

Intracardiac thrombus in children with dilated cardiomyopathy

Dilate kardiyomiyopatili çocuklarda kalp içi trombüs

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ABSTRACT

Objectives: The risk of fatal pulmonary and systemic thromboembolism is high in patients with dilated cardiomyopathy with cardiac thrombus. This study was planned to reveal the efficacy of antiaggregant therapy in patients with low left ventricular systolic ejection fraction (LVEF).

Study design: The present study retrospectively reviewed the files of 83 cases (42 males, 41 females) with dilated cardiomyopathy who were followed between June 2004 and December 2011.

Results: Intracardiac thrombus was detected in five (6%) cases; of these five patients, dilated cardiomyopathy was idiopathic in four and secondary to chronic renal failure in one. The cases were followed for a mean of 33.6±35.6 months (3 days-168 months). Mean LVEF on transthoracic echocardiography was found as 35.2±2.7% (32-38%) for the cases with intracardiac thrombus, whereas it was 34.7±11.0% (10-55%) for the cases without intracardiac thrombus. No statistically significant difference was found between the groups (p=0.910). Cases with LVEF ≤30% were routinely receiving acetylsalicylic acid at antiaggregant dose.

Conclusion: We think that prophylactic antithrombotic/antiaggregant therapy should be started at the time of diagnosis even in patients with LVEF >30%, as thrombus development was seen in cases with LVEF >30% without any antiaggregant therapy.

ÖZET

Amaç: Dilate kardiyomiyopatide kalp içinde trombüs gelişiminde hayatı tehdit edici pulmoner veya sistemik tromboemboli riski yüksektir. Bu çalışmada, sol ventrikül sistolik ejeksiyon fraksiyonu (SVEF) düşük olan olgularda antiagregan kullanımının etkinliği değerlendirildi.

Çalışma planı: Bu çalışmada Haziran 2004 ile Aralık 2011 tarihleri arasında takip ettiğimiz 83 dilate kardiyomiyopatili olgunun (42 erkek, 41 kadın) dosyaları geriye dönük olarak incelendi.

Bulgular: Beş olguda (%6) kalp içi trombüs (KİT) vardı. Beş olgunun dördünde idiyopatik dilate kardiyomiyopati, birinde ise kronik böbrek yetersizliğine sekonder dilate kardiyomiyopati vardı. Olgular ortalama 33.6±35.6 ay (3 gün-168 ay) boyunca izlendi. Transtorasik ekokardiyografide trombüslü olguların SVEF ortalama %35.2±2.7 (%32-38), KİT olmayan olguların ise ejeksiyon fraksiyonu ortalama %34.7±11.0 (%10-55) bulundu (p=0.910). İki grup arasında istatistiksel olarak anlamlı fark saptanmadı. Sol ventrikül ejeksiyon fraksiyonu ≤%30 olan olgular, rutin olarak antiagregan dozda asetilsalisilik asit kullanıyordu.

Sonuç: Kalp içi trombüs gelişen SVEF >%30'un olan ve antiagregan başlamadığımız olgularda trombüs geliştiği için SVEF >%30 olsa bile tanı anında profilaktik amaçlı rutin antitrombotik/antiagregan tedavisine başlanmasının yararlı olduğunu düşünüyoruz.

Dilated cardiomyopathy (DCMP) is the most common type of cardiomyopathy in children. It is a myocardial disorder characterized by left ventricular (LV) dysfunction and dilatation, and progresses to congestive heart failure.^[1] Intracardiac thrombus (ICT) in patients with pediatric DCMP is a serious

complication and potential source of important morbidity and mortality. ICT may result from various conditions including LV systolic dysfunction, low cardiac output, abnormal endocardial surface, dysrhythmia, and tendency towards thrombosis.^[2-6] Prompt anticoagulant therapy is mandatory for DCMP patients who

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have rapidly worsening ventricular function history for ICT or history for embolic infarction to any part of the body.^[7] The strongest indication for thrombolytic therapy includes either a life- or limb-threatening thrombotic event. Significant bleeding and thromboembolism are known complications of thrombolysis. Contraindications to thrombolytic therapy need to be considered, including active bleeding, an inability to maintain the platelet count $>75.000/\mu\text{L}$ or fibrinogen >100 mg/dl, a major surgery or site of hemorrhage within 7 to 10 days, seizures within 48 hours, central nervous system surgery/ischemia/trauma/hemorrhage within 30 days, preterm infant <32 weeks, or uncontrolled hypertension.^[8]

Although the present study was designed as a retrospective study, we intended to attract attention to the follow-up of DCMP patients, the approach to the anticoagulant/antiaggregant therapy, and the relation between the incidence of ICT and ventricular dysfunction and dilatation.

PATIENTS AND METHODS

The present study retrospectively reviewed the files of 83 cases with DCMP who were followed between June 2004 and December 2011. The diagnosis of DCMP was based on the definitions and classification of the cardiomyopathies by the American Heart Association in 2006, as ventricular chamber enlargement and systolic dysfunction with normal LV wall thickness determined by two-dimensional transthoracic echocardiography (TTE).^[9] Totally, five cases had ICT associated

with DCMP. LV systolic function was measured using M-mode TTE and the Simpson method.

