table(1). Etiology of Acute arterial occlusion [2 :p.406].

Embolic
Cardiac
Atrial Fibrillation
Valvular Heart Disease (Rheumatic Heart Disease Or Endocarditis)
Myocardial Infarction (With Or Without Ventricular Aneurysm)
Prosthetic Heart Valves
Left Atrial Myxoma
Paradoxical Embolus
Congestive Cardiomyopathy
Hypertrophic Cardiomyopathy
Mitral Annulus Calcification
Mitral Valve Prolapse
Non Cardiac
Ulcerating Atherosclerotic Lesions
Aneurysms (Aortic, Iliac, Femoral Popliteal, Subclavian , Axillary)
Arterial Catheterization Complications
Thrombotic
Narrowed Atherosclerotic Segment (With Or Without A Flow-Related Disorder)
Intraplaque Hemorrhage
Drugs Of Abuse
Vasospasm
Bypass Graft Occlusion
Aortic Or Arterial Dissection

Pathophysiology

Theories of Atherogenesis

There is an abundance of theories regarding the pathogenesis of atherosclerosis; monoclonal hypothesis, Lipid hypothesis, inflammatory theory, Lesion regression, unstable plaque and the response to injury theory. The "response to injury" theory is one of the hypotheses that bring together all other theories with a wide range of context. The response-to-injury hypothesis initially suggested that endothelial denudation was the first step in atherosclerosis. More recent data suggest that endothelial dysfunction rather denudation is the primary problem. According to this theory, atherogenesis is a response to injury of the vascular endothelium. At its mildest, atherosclerosis may represent a reparative process that leads to thrombus formation and smooth muscle cell proliferation at the site of endothelial injury. The production of the endothelial injury may be from hypertension, cytotoxic molecules, or blood flow changes .Atherosclerosis develops at sites exposed to unusual shear stress, such as in the abdominal aortic bifurcation. Hypercholesterolemia, hyperhomocystinemia and smoking may all contribute to endothelial injury[(5)].

The Atheroma

Irrespective of the initiating process, the first lesion of arteriosclerosis happens with the entry of LDL through the intima and into the arterial wall.

These early lesions, named (fatty streaks), are slightly raised yellow lesions found in the aorta of infants and children. The lipid deposits in these lesions are found inside macrophages and smooth muscle cells .Foam cell macrophages which sequester lipid particles, are characteristic of these early lesions. It is believed that these lesions represent the precursor to more advanced atherosclerotic lesions. As these lesions grow they then intrude into the arterial lumen.

Fibrous plaques consist of large numbers of smooth muscle cells and connective tissue forming a cap over an inner core containing mainly lipid cholesterol esters thought to be from disrupted foam cells. The fibrous cap may provide structural support or may work as a barrier to sequester thrombogenic debris in the underlying plaque from the arterial lumen. These plaques can express evidence of irregular and periodic growth. Ulceration and healing may happen intermittently, and there is evidence that thrombi made on lesions are merged into them and resurfaced with a fibrocellular cap and an intact endothelial layer. Whether all fibrous plaques are characteristic of advanced atherosclerosis developing from the fatty streak is unclear. However, fibrous plaques often appear chronologically after fatty streaks in the same anatomical locations and characterize clinically obvious atherosclerosis.

Complicated plaques comprise the end stage of atherosclerosis and cause clinical symptoms. These are fibrous plaques that have become calcified, ulcerated, or necrotic. In most patients

ischemia occurs as a result of erosion or uneven thinning and rupture of the fibrous cap, often at the shoulders of the lesion where macrophages enter and accumulate. Degradation of the fibrous cap may occur by release of metalloproteinases, collagenases, and elastases by these cells[6].

Emboli of cardiac origin tend to occlude bifurcations of large arteries where the luminal diameter suddenly decreases. These arteries are usually more than 5mm in diameter. Atheroemboli, consisting of debris from atheromatous lesions of the proximal arterial system, are smaller and occlude vessels less than 5mm in diameter. The size of the obstructed vessel can thus help in distinguishing whether the emboli originated from the heart, aorta or common iliac arteries. As soon as an embolus lodges in an artery, or once a thrombus occludes a previously diseased artery, the vasculature distal to the obstruction goes into spasm. Clot then forms proximal to the site of the obstruction, back to the point of sufficient collateralization. The distal spasm takes approximately 8 hours and then subsides. At this time clot forms in the arterial system distal to the site of obstruction and propagates downward, blocking any residual collateral flow, resulting in worsening of the ischemia. As a result the skin usually becomes patchy, blue, and mottled. Skeletal muscle and peripheral nerves tolerate acute ischemia for around 8 hours without permanent damage; skin can tolerate severe ischemia for as long as 24 hours. The degree of the ischemic necrosis depends on the patency of collateral circulation, the patient's underlying cardiovascular function, blood viscosity, oxygen-carrying capacity of the blood, propagation of clot into the microvasculature, and efficiency and promptness of treatment. If muscle ischemia progresses to necrosis, the muscle becomes paralyzed and gains a rigid, spastic consistency. When peripheral nerves become ischemic, they stop to function, and the affected parts become anesthetic.

