

NEWSLETTER

JANUARY - MARCH 2019

KULSUM



- HEART ATTACK DEPRESSION
- TRUE BRACHIAL ARTERY ANEURYSM
IN A CHILD AGED 2 YEARS

We understand your

HEART

thoroughly



Cardiology Procedure

1st Case

Abbreviations

STEMI - ST Elevation Myocardial Infarction

MI - Myocardial Infarction

PCI - Percutaneous Coronary Intervention

LAD - Left Anterior Descending

TIMI - Thrombolysis in Myocardial Infarction

GP - Glycoprotein

DES - Drug-Eluting Stent

29 years old male presented with severe crushing chest pain and Acute STEMI was referred from a well-reputed cardiac hospital. He had a positive family history of ischemic heart diseases.

His cardiac enzymes were significantly elevated and Echo showed Antero-Apical Hypokinesia with ejection fraction of 50%. No other abnormality noted in Echo.

Patient was taken to Cath Lab for Primary PCI. His Coronary Angiography showed huge mobile thrombus with filling defect & sluggish flow in LAD with atheroma in proximal segment. Other coronaries were normal.

Keeping in view a mobile Ostio-Proximal LAD thrombus, our Cardiologist became suspicious of an embolic event and therefore used Thrombuster II (Thrombus Aspiration Catheter) which aspirated a big serpiginous red clot and small white thrombus. This achieved a good TIMI III flow in LAD and patient became symptom free with resolution of ECG changes.

No stenting was performed and patient was started on Intravenous Aggrastat (GP IIB/IIIA Inhibitor). Moreover, complete thrombophilia screen was sent out considering an embolic event at such a young age.

2nd Case

Abbreviations

LV - Left Ventricular

ETT - Exercise Tolerance Test

LAD - Left Anterior Descending

RCA - Right Coronary Artery

LCx - Left Circumflex

PCI - Percutaneous Coronary Intervention

CTO - Chronic Total Occlusion

POBA - Plain Old Balloon Angioplasty

TIMI - Thrombolysis in Myocardial Infarction

54 years old male with the background of hypertension was presented with exertional and post prandial angina for 5 months. Good LV function observed on Echo but ETT became positive for Ischemia.

Angiography showed calcified totally blocked LAD and faint contralateral collaterals distally. Also, mild to moderate disease was observed in RCA and LCx respectively.

PCI of CTO-LAD and POBA to D2 were performed by engaging left system with BL 3.06 guided catheter. Good results were obtained with TIMI III flow in LAD and D2. Patient became asymptomatic and discharged next day in a stable condition.



KIH - A Leader in Cardiac Services

Next day this case was discussed with panel of cardiologists and a restudy was performed which showed mild haziness in Proximal LD with the background of Atheroma. Therefore, it was stented with one DES with good results.

To rule out any recurrence, patient was kept under observation for two days and discharged with the follow up visit for bubble study and routine assessment after two weeks.

Bubble study was performed and became negative but the patient found to be positive for Protein S Deficiency. Patient was counseled about the disease and advised warfarin for life time along with dual antiplatelet therapy for at least 6 months.



POST - HEART ATTACK DEPRESSION

Dr. Beena Mamoon

Keywords

AMI: (Acute Myocardial Infarction) **CAD:** (Coronary Artery Disease)
CHD: (Coronary Heart Disease) **HRV:** (Heart Rate Variability)
SSRIs: (Serotonin Re Uptake Inhibitors) **BDI:** (Beck Depression Inventory)
LOS: (Length Of Stay)

A number of studies have demonstrated a relationship between depression and low perceived social support and increased cardiac morbidity and mortality in patients with coronary heart disease. There is also an evidence that depression increases the risk of AMI. Depression increases hospital length of stay, procedures, readmission rates, and the cost of medical care.

In a developing country like Pakistan and especially in the low-income population, financial considerations may play a role in the genesis of depression in CAD patients. Those patients who believed that their disease was responsible for changes in their behavior also had higher prevalence of depression as compared to those who did not believe so (1). Recent studies have identified depression and depressive disorders as risk factors for AMI and as being associated with increased Post-AMI morbidity and subsequent mortality (2,3) yet, the influences of depression on AMI have not been widely appreciated in clinical practice (4,5).

