Insitu cardiac thrombosis: six cases with review of literature

¹Hamid Abdulwahab, ²Abdulamer Abdulbari

ABSTRACT

Background: Cardiac thrombus can occur whenever a local pathology favoring stasis of blood within cardiac chambers, however despite extensive clinical studies there is no general agreement as to the best management modality.

Method: We study the outcome of six cases of insitu cardiac thrombus by different treatment methods as surgery, low molecular heparin, or maintained on long period of warfarin

Result: The best result achieved with surgery in cases of cardiac thrombosis with adequate ventricular function, although in the setting of comorbidity, less invasive therapies as with long term treatment with warfarin or heparin therapies is an alternative safe therapeutic approach.

Conclusion: Treatment has to be individualized whether surgery or medical therapy, since many factors contribute to the outcome like ventricular function, treatable or untreatable underlying cause.

Key word: Intra Cardiac thrombus

الخثرة القلبية الموضعية: ستة حالات نادرة مع مراجعه المقالات المنشورة

الخلفية: الخثرة القلبية نادرة الحدوث عادة وتنتج عن تباطي الدورة الدموية داخل حجيرات القلب ورغم الدراسات السريرية المكثفة، لايوجد اتفاق عام حول العلاج الامثل ويكون العلاج عادة تابعا لحالة كل مريض على حدة.

الهدف من الدراسة: دراسة النتائج المترتبة على عدة طرق علاجية للخثرة القلبية.

الطرق: بحث دراسة ستة حالات من تخثر القلب الموضعي في مركز امراض القلب في البصرة خلال كانون ٢١٠٦ الى ايار ٢١٠٤، تم اتباع طرق علاجية مختلفة كالجراحة وعقاقير مسيلة للدم كالهيبارين والوارفرين.

النتائج: احسن النتائج كانت بالطرق الجراحية في حالة الخثرة القلبية الموضعية داخل القلب عندما يكون عمل البطين الايسر طبيعي، وبرغم ذلك عند وجود اعتلالات صحية اخرى فان استخدام طرق علاجية اقل خطورة كالمسيلات الدموية لمدة طويلة نافعا.

الاستنتاج: العلاج يجب ان يكون محدد حسب حاله المريض سواء كان جراحيا او طبيا حسب عدة عوامل تودي الى اختيار طريقة لعلاج مريض على حدة من ضمنها وظيفة البطين الايسر واذا كان سبب التخثر هو مؤقت او دائمي.

INTRODUCTION

n situ formation of thrombi within the cardiac cavities carries a substantial risk of morbidity and mortality due to the risk of embolization to vital organs.^[1] This typically occurs in patients with underlying cardiac disorders associated with low flow state favoring regional stagnation of blood and subsequent thrombosis. The appropriate treatment such thrombi remains of controversial.^[2] Cardiac thrombus may be a complication of primary cardiac, hematological or rheumatological disease Cardiac-source of thrombo-embolism can be predicted on the basis of echocardiographic findings such as the

presence of spontaneous echocontrast, large hypokinetic cardiac chambers, and mitral stenosis. Other factors may play a role such as history of thromboemboli, atrial fibrillation and increased coagulation markers. Most thrombotic lesions develop spontaneously; others associated with trauma, surgery or pregnancy. Acquired protein c and s deficiencies have been reported in patients with deep venous embolism.^[1-3] thrombosis & pulmonary Antiphospholipid syndrome (APS) important systemic autoimmune disease that associated with a hypercoagulable state. Cardiac manifestations of APS can cause intracardiac

¹ FICMS (Med), FICMS (CARD). Head of CCU Department, Basrah Teaching Hospital, hamidcardio20132gmail.com

² FICMS, CABM (Med), FICMS (CARD), Assistant Professor, Department of Medicine, College of Medicine, hamidcardiozol3@gmail.com

thrombi & pulmonary hypertension. Surgical removal of emboli has been validated but cannot be proposed to all patients since it is a high-risk procedure. Fibrinolysis is generally efficient but exposes the patient to risk of embolization of the intracardiac thrombus, with occasionally deleterious outcome.^[3,4] About a third of patients with dilated cardio-myopathies have left ventricular thrombi, definitive treatment of these thrombi has yet to be established.