As the skin suffers profound ischemia, maximum oxygen extraction results in a cyanotic, blotchy look. When these blotchy, cyanotic regions no longer blanch with pressure, the skin is gangrenous and the ischemia is irreversible[7].

Presentation

The size of the occluded artery and the presence of collaterals are the main factors that determine the presenting symptoms. Abrupt occlusion of a proximal artery without present collaterals leads to an acute white leg, whereas occlusion of the superficial femoral artery in the presence of well-established collaterals may be completely asymptomatic. Loss of sensation is one of the earliest signs of acute leg ischemia because acute ischemia affects sensory nerves first. Motor nerves are then affected, causing muscle weakness; then skin and finally muscles are affected by the reduction in arterial perfusion. This explains muscle tenderness as one of the end-stage signs of acute leg ischemia. When ischemia is established, the initial skin pallor changes dusky blue as capillary venodilatation occurs. Pressure over the discolored skin leaves it white because the vessels are still empty at this time. The end stage of skin ischemia is caused by blood leak through the disrupted capillary .digital pressure over the discolored skin produces no blush. At this stage, the skin is nonviable, and revascularization of necrotic tissue risks compartment syndrome and renal failure without saving the extremity[8].

Work up and Treatment

Patient History

The severity of the initial symptoms depends on the severity of ischemia and can range from sudden onset of mild claudication to the devastating pain. Severe acute ischemia is usually noticeable, with severe pain and loss of sensation and movement in the limb .mild ischemia can be challenging to diagnose and may be confused with musculoskeletal pain, sciatica, and other causes of limb discomfort. The onset of symptoms is the most significant part of the history, irreversible muscle necrosis occurs within 6 to 8 hours if the condition is untreated.

Historically, patients with emboli had valvular heart disease but no evidence of peripheral vascular disease or other atherosclerotic conditions; however, the presence of atherosclerosis no longer rules out embolism. Patients with acute-on-chronic thrombosis often give a history of prior intermittent claudication in the ipsilateral or contralateral leg. A full medical history is important because it may reveal other associated diseases such as diabetes mellitus. Risk factors for atherosclerotic disease should be considered, including smoking, hypertension, hyperlipidemia, and family history.

Physical examination

It is essential to examine the leg in order to define the severity of the ischemia. The famous rule of six Ps: pain, pallor, paresis, pulse deficit, paresthesia, and poikilothermia remains a good guide to both symptoms and signs. The skin color reflects its vascular supply. Marble white skin is concomitant with acute total ischemia. Slow capillary refill is a sign that poor distal flow is present and runoff vessels are probably patent. Aesthesia, paraesthesia and loss of fine touch and proprioception should all be determined. Calf muscle tenderness is a definite sign of advanced ischemia. Peripheral pulses should be examined to define the level of arterial occlusion. Good pulse volume in the other limb suggests embolism is a possible cause. Atrial fibrillation, aortic or popliteal aneurysm is potential causes for embolization which may be evident during examination. A full physical examination should be performed for patients with acute limb ischemia to exclude associated comorbidities that may affect the prognosis[(9)] .

Investigation

Acute limb ischemia is usually critical and may require urgent surgical intervention thus giving no time for investigation. However, time permitting; several methods may be used to ascertain the site and nature of vascular occlusion[(10)].

Hematocrit, PT/PTT, platelets
Electrolytes, BUN, creatinine, glucose
Urinalysis—test for myoglobinuria
CPK with isoenzymes
Chest X-ray
Echocardiography
CT angiography
Magnetic resonance angiography

Treatment

Once the diagnosis of acute limb ischemia has been established and its severity classified (table 2), a number of immediate interventions are important to improve limb saving and patient outcomes[(11)].

Anticoagulation and Supportive Measures

To minimize the risk of additional clot propagation and to avoid microvascular thrombosis of hypoperfused distal vessels, Systemic anticoagulation with unfractionated heparin should be initiated. A starting weight-based bolus of 100 mg/kg is suitable for most patients followed by an intravenous infusion of 1000 U/hr. If surgery is postponed, the heparin dose should be titrated to maintain an activated partial thromboplastin time between 60 and 100 seconds or 2.0 to 3.0 times normal values. Patients with acute ischemia are often relatively volume depleted, and careful fluid resuscitation is necessary, with fluid administration titrated to urine output. Intravenous analgesia and supplemental oxygen are also important supportive measures for these patients.