Most current studies of depression define it according to the criteria established by the American Psychiatric Association. These criteria are listed in association's Diagnostic and Statistical Manual of Mental Disorders. Following nine symptoms must be present for diagnosis (6); (i) depressed mood (ii) diminished interest especially in pleasure (Anhedonia) (iii) weight loss or gain more than 5% with increase/decrease in appetite (iv) insomnia or hypersomnia (v) psychomotor agitation or depression (vi) fatigue (vii) feeling of worthlessness or guilt (viii) diminished ability to concentrate (ix) recurrent thoughts of death or suicide. Depression was suggested to increase the risk of sudden cardiac death after AMI by Irvine et al (7).

Clinical experience now suggests that all who experience an AMI usually fall into one of two categories (i) Those who view the event as a major life disaster from which they will never recover (ii) Those who have a positive attitude about recovery. It is among the former group that depression can occur, accounting for the high incidence of this comorbidity among all AMI patients.

Exact mechanism is not fully clear. A number of biological mechanisms have been proposed, like increased sympathetic activity. On the other hand, as postulated by Alexopoulos et al (8), depression may be the result of Cerebral Arterial Atherosclerosis. It is also possible that depression and cardiovascular disease may share common causes such as dysregulation of the serotonin transporter (9) immune activation (10), or reduced dietary intake of Omega fatty acids (11).

Diminished ejection fraction after AMI is known to be correlated with a higher mortality. Fifty percent of patients with depression post-AMI have been noted to have a decreased ejection fraction compared with 33% of non-depressed patients ($p=0.084$) (12). Hypercoagulability in depression may occur via activation of procoagulant factors (Fibrinogen, Von Willebrand factor, Factor VII) and a reduction of fibrinolytic activity by tissue type plasminogen (13). In addition, Lederbogen et al [14] found increased platelet activation in patients with depression, further accounting for increased coagulopathy and coronary artery obstruction.

A loss of heart rate variability (HRV), a known sign of poor prognosis in CHD, is independent of other risk factors. Decreased autonomic control of HRV is associated with depression but not anxiety and is reversed by β blockers (15).

Magnetic Resonance Imaging (MRI) studies have also demonstrated changes in the Prefrontal Cortex and Amygdala as well as the Hippocampus. Vascular endothelial dysfunction and its related endothelial-dependent, flow-mediated vasodilation is impaired in depressed patients. Abnormalities have also been found in the regulation of neurotransmitters (chemicals released in body), including serotonin, norepinephrine, dopamine, glutamate, and neuropeptides, such as cortisol, corticotrophin-releasing hormone, neuropeptide Y, and substance P^(16,17)

SSRIs (Serotonin Re uptake Inhibitors) have been shown to be beneficial in the treatment of depression⁽¹⁸⁾. The SSRI sertraline not only provide treatment of depression but also exhibit significant dose-dependent inhibition of human platelets, thereby canceling the adverse platelet effect of depression⁽¹⁹⁾. Sertraline was shown to be effective and safe in the treatment of depression accompanying recent AMI in the Sertraline Anti-Depressant Heart Attack Randomization Trial (SADHART)⁽²⁰⁾.

A loss of (HRV), a known sign of poor prognosis in CHD, is independent of other risk factors. Decreased autonomic control of HRV is associated with depression but not anxiety and is reversed by β blockers⁽¹⁵⁾.

There is no laboratory test for depression. The diagnosis requires a careful patient interview and/or the performance of standard tests for depression. The timely diagnosis and treatment of depression in persons with AMI can be expected to favorably affect prognosis, morbidity, and mortality in this population.

CONCLUSIONS Depression is often undiagnosed and untreated in patients with AMI. The results of a number of studies indicate that depression represents both a risk factor for AMI and a predictor for a poorer outcome following it. Depression plays an important role in affecting blood coagulation, endothelial activity, heart rate variability, patient compliance with medication, adherence to a healthy diet, and smoking cessation, all of which influence the morbidity and mortality associated with AMI.

The DI and similar evaluation tools have been shown to be of value in assessing patients for depression and should be more regularly used in clinical practice.

When associated with AMI, depression increases hospital LOS, readmission rates, and consequently the cost of medical care. The SSRIs exert a positive effect in patients and also appear to reverse enhanced platelet activity seen in depressed patients with AMI. The available evidence suggests that there is significant need and merit to treat depression associated with AMI.

Key Recommendations

Patients having a Myocardial Infarction should be screened for depression using a standardized depression symptom checklist at regular intervals during the Post-Myocardial Infarction (Post-MI) period, including during hospitalization.

Post-MI patients with a diagnosis of depression should be treated to improve their depression symptoms, with systems in place to ensure regular follow-up and monitoring of their treatment response and adherence to treatment.