REVIEW OF LITERATURE

Thrombus is differentiated from a tumor, because the former is associated with abnormal regional wall motion, contrast echocardiography can be helpful in differentiating an apical mass from a thrombus. Thrombus is visualized as dark structure on contrast echocardiography, because it is not vascular. [5] The concept of dealing with an in situ cardiac thromboses still controversial, Hassen et al review two cases of in situ cardiac thrombosis successfully treated with streptokinase infusion, both cases were having chronic renal impairment as well. streptokinase in low dose infusion also used by Ertugrul et al. for a case of multiple cardiac thromboses involving the right atrium, right ventricle in a 56 year- old man with protein c defiancy, who presented with chest pain and shortness of breath, in whom surgery was not satisfied due to multiple thrombi proved by transesophageal echo these thrombi were measured 1*1*5 cm, 1*1*4.5cm and 1*1*3.5 cm, all disappear after prolonged low dose streptokinase with the disappearance of dyspnoe and chest pain. [2] Other modality of treatment include low dose of long term anticoagulant like warfarin as reported by Sait et al. who report a case of right ventricular thrombus in a 33 year old man with Behcet syndrome presented with shortness of breath, and after one month of warfarin and immunosuppressive treatment the disappears. [6] thrombus Other causes hypercoagulable state like protein c and s deficiency, treatment of symptomatic patients is usually with initial heparin therapy followed by lifelong oral anticoagulant therapy. [2] Gurgun et al also report a case of Behcet disease with left ventricular thrombus who were adequately managed by anticoagulant and immunesuppressive agent.^[7] Surgical treatment might be considered in Behçet's disease complicated by cardiac thrombi, when a thrombus recurs despite medical treatment. as reported by Kirali K who successfully treat young man with right ventricular thrombus by surgical excision.^[8] Besides hypercoagulable causes, end stage DCM have a percentage of 11-44% of cardiac thrombus, Kaghasadeghi reported a case of DCM with big thrombus that reach to the aortic cusp was successfully treated by emergency surgical removal. [4] Also Nilly et al reported 4 cases of post MI left ventricle thrombosis, two of them removed during bypass surgery and the third after 6 months with good result. [9] Heik et al reported that use of low molecular weight heparin for 14 days in 59 year-old man with nonischemic cardiomyopathy & EF=10% with disappearance of the thrombus. [10] Keren et reported a series of 198 patients with acute myocardial infarction a predischarge thrombus was found in (31%). Anterior infarction, reduced ejection fraction below 35% and apical dyskinesia or aneurysm were significantly related to the thrombus formation during hospitalization, echocardio-graphic follow-up for at least 6 months revealed that the thrombus disappeared in (48%) of patient, it resolved during further follow-up. Thus a predischarge echocardiography can identify patients who are likely to have thrombus formation and assess the risk of embolization.^[11] Conclusion of literature: It appears that most of the cases reported either they use streptokinase (because bleeding is more with other thrombolytic), long term anticoagulant or surgery, although the cases that reported didn't show increase risk of embolization with the use of streptokinase or urokinase especially if it is used in low dose with prolonged infusion to avoid fragmentation of thrombi. Oral anticoagulation has had variable success, with resolution rates ranging

from 13 to 59 percent. Thrombolysis with streptokinase, urokinase, and tissue plasminogen activator has been reported, but the risks of hemorrhagic or embolic complications may be unacceptably high.^[5-7] High-dose intravenous heparin effectively treats thrombi that are mobile or that protrude into the left ventricular cavity with a Complete resolution of 83 percent of thrombi has been documented after a mean duration of 14 days of treatment, but hospitalization and monitoring of the partial-thromboplastin time are required. [8] Surgical thrombectomy is also advocated but

Case No. 1

A forty-year old multiparous female not known to had previous chronic illness presented to outpatient clinic because of progressive shortness of breath for 3 weeks after delivery, past history was unremarkable apart from two first trimester abortions, with history of contraceptive pills for three months before the last pregnancy, she was conscious, dyspnoec, pale, although not cyanosed with mild pitting leg edema, BP 130/90 mmHg, PR 102 b/min regular, no organomegaly, S3, S4 and short murmur. **ECG** diastolic showed sinus tachycardia with biphasic p wave and flat T wave in lateral leads, Echocardiography study showed dilated both ventricle with global