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Table (2)Classification of acute limb ischemia [4:p.2523]

		Findings		Doppler Signals	
Category	**Description/Prognosis**	**Sensory Loss**	**Muscle Weakness**	**Arterial**	**Venous**
I. Viable	**Not Immediately Threatened**	**None**	**None**	**Audible**	**Audible**
Ii.Threatened A. Marginally	**Salvageable If Properly Treated**	**Minimal (Toes) or None**	**None**	**Inaudible**	**Audible**
B. Immediately	**Salvageable With Immediate Revascularization**	**More Than Toes, Associated With Rest Pain**	**Mild, Moderate**	**Inaudible**	**Audible**
Iii. Ireversible	**Major Tissue Loss or Permanent Nerve Damage Inevitable**	**Profound Anesthetic**	**Profound, Paralysis (Rigor)**	**Inaudible**	**Inaudible**

Endovascular Treatment

Endovascular procedures offer less invasive revascularization strategies for sick or elderly patients with decreased morbidity and mortality. Currently available percutaneous endovascular procedures include

Catheter-directed thrombolysis CDT

Pharmacomechanical thrombolysis

Catheter-directed thrombus aspiration

Percutaneous mechanical thrombectomy.PMT

These techniques clear the occluding thrombus from a peripheral artery with a minimally invasive approach, restore blood flow to the extremity, and allow the identification of underlying lesions responsible for the occlusive event. Targeted lesions may then be addressed in a directed fashion with an endovascular procedure such as angioplasty, stenting, or atherectomy.

Surgical Revascularization

The thrombectomy or embolectomy balloon catheter is the cornerstone of therapy for the surgical management of acute limb ischemia (ALI). Severe ALI (class IIb), manifested by both sensory and motor deficits, requires urgent intervention, and surgical therapy has remained the treatment of choice. With the increasing availability of hybrid operating rooms, surgeons can now perform diagnostic imaging, endovascular intervention, open surgical revascularization, and hybrid procedures in single settings

Balloon catheter thrombectomy or embolectomy

Bypass procedures to direct blood flow beyond the occlusion

Endarterectomy with or without patch angioplasty

Hybrid procedures combining open and endovascular techniques.

Aim of the Study

This study was designed to discuss the effectiveness of embolectomy procedure in the management of acute vascular occlusion in our center and discussing the outcomes with other local, regional and national studies.

Patients and Method

A retrospective study was carried out from October 2014 to October 2017 at AL Sadder teaching hospital in Al Basrah city. A total of ninety seven patients were admitted to the vascular ward with signs and symptoms of acute vascular occlusion. Traumatic vascular occlusions were excluded from this study.

The data was collected from the clinical database of Al Sader teaching hospital and reviewed retrospectively including: age, sex, associated comorbidities (smoking, diabetes, HTN, IHD, renal failure), time before initial symptoms and outcome after embolectomy.

All patients had ECG, CBC, RBS, BUN, serum creatinine at time of arrival to the emergency department. Doppler study was done for some patients, while all patients were unable to underwent Echocardiography and CT angiography because of their urgency for surgery.

The initial management started at the emergency department with oxygen supplementation, sufficient IV fluid resuscitation, IV analgesia and heparin with 100 IU/Kg starting dose intravenously.

The decision for embolectomy was made by the most senior surgeon depending on the history (time of onset of the initial symptoms) and clinical presentation (limb viability).

All embolectomy procedures were performed under local anesthesia 2% Xylocaine ,some patients required superadded sedative treatment for their intractable pain.

The embolectomy procedure was performed through exposure of the common femoral artery via a vertical infrainguinal incision, brachial and popliteal arteries exposed with a lazy S shape incision, followed by proximal and distal control of the targeted artery with a rubber tape. Heparin 100 IU/kg was given IV before the transverse arteriotomy. The proper size Fogarty catheter was introduced as required proximally and then distally, the balloon then inflated with

an appropriate amount of saline according to the changing diameter of the vessel. Proper traction was applied until all thrombus or embolus were cleared out and accepted forceful pulsatile blood flow was obtained. The distal artery was flushed with heparinized saline routinely via irrigation catheter. The arteriotomy was closed with prolyne interrupted stiches and meticulous hemostasis was achieved. Patients were kept on IV Heparin 100 IU/kg /6 hourly for the 4-5 postoperative days superadded with Aspirin, proper Antibiotics and Analgesia covered was ensured.