Selective Serotonin Reuptake Inhibitors (SSRIs) are preferred to tricyclic antidepressants for treatment of depression in post-MI patients.

Psychotherapy may be beneficial for treatment of depression in post-MI patients. The existing evidence does not establish what form of psychotherapy is preferred.

Kulsum International Hospital (KIH) is a tertiary cardiac center that caters wide range of cardiac patients from farflung areas of the country. In keeping with international standards, we have a system in place where regular psychiatric assessment, treatment, counseling sessions, cognitive behavioral therapy (CBT), psycho education and follow ups are organized for patient safety and to improve their outcomes.

Courtesy to Dr. Mamoon Qadir (Cardiologist - KIH) for his help and initiative in highlighting this neglected aspect of critically sick cardiac patients.

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TRUE BRACHIAL ARTERY ANEURYSM IN A CHILD AGED 2 YEARS

Dr. Aamir Ghazanfar

(General, Laparoscopic and Vascular Surgeon, KIH and KRL Hospital)

SUMMARY

Congenital brachial artery true aneurysms are exceedingly rare. Most are pseudoaneurysms secondary to trauma or infection. We report a child aged 2 years who was presented with painless, pulsatile swelling on the medial aspect of the right arm, 4cm above the elbow joint, that had been present since birth. Spiral CT Angiography showed a Fusiform Aneurysm of the distal right brachial artery with a peripheral crescent-shaped thrombus. Distal arteries were normally opacified. There was no evidence of abnormal dilation or stenosis in any other artery. The aneurysm was surgically resected, with vascularisation reestablished using a reversed great saphenous vein graft. His postoperative course was uneventful. Early surgery should be performed for moderately sized to large aneurysms that recently increased in size, exhibited luminal thrombus formation or caused neurovascular distal limb compromise. Early surgery could prevent complications such as a ruptured aneurysm, thromboembolism or limb ischaemia or loss.

BACKGROUND

An aneurysm is defined as an abnormal, localised, widened/dilated, ballooning of the wall of an artery, vein or heart, leading to its weakness.

Among peripheral artery aneurysms, those of the lower extremity (eg, femoral, popliteal) are common. Upper extremity aneurysms are rare.

Most of the brachial artery aneurysms are pseudoaneurysms that are commonly caused by arterial damage due to a diagnostic or therapeutic intervention. It may also be due, however, to direct trauma leading to wall disruption and damage.

Congenital brachial artery aneurysms are rare. Only 5% of peripheral artery aneurysms are located in the upper extremity. Most congenital brachial artery aneurysms are associated with other systemic diseases.

Only 14 cases of brachial artery aneurysms in patients less than 12 years of age have been reported since 1950. In nine of these cases, a brachial artery aneurysm was associated with other aneurysms. Only five cases had a true brachial artery aneurysm not associated with other central or peripheral artery aneurysms or any systemic disease.

CASE PRESENTATION

A boy aged 2 years presented with a painless, pulsatile swelling on the medial aspect of his right arm 4cm above the elbow joint that had been there since birth. The swelling recently increased, almost doubling, during the last 2 months. There were no symptoms of pain, numbness, bruising, skin discolouration or wasting of the right forearm or hand. There was no past history of birth trauma, fracture, therapeutic or diagnostic venipuncture, or intrarterial cannulation. He had not undergone any surgery or hospitalisation since birth. There was no history of fever, dyspnoea, loss of consciousness, black-outs, headache, fits, joint dislocation, joint or skin laxity, or other swelling on the body.

Most congenital brachial artery aneurysms are associated with central or peripheral arterial aneurysms due to a systemic disease (e.g, Ehlers-Danlos Syndrome, Marfan Syndrome, Kawasaki Disease, Giant Cell Arteritis, Polyarteritis nodosa). The whole course of his gestation was uneventful. There was no family history of aneurysm congenital vascular disease or connective tissue disorder.

On examination, his height and weight were appropriate for his age. There was no pallor or lymphadenopathy. Physical

examination of the swelling showed a 40x30mm fusiform, pulsatile, expansile swelling on the medial aspect of the right arm 4cm above the medial epicondyle of the right humerus. The swelling was nontender, nonreducible and transversely mobile. There was no scarring, skin discoloration or prominent veins visible over the swelling. Right radial, ulnar and all other pulses were palpable with normal volume. Radial, and median nerve sensory and motor functions were intact. No bruit was audible on auscultation. Heart rate was 100 bpm with no added heart sounds or murmurs. The remainder of the systemic examination was unremarkable.