generally patients with dilated cardiomyopathies are at an increased risk for perioperative morbidity and mortality. So it has been validated but cannot be proposed to all patients since it is a high-risk intervention. During treatment follow up by echocardiography is the most helpful diagnostic test initially but for complete screening of cardiac thrombi transesophageal echocardiography TEE is the gold standard for identifying thrombi. It may be use for complete evaluation of the cardiac mass and to obtain an accurate diagnosis. [5,12]

hypokinesia, right ventricular systolic pressure estimated to be 36 mmHg from tricuspid regurgitation. Ejection fraction =38%, there is a big highly mobile free-floating left atrial thrombus. The patient diagnosed as a case of peripartum dilated cardiomyopathy complicated by intracardiac thrombus, she was given 5000 units heparin and admitted to intensive coronary care unit with the idea of possible thrombolytic therapy, after few minutes of arrival she developed loss of consciousness decerebrate position with a left sided gaze palsy, and suddenly collapsed with VF, resuscitation fail to help the patient and unfortunately the patient died. (Fig 1&2)



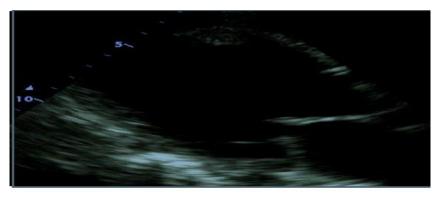
Fig. 1 Five chamber view show LA thrombus 3.2x2.8 cm in diameter



Fig. 2 Five chamber view with colour Doppler study showed large LA thrombus

A 35 year-old male patient, with no history of diabetes, hypertension, had family history of dilated cardiomyopathy, known to have dilated cardiomyopathy for the last three year, he was compensated, presented for follow up assessment of his recent shortness of breath. Clinically the patient was dyspnoec, with elevated JVP, BP of 120/80mmHg, PR of 90 b/min regular. Examination showed Palpable liver, mild ascites, bilateral basal crackles and

S3 gallop. ECG showed LBBB, echo study revealed multiple mural thrombosis over areas of hyperkinesia's therefore after 3 weeks of enoxaparin, follow up echo study (Fig-3) shows no more left ventricular thrombus, his ejection fraction improved from 35% to 45%. No more thrombus during follow for 6 months after discharge. No leg edema, with no other congestive symptoms .the patient show a good response to ant failure and anticoagulant drugs.



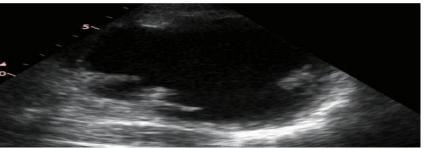


Fig 3. Multiple mural masses seen in two chamber long and short axis views

A sixty-five year old diabetic man with a history of anterior myocardial infarction one year ago, previously smoker, presented with exertional shortness of breath, his ECG showed (Q wave) in V1-4, mild cardiomegaly on chest x-ray. Echo study showed apical thrombus over area of aneurysm formation, ejection fraction was 35%, the patient was kept on warfarin to prevent

propagation of thrombus, his INR kept above 2, echo after two months showed that the thrombus shrinkage although not disappear additional visit after 12 months. The patient was well, with good functional capacity although the apical thrombus well organized, no showering of emboli occurs (Fig-4).



Fig 4. Apical thrombus seen in the apical aneurysm on apical two chamber view

Case No. 4

A sixty-five year old diabetic man presented with shortness of breath, gradually increasing in severity, he had a history of acute anterior myocardial infarction 5 years ago and post MI angina, he underwent coronary angiography in Baghdad, but failed to establish coronary perfusion. Now he had basal crackles, S4, S3 gallop, bilateral leg edema with raised JVP. His ECG revealed Q wave in V1-V6. Echo four-D shows organized thrombus in apical aneurysm, ejection fraction approximately 30%, he was

kept on full antifailure therapy including diuretics and Angiotensine receptor blockers, with warfarin 5 mg then 2.5 mg to keep INR about 1.5-2 after five days cover of enoxaparin, the patient reevaluated after one and five months and was better regarding his shortness of breath and functional capacity, no history of embolic phenomenon, reecho after 4 months showed well organized thrombus, the patient still on regular follow up, no events had occurred even after two years.