Statistical analysis of data was expressed in the form of means and percentage. Statistical analysis was performed using ANOVA and MANOVA.

P value < 0.005 considered significant.

Results

There was a total Ninety Seven patients in this study with 50 female patients (51.5%) and 47 male patients (48.5%), figure 1.

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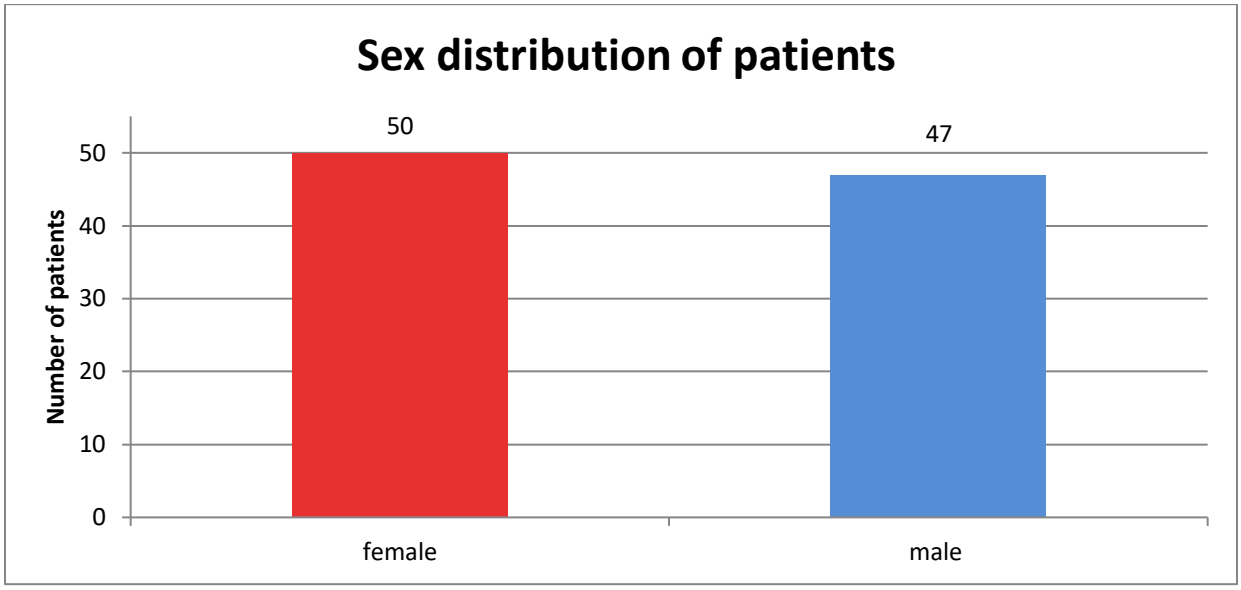


Figure (1). Sex distribution of patients

The youngest patient was 23 years old, the oldest one was 90 years old and most of the patients were between 53-63 years of age. The mean age was 56 years old (figure 2).