INVESTIGATIONS

The complete blood picture showed a haemoglobin concentration of 9.8 g/dL with normal platelet and white cell counts. Erythrocyte Sedimentation Rate, Coagulation Profile, C Reactive Protein, Renal Function Tests, Liver Function Tests, Lipid Profile and Proteins C and S were within the normal range. The anti-dsDNA Immunoglobulin G test was negative.

Abdominal Ultrasonography and Echocardiography showed no abnormalities. Arterial Doppler images of the right upper limb showed a right brachial artery aneurysm with turbulent flow through the aneurysm and normal arterial flow of the right radial and ulnar arteries. CT Angiography (CTA) showed fusiform dilation of the distal right brachial artery 4cm proximal to the right elbow joint (figure 1). The aneurysm was thick walled with a luminal peripheral crescent shaped thrombus. The right axillary, radial, ulnar and distal palmar arteries and arteries of the left upper limb, lower limbs, chest, abdomen, neck and brain were normally opacified with no evidence of abnormal dilation or stenosis. Preoperative lower limb venous mapping was performed to select a great saphenous vein graft.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for brachial artery aneurysm includes pseudoaneurysm, haematoma and arteriovenous malformation.

TREATMENT

As brachial artery aneurysms are rare, with few cases reported to date, no definitive treatment or surgical procedure has been established.⁽⁷⁾ Although endovascular stenting to repair popliteal artery aneurysms has been reported, no such technique has been suggested for repairing a true brachial artery aneurysm.⁽⁸⁾ Hence, the best therapeutic option for a brachial artery aneurysm is surgical repair, thereby preventing upper extremity ischaemia.

We planned early surgery because of the recent increase in the size of the aneurysm and the presence of the thrombus seen on CTA. It was designed to prevent thromboembolic complications, rupture of the aneurysm and/or limb loss.

Under general anaesthesia, 7cm vertical skin incision was made over the right brachial artery aneurysm on the medial aspect of the right arm. A sacular aneurysm was identified arising from the distal end of the right brachial artery (figure 2). Vascular clamps were applied to the right brachial artery proximal and distal to the aneurysm. The aneurysm was resected, 5cm reversed great saphenous vein graft interposed and an end-to-end anastomosis created using the proximal and distal ends of the right brachial artery. It was held in place with polypropylene 6/0 sutures (figure 3). Histopathological examination confirmed that it was a true aneurysm. All three vessel wall layers were intact with an organised thrombus in the vessel lumen.

OUTCOME AND FOLLOW-UP

The patient remained hospitalised for 5 days and was discharged on 3rd post-operative day after surgery. He was prescribed 75mg aspirin orally once per day for 6 months. CTA performed 1 week after surgery showed a patent vascular graft. The patient was advised to appear for follow-up at 10 days post operatively and then monthly for 3 months, quarterly for a year and then yearly. CTA/M was advised at 6th month and then annually.

There were no thromboembolic complications, vascular graft stenosis, right forearm or hand ischaemia or aneurysm recurrence development at any other site during 6 months of followup. Distal neurovascular status of the right upper limb was intact throughout the postoperative period.

DISCUSSION

Aneurysms may be true or false. If all three layers of the vessel wall are involved, it is said to be a true aneurysm. In false aneurysms, only a single layer of fibrous tissue is involved. An aneurysm may also be congenital or acquired (mycotic, syphilitic, traumatic) and is classified as central or peripheral according to its site.⁽¹⁾

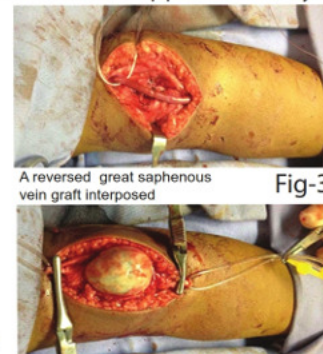
Most brachial artery aneurysms are pseudo aneurysms that are commonly caused by arterial damage due to a diagnostic or therapeutic intervention. They may also be caused by direct trauma leading to wall disruption and damage.

Congenital brachial artery aneurysms are rare. Only 5% of peripheral artery aneurysms are in the upper extremity.



CT angiography showing balloon like dilation distal end of the right brachial artery

Fig-1



Operative view of fusiform aneurysm arising from the right brachial artery.

Fig-2

Most congenital brachial artery aneurysms are associated with other systemic diseases (e.g. Ehlers Danlos Syndrome, Marfan Syndrome, Kawasaki Disease, Giant Cell Arteritis, Polyarteritis nodosa).