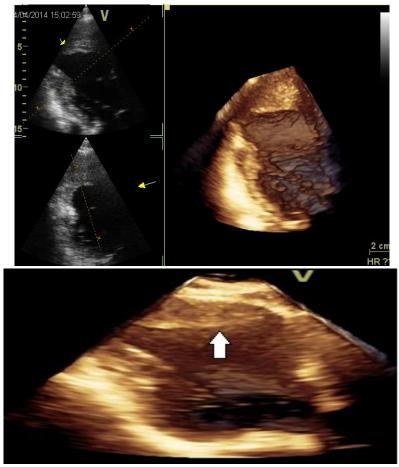


Fig 5. Four- D echo study shows thrombus in area of apical aneurysm arrow



Fig 6. Two dimension echo study of the apical thrombus

A thirty five year old man underwent metallic mitral valve replacement for rheumatic severe mitral valve stenosis, he was kept on warfarin with INR above 2.5, although he stopped warfarin after 6 months by himself, after 12 months he presented with shortness of breath, occasional syncopal attack, on examination he looked thin, dyspnoec, BP 100/60 mmHg, PR=90 b/min, his ECG unremarkable apart from

sinus tachycardia, ECHO study revealed a floating big organized thrombus ball about 1.8 cm by 1.9 cm in the left atrium, the thrombus was not attached to the metallic mitral valve, the patient was kept on heparin and was referred for surgical removal which is done after one week and was uneventful surgery, the patient was resumed to take warfarin postoperatively.



Fig 7. Modified five chamber view shows a thrombus ball in the left atrium not attached to metallic mitral valve.

A forty year old diabetic man previously smoker, with positive family history of ischemic heart disease presented for the first time to coronary care unit because of acute coronary syndrome, anterior myocardial infarction outside the golden period of thrombolytic therapy, his ECG showed Q wave in (v4-v6), his serum troponin was elevated, chest

examination shows fine basal crackles BP=120/70 mmHg, echo study shows apical wall hypokinesia, the patient was kept in the CCU for five days on full antischemic, insulin therapy and enoxaparin, reecho after two weeks, shows resolution of the apical thrombus although the a apical area less hypokinetic, no evidence of embolic phenomenon.

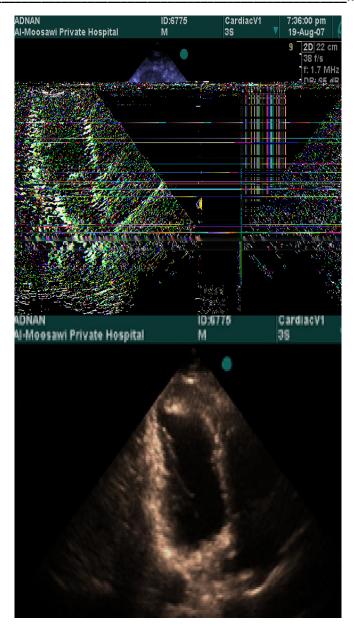


Fig 8. Shows the apical thrombus over the area of akinetic segment and it's follow up after 2 weeks

DISCUSSION

We reviewed six uncommon cases of intracardiac thrombi with different treatment methods. The first case was peripartum dilated cardio-myopathy (DCM) during pregnancy with a big thrombus in the left atrium as shown in (Fig-1,2), possibly no adequate antenatal care, despite that there is also multiple risk factors for cardiac thrombosis, besides DCM the patient

clinically had evidence of multiple abortions and this rise the possibility of antiphospholipid antibody syndrome there was no adequate time to manage such a case soon after admission to hospital she deteriorates rapidly, the patient was given heparin although no response and the patient unfortunately passed. This case raised the importance of antenatal care especially in

those pregnant women with cardiac comorbidities for early diagnosis & treatment of any complications. The second case was DCM although well compensated and with a good functional capacity, he showed decompensation for a short period of time during that period he developed a cardiac thrombus as shown in (Fig-3) and was kept on enoxaparin for four weeks with serial echo follow up after few weeks he showed disappearance of thrombus with improvement of ejection fraction. Third and fourth case had a structural heart disease (aneurysm) and therefore unlikely to be a transient or treatable condition in the short term, so they were given anticoagulant for a long term to prevent propagation & recurrence of thrombus which is shown in (Fig-4, 5, 6). The fifth case the patient had a big thrombus, although the patient was fortunate because the metallic valve prevent showering the thrombus ball as shown in (Fig-7), the patient underwent surgical evacuation and was a successful surgery without complications especially the patient had no co-morbidities that increase the risk from surgery. In sixth case the thrombus was over an area temporary akinetic and spontaneous resolution with enoxaparin after two weeks and then discontinued after that as shown in (Fig-8).