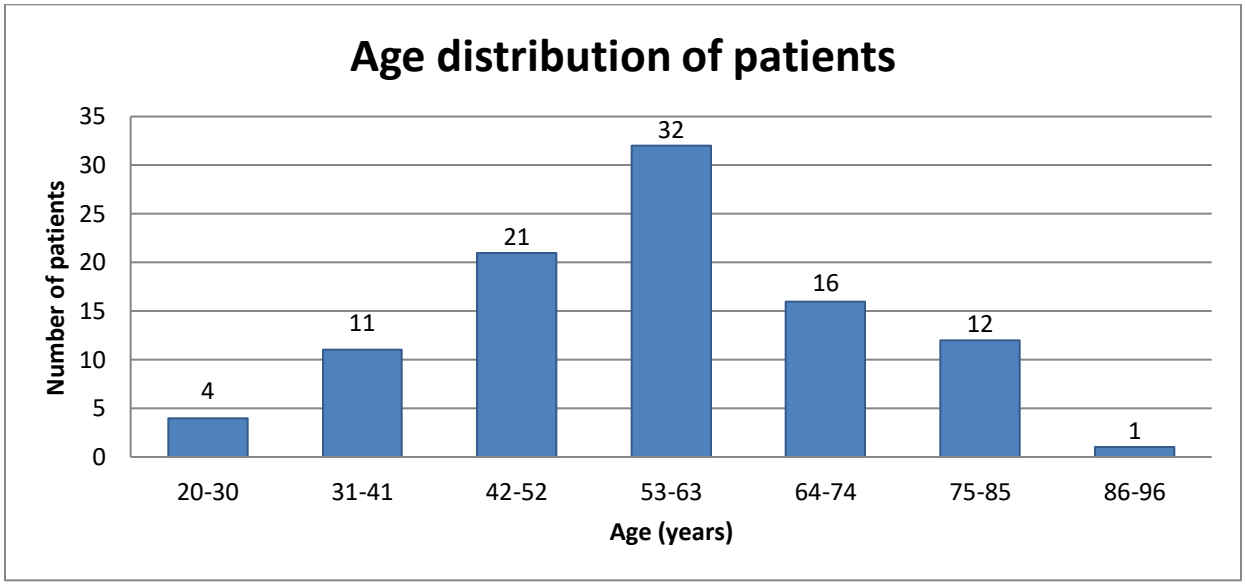


Figure (2). Age distribution of patients

Pre-operative demographic data for our 97 patients show a relationship between acute limb ischemia with the advanced age (mean age 56), hypertension, ischemic heart disease, arrhythmia (AF), smoking and DM.

Table (3). Preoperative demographic data of ninety seven (97) patients.

Risk Factors	Number of Patients	Percentage
Sex	50 Female 47 Male	--------
Hypertension	36	37.1
Diabetes Mellitus	28	28.8
Ischemic Heart Disease	49	50.5
Chronic Kidney Disease	9	9.2
Arrhythmia AF	48	49.48
Cancer	15	15.4
Smoking	45	46.3
Stroke	20	20.6

The most common presenting signs and symptoms were pain, cold extremities and absent peripheral pulses which were present in all our 97 patients, while 72 patients show additional signs of ischemia such as pallor and cyanosis, parasthesia was reported in 41 patients and paralysis in 24 patients(figure 3).

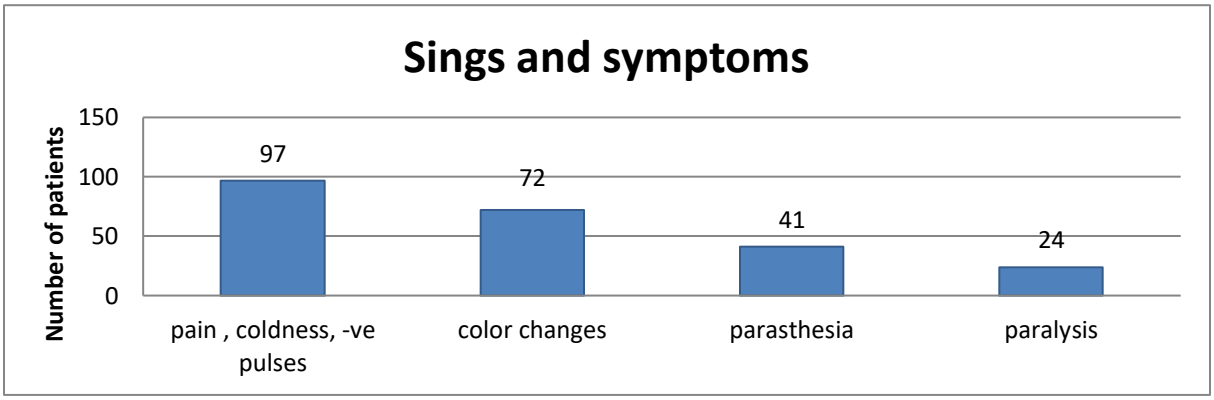


Figure (3). Presenting signs and symptoms of patients

The 97 patients whom presented with ALI are subdivided according to the site of occlusion into two groups upper and lower limb ischemia, with lower limb predominance 67%(figure 4).