Since 1950, only 14 cases of brachial artery aneurysms in children less than 12 years of age have been reported. In nine of these cases, the brachial artery aneurysm was associated with other aneurysms. There were only five cases of an isolated true brachial artery aneurysm not associated with other central or peripheral artery aneurysms or any systemic disease.⁽⁵⁾

Patients with brachial artery aneurysms may be asymptomatic or present with a pulsatile mass. Other findings appear when thromboembolic complications occur.⁽⁹⁾ Complications include profuse bleeding, vascular collapse and/or thrombus formation leading to emboli and the risk of limb loss.⁽¹⁰⁾

Since brachial artery aneurysm is rare and few cases have been reported up to now, there is no established definitive treatment or surgical procedure.⁽⁷⁾

Although endovascular stenting has been reported for repair of popliteal artery aneurysms, no such technique to repair true brachial artery aneurysms has appeared in the literature.⁽⁸⁾

Therefore, to prevent upper extremity ischaemic sequelae, the best therapeutic option for brachial artery aneurysm is surgery.⁽¹⁾

Urgent surgery is not required if a brachial artery aneurysm is small and asymptomatic. Early surgical intervention is required, however, if the aneurysm is moderately sized or large, has recently increased in size, exhibits thrombus within the aneurysm or there is neurovascular compromise of the distal limb. If the aneurysm is small, surgical treatment involves aneurysm resection with end-to-end anastomosis. For large aneurysms, the definitive treatment is aneurysm resection and revascularisation of

the brachial artery with a reversed saphenous vein graft interposed, anastomosed with the proximal and distal ends of the brachial artery.^(5, 6)

In the present case, the brachial artery aneurysm size had recently increased and CTA showed thrombus formation. Early surgical reconstruction was planned to avoid aneurysm rupture and thromboembolic complications.

Children who undergo surgical resection of an aneurysm and arterial reconstruction should take oral aspirin for 6 months postoperatively to reduce the risk of occlusion at the sites of anastomosis.^(5, 6)

Long-term follow-up and screening studies are recommended in children with brachial artery aneurysm because of the risk of recurrence and the development of peripheral or central arterial aneurysms at other sites of the body, which can form even several years.

Diligent workup is needed for accurate diagnosis of an aneurysm. Early surgical intervention is required for moderate to large aneurysms, aneurysms whose size recently increased, intra-aneurysm thrombus formation or if there is neurovascular compromise of the distal limb. In this case, meticulous workup confirmed the presence of a congenital, idiopathic, right brachial artery true aneurysm. It was not associated with central or peripheral arterial aneurysms at other sites of the body or any systemic disease.

Amir Ghazanfar is the main surgeon who operated the case. He also contributed greatly in the drafting and revision of this document.

This article was originally published in BMJ Journal (2016).

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New Consultants

Dr. Mohammad Akmal

Dr. Mohammed Akmal, a globally renowned Spinal Surgeon joins Kulsum International Hospital. He is working as a Consultant Orthopedics and Spinal Surgery at London Spine Unit and Imperial College Healthcare NHS Trust, United Kingdom. He frequently visits Kulsum International Hospital and conducts spine clinic for the patients with back pain and spinal deformities, defects and trauma.

Dr. Asjad Hameed

Dr. Asjad Hameed is region's renowned Endocrinologist and Diabetologist having decades of experience in UK, Ireland and UAE. He is also a leading philanthropist and Chairman of The Diabetes Center, a non-profit organization which runs and manages all healthcare facilities related to diabetes.

Dr. Adil Jadoon

Dr. Adil Jadoon is a Nephrologist with a professional experience of USA. He is a graduate of Rawalpindi Medical College and completed his training from United Kingdom. Dr. Adil completed his Nephrology fellowship from University of Michigan and joined there as a faculty member. His primary areas of interest are hemodialysis, CRRT and glomerular diseases.



Visit of KIH Management to Cleveland Clinic, Abu Dhabi

Dr. Iqbal Saifullah Khan (Advisor, Saif Healthcare Limited) and Dr. Muhammad Saleem Khan (CEO, Saif Healthcare Limited) visited Cleveland Clinic Abu Dhabi. Aim of the visit was to learn the health care practices adopted by Cleveland Clinic.

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Kulsum International Hospital has launched a patient friendly website and mobile application. Website provides more convenience, information and a service of appointment. Mobile application is designed for Android and iOS users. Patients can get appointments and access the diagnostic reports through application. Suggestions or improvements can be given at

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After



Before



Department of Spine Surgery