Table 1. Summary of the six cases and their characteristic features

Case no.	Age In years	Sex	DX	Treatment	Result
1	40	F	DCM	Heparin	Failure
2	35	M	DCM	Heparin	Success
3	60	M	MI	Warfarin	Shrink
4	65	M	Aneurysm	Warfarin	Shrink
5	35	M	Prosthetic	Surgery	Success
6	40	M	MI	Heparin	Success

If the success rate defined as complete disappearance of the thrombus then surgery is more effective as shown in the figure below

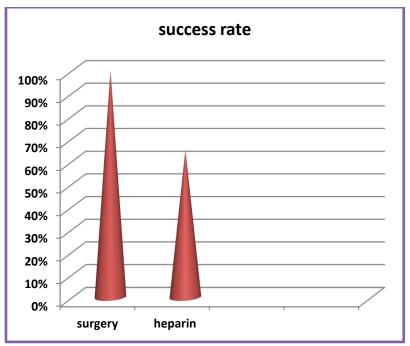


Fig 9. The difference in outcome between surgery and heparin treatment

RECOMMENDATION

- Surgery is the best treatment option in the presence of big thrombus floating in cardiac chambers, although should be weighed against the increased mortality during perioperative period as cases of dilated cardiomyopathy.
- In cases of advance DCM with expected long period of decompensation and high risk surgery, using warfarin result in shrinkage of the thrombus with stabilization of medical condition.
- If the cause seems to be transient as for examples post ischemic regional wall hypokinesia then using prolonged enoxaparin therapy is quite effective in potentially reversible causes.

REFERENCES

- 1. Walid Hassan, Fayez ElShaer, Mohamed E.I.D. Successful lysis of intracardiac thrmbus. Journal of Thrombosis and Thrombolysis: Volume 18, Number 2, 145-149.
- 2. Ertugrul Ercan, Istemihan Tengiz, Cevad Sekuri, Fahri Sahin, Emil Aliyev, Mustafa Akin and

- Unal Ac kel. Cardiac thrombi in a patient with protein-C and S deficiencies: a case report Thrombosis Journal 2004; 2: 2.
- 3. Pescatore SL. Clinical management of protein C deficiency. Expert Opin Pharmacother 2001; 2:431-439.
- 4. Kaghasadeghi, Y Mahmoody, M Shafa. Boxer thrombus, A case report Iranian Cardiovas Research Journal 2009; 3:3.
- 5. Gottdiener JS, Gay JA, VanVoorhees L, DiBianco R, Fletcher RD. Frequency and embolic potential of left ventricular thrombus in dilated cardiomyopathy: assessment by 2-dimensional echo, Am J Cardiol 1983; 52:1281.
- 6. Sait M. Dogan, Alparslan Birdane, Cengiz Korkmaz, Necmi Ata, and Bilgin Timuralp. Right Ventricular Thrombus with Behçet's Syndrome a case report, Tex Heart Inst J. 2007; 34(3): 360-362.
- 7. Gurgun C, Sagcan A, Cinar CS. Right atrial and ventricular thrombi in Behcet's disease: a case report and review of literature. Blood Coagul Fibrinolysis 2000; 11: 107-110.
- 8. Kirali K, Civelek A, Dağlar B, Sişmanoğlu M, Akinci E, Berki T, Işik O, Yakut C. An uncommon complication of Behçet's disease, A

- case study, Thorac Cardiovasc. Surg. 1998 Apr; 46(2): 102-105.
- 9. Nili M, Deviri E, Jortner R, Strasberg B, Levy MJ. Surgical removal of a mobile, pedunculated left ventricular thrombus: report of 4 cases. Ann Thorac Surg 1988; 46: 396-400.
- 10. Heik SCW, Kuper W, Ham C, et al. Efficacy of high dose intravenous heparin for treatment of left ventricular thrombi with high embolic risk. J Am Coll Cardiol 1994; 24: 1305-1309.
- 11. Keren A, Goldberg S, Gottlieb S. Natural history of left ventricular thrombi: their appearance and resolution in the posthospitalization period of acute myocardial infarction. J Am Coll Cardiol 1990; 15: 790-800.
- 12. Hayashi H, Kumon K. Trans esophageal echo cardiography: Clinical application. Masui 1996; 45: 980-990.