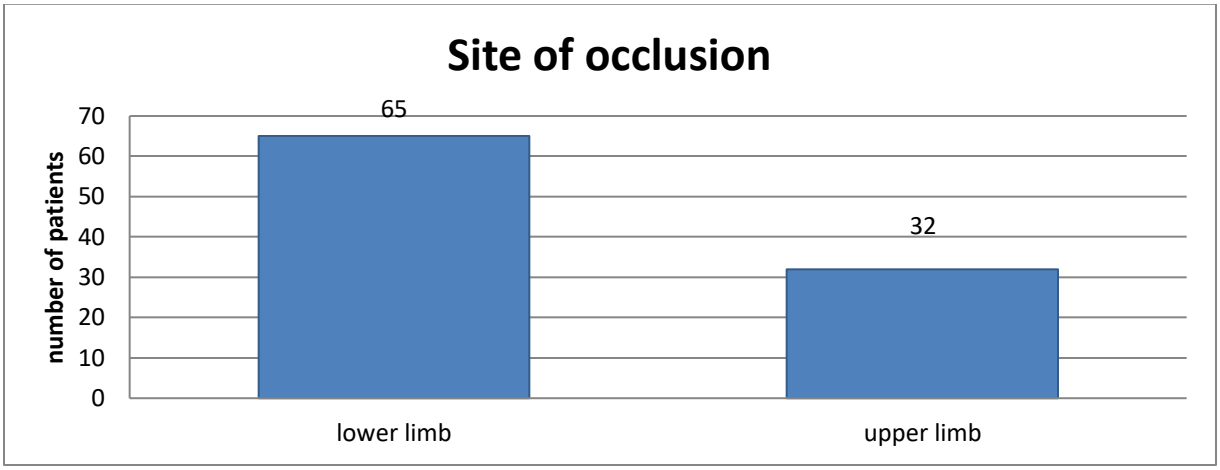


Figure (4). Site of vascular occlusion

The majority of embolectomy site was the femoral artery in 50 patients followed by the brachial artery in 32 patients, then the popliteal artery in 15 patients (table 3).

Table(4). Distribution of embolectomy sites amongst patients

Embolectomy site	No. of patients	%
Femoral	**50**	**51.54**
Brachial	**32**	**32.98**
popliteal	**15**	**15.4**
Total	**97**	**100**

The Operative finding of embolectomy showed that the mainstream of vascular occlusion caused by well-defined embolus in 77 patients (74.69%), while acute arterial thrombosis was the cause of vascular occlusion in 20 patients(19.4%)(table 4).

Table(5). Operative findings

Type of occlusion	No. of patients	%
Embolic	77	74.69
Thrombosis	20	19.4
Total	97	100%

Cardiac embolism was established as a cause of vascular occlusion in 70 patients (72.16%);26 patients had atrial fibrillation, 20 patients had history of recent myocardial infarction,21 patients had history of rheumatic heart disease and three patients had prosthetic valve and discontinued their medication. Two patients had non-cardiac embolization by peripheral aneurysm. Twelve patients had acute thrombosis on chronic atherosclerosis. While 13 patients had unclear cause of vascular occlusion (table 5).

Table (6). Causes of vascular occlusions

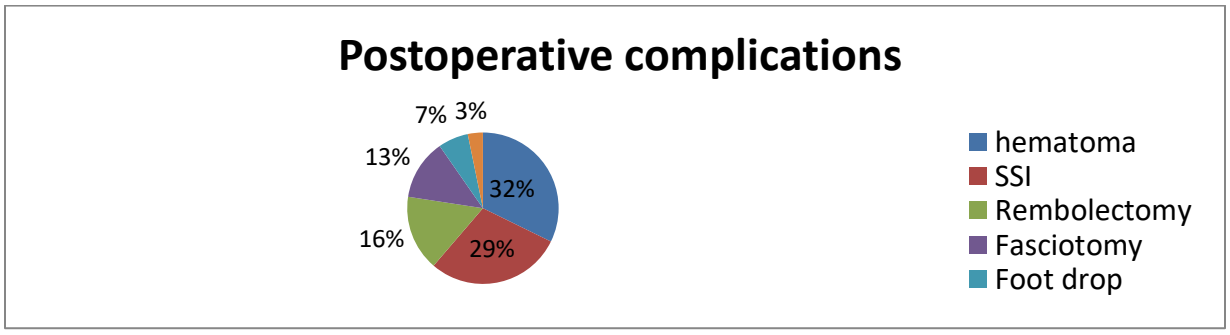
Causes of vascular occlusion	No. of patients	% of patients	Total	
			No.	%
1) Cardiac embolization			70	72.16
2) Atrial fibrillation	26	26.8		
Recent Myocardial infarction	20	20.6		
Rheumatic heart disease	21	21.6		
Prosthetic valve	3	3.09		
3) Non cardiac embolization Peripheral aneurysm	2	2.06	2	2.06
4) Thrombotic (acute on chronic atherosclerosis)	12	12.37	12	12.37
5) Unrecognized cause	13	13.4	13	13.4
Total			97	100

The time from the onset of symptoms to the surgical intervention was an important predictor to the outcome of limb salvage, as the results show that 46 patients whom underwent surgical intervention at the first 6 hours from the onset of symptoms had the highest limb salvage rate of 93.47%. On the other hand, the percentage of limb amputation increased with more delay for surgical intervention (31.25%). ANOVA study was carried out on the results and produced a P value of 0.0009 which showed a significant relationship between the time factor and amputation rate, table 6.

Table(7). Relationship between time interval and limb outcome.

Time interval for embolectomy	Patients		Limb salvage		Limb amputation		P-value
	No.	%	No. of patients	%	No. of patients	%	
0-6 hours	46	47.4	43	93.47	3	6.5	
7-12 hours	24	24.47	22	91.66	2	8.3	
13-24 hours	11	11.34	10	90.9	1	9.09	
>24 hours	16	16.49	11	68.75	5	31.25	
Total	97	100%	86	88.65%	11	11.34 %	0.0009

The postoperative complications occurred in 31 patients (31.95%), in the form of; hematoma in ten patients, SSI occurred in nine patients, five patients developed ischemic limb that required re-embolectomy, four patients developed compartment syndrome which was relieved by fasciotomy, two patients developed foot drop and one patient resulted with AKI, figure 5.



Figure(5). Pie chart showing distribution of postoperative complication

The embolectomy outcome in 97 patients was effective in limb salvage in 86 patients (88.65%), amputation was performed in 11 patients (11.34%) who had life threating risk factors; these included seven patients who had ongoing ischemia with well demarcation line despite successful primary embolectomy,three patients developed fasciotomy site infection and one patient had reperfusion syndrome. Thirteen patients died in the post-operative period with a hospital mortality rate of 13.4%. The mortality cases included patients who were particularly old age and mostly with cardiac problems.

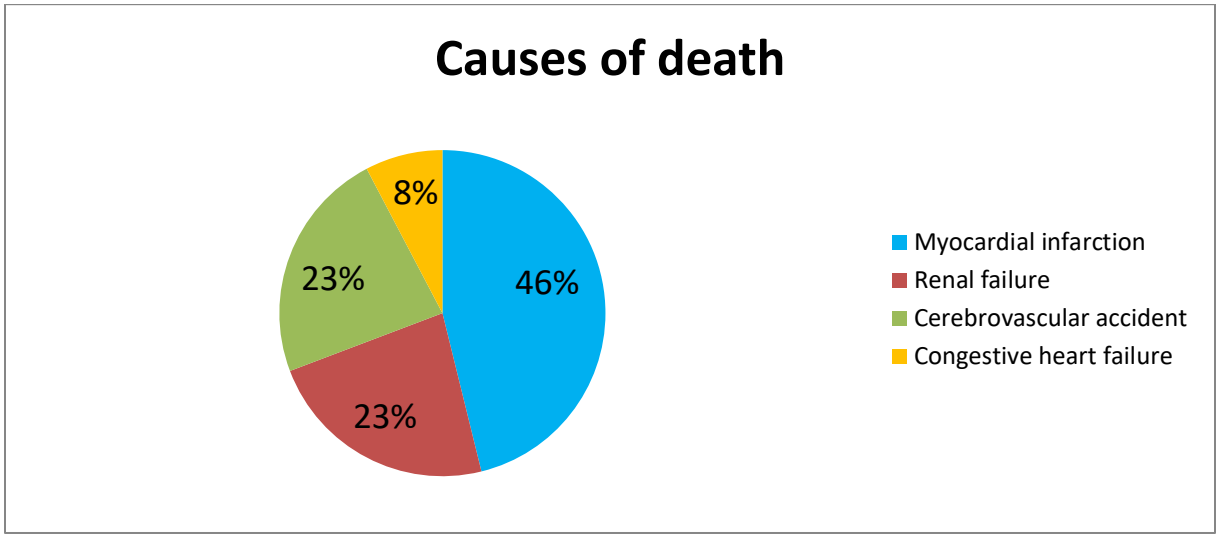


Figure (6) Pie chart showing the causes of postoperative death.

Discussion

Acute vascular occlusion is a critical condition that requires proper history and physical examination with urgent medical and surgical intervention. Despite the efforts to optimize patients prior to surgery, patients with ALI remain to be a high-risk group with significant morbidity including limb loss as well as mortality. This is explained by the fact that this cohort of patients has elevated acuity of sickness at time of presentation due to their associated comorbidities Morris-Stiff[12]. Acute arterial occlusion resulting in limb ischemia may be caused by a many conditions but most often is the result of an arterial embolism or thrombosis of a previously diseased vessel Brewster[13].

In our study 97 patients presented with ALI. They had no significant sex distribution with 50 female patients (51.5%) and 47 male patients (48.5%) which is similar to the sex distribution of Mozaffar [14]and Satiani et al. [15]studies.

Majority of patients were between (53-63) years of age. The more advanced age of patients, the more risk factors and co morbidities were present and so have more chance to develop PAD ,this go with studies of Criqui et al. [16], Hirsch et al. [17] and Norgren et al [18].

The demographic data of 97 patients showed that the advanced age, smoking, IHD, arrhythmia (AF), hypertension and diabetes mellitus are the major risk factors for ALI and that was similar to Scott [19]and Fagundes[20] studies.

Pain, cold extremities and absent peripheral pulses were the main presenting signs and symptoms that all our patients had; 72 patients had additional symptoms as paler and cyanosis, neurological symptoms like parasthesia and paralysis reported in 41 and 24 patients

respectively. This variation in presentation of acute vascular occlusion is probably due to the differences in the collaterals availability, the extent of atherosclerotic occlusive disease and the risk factors of individuals ,this variation in presentations met with the study of Steurer and Schneider [21].

The lower limbs were the main site of vascular occlusions in our study with 67% (51.54% Femoral artery, 15.4% popliteal artery). This can be explained by the fact that emboli usually lodge at the bifurcation of great vessels where the diameters abruptly narrows Earnshaw [4]and Satiani [15].

The diagnosis of ALI and decision making for embolectomy in our study depended on the history, physical examination and the use of a portable Doppler in some suspicious cases. CTA or MRA was difficult to be obtained in our institute and needs more time to be done, so these diagnostic tools were ruled out from our routine evaluation of patients with ALI.

Embolism was the foremost cause of ALI in our study with 74.22%; cardiac embolism had the bulk with 72.16% including AF, recent MI, prosthetic valve and rheumatic heart disease. While 2.06% of the embolisms were non cardiac and were caused by peripheral aneurysms, this results was documented by ECG ,Doppler study and Echocardiography done in the pre and postoperative period.

On the other hand, thrombosis represented 12.37% of the total cases with ALI that required embolectomy.

The remaining patients (13.4%) had unrecognizable cause of ALI due to post-operative death or patient refusal for further post-operative evaluations and leaving the hospital on their responsibilities. These results are compatible with Morris-Stiff et al[12], Satiani et al [15] and Galbraith et al [22].

There is a strong relationship between the delay from the onset of symptoms to the revascularization and subsequent limb loss. Morris-Stiff and col- leagues[12]recently compared the results after peripheral arterial embolectomy from two different historical time periods in the same community hospital. In both time periods, patients with an interval between symptoms and treatment of less than five hours had a more favorable outcome than those who suffered a longer delay. In our study there was a significant correlation between time interval for embolectomy and limb amputation P value < 0.0009 ;46 patients (47.4%) underwent embolectomy within six hours from the beginning of symptoms, 43 patients of them had limb salvage (93.47%), while three patients resulted with amputation (6.5%) due to secondary thrombosis. Surgical intervention delayed over 24 hours from onset of symptoms resulted in a higher amputation rate (31.25%). This result is comparable with the local study of Alaa A. Abd Alwahid [24] .

The embolectomy outcome in our 97 patients was effective limb salvage in 86 patients (88.65%), while 11 patients (11.34%) ended with amputation ,this is comparable with study of Scott [19], Vohra [25] and Pemberton [26].

The post-operative complications occurred in 31 patients (31.95%), hematoma being the most common developed in 10 patients (10.3%); this can be explained by the use of heparin in the post-operative period. Nine patients (9.2%) developed SSI which was an acceptable percentage for elderly high risk patients for whom embolectomy performed Green et al [1], Mozaffar et al[14] , Iyem and Eren [27].

Five patients required second time embolectomy due to residual clots or re embolization and subsequent thrombosis. Four patients had compartment syndrome that required fasciotomy to relive the swollen limb to prevent permanent neurological damage. Two patients had foot drop due to infarction of the anterior compartment of the leg due to delayed ischemia time. One patient developed AKI because of reperfusion syndrome. Most of our results of the post-operative complications met with Vohra[25]and Haimovici [28]studies.

Thirteen patients died in the postoperative period with a hospital mortality rate of 13.4% most of them was elderly and had cardiac problems, which is comparable with the mortality rates of other studies of Galbraith et al [22], Wolosker et al[29] , Neuzil et al [30], Dormandy et al [31]and Von Allmen et al [32].

Conclusion and Recommendations

Embolectomy can be considered a simple procedure that can be performed under local anesthesia to patients with acute vascular occlusion, with a high limb salvage rate of up to 88% and reduced number of amputations and comorbidities.

Time interval between the initial symptoms of vascular occlusion and the surgical intervention is an important predictor to the outcome of the limbs after embolectomy; the earlier the treatment the better the results.

Patients with acute vascular occlusion have different comorbidities and acute limb ischemia represents only the tip of the iceberg, this explains the elevated death rate despite proper peri-operative interventions.

Recommendations

We recommend early and fast referral of patients with ALI to vascular surgeons, so that proper management and intervention would reduce the patients' morbidities.

Improve public awareness about the signs and symptoms of acute limb ischemia and how time is an important factor for limb salvage.

Encourage exercise, smoking cessation programs and proper diabetic and hypertension control to reduce the risk factors of vascular disease.

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