Causes of DCMP (viral anal-

yses, screening for metabolic disease, carnitine level, endocrine-related causes, collagen vascular diseases, Kawasaki disease, abnormal origin of left coronary artery, atherosclerosis, structural heart diseases, renal diseases, dysrhythmia) were investigated in all groups. The thrombosis panel (homocysteine, antithrombin III, protein C and protein S levels, methylene tetrahydrofolate reductase, factor V Leiden, prothrombin 20210 gene mutation, factor levels, plasminogen levels), which was studied in the cases with ICT, was considered normal.

Statistical analyses were performed using PASW 17.0 (Statistical Package for the Social Sciences [SPSS], Chicago, IL). Data are described as frequencies, proportions, median with ranges, and means with SDs. For all tests, a p value ≤ 0.05 was considered statistically significant. Comparison of two groups was done using Mann-Whitney U-test.

RESULTS

Table 1 demonstrates the general characteristics of the patients. Totally 83 cases (42 males, 41 females) who

Abbreviations:

| | |
|-------|--|
| DCMP | Dilated cardiomyopathy |
| ICT | Intracardiac thrombus |
| LV | Left ventricular |
| LVEDD | Left ventricle end diastolic dimension |
| LVEF | Left ventricular ejection fraction |
| LVFS | Left ventricle fractional shortening |
| TEE | Transesophageal echocardiography |
| TTE | Transthoracic echocardiography |

Table 1. General characteristics of the cases with dilated cardiomyopathy

| | Overall cases (n=83) | | | Thrombus cases (n=5) | | | Non-thrombus cases (n=78) | | |
|---------------------------------|----------------------|-----------|-------------------------|----------------------|-------|------------------------|---------------------------|----------|-------------------------|
| | n | % | Mean \pm SD (M) | n | % | Mean \pm SD (M) | n | % | Mean \pm SD (M) |
| Sex (Male / Female) | 42/41 | 50.6/49.4 | | 3/2 | 60/40 | | 39/39 | 50/50 | |
| Median age (year) | 2.0 | 0.8-14.8 | | 2.0 | 1-12 | | 2.0 | 0.8-14.8 | |
| Idiopathic DCMP | 80 | 96.4 | | 4 | 75 | | 76 | 97.4 | |
| DCMP secondary to CRF | 2 | 2.4 | | 1 | 25 | | 1 | 1.3 | |
| DCMP secondary to dysrhythmia | 1 | 1.2 | | – | – | | 1 | 1.3 | |
| Receiving antiaggregant therapy | 29 | 35 | | 0 | (0) | | 29 | 35 | |
| Initial LVEF | | | 34.8 \pm 11 (10-55) | | | 35.2 \pm 2.7 (32-38) | | | 34.7 \pm 11 (10-55) |
| Initial LVEDD (cm) | | | 4.35 \pm 1 (2.2-6.27) | | | 5.0 \pm 0.7 (4-5.93) | | | 4.3 \pm 1 (2.20-6.27) |

CRF: Chronic renal failure; DCMP: Dilated cardiomyopathy; LVEF: Left ventricular ejection fraction; LVEDD: Left ventricle end diastolic dimension; SD: Standard deviation; M: Median.

Table 2. General characteristics of the cases with intracardiac thrombus

| Characteristics | Case 1 | Case 2 | Case 3 | Case 4 | Case 5 |
|---------------------------|-------------------|----------------------|----------------------|--------------------------------|----------------|
| Age (years) | 2 | 12 | 1 | 2 | 6 |
| Initial EF (%) | 33 | 35 | 32 | 38 | 38 |
| LV thrombus | Yes | No | Yes | Yes | Yes |
| RV thrombus | No | No | No | No | No |
| RA thrombus | No | Yes | No | No | Yes |
| LVEDD | 5.93 | 4.8 | 4 | 5.4 | 4.95 |
| Mitral insufficiency | Moderate | Mild | Mild | Mild | Mild |
| Cerebral thromboembolus | No | No | No | No | Yes |
| Additional cardiac defect | No | Secundum ASD | No | No | No |
| Pulmonary thromboembolism | Yes | No | No | No | Yes |
| Treatment | Heparin | Heparin- Warfarin | Heparin- Warfarin | Heparin- Warfarin | rt-PA, heparin |
| Length of hospital stay | 3 days | 17 days | 14 days | 14 days | 2 days |
| Outcome | Dead | Alive | Alive | Alive | Dead |
| Cause of death | Pulmonary infarct | | | Cerebral and pulmonary infarct | |

ASD: Atrial septal defect; EF: Ejection fraction; LV: Left ventricle; LVEDD: Left ventricle end diastolic dimension; RA: Right atrium; RV: Right ventricle, rtPA: Recombinant tissue plasminogen activator.

were diagnosed with DCMP were followed. The median age of the patients was 2.0 years (1 month-14.8 years). The cases were followed for a mean of 33.6 ± 35.6 months (3 days-168 months). Thrombus was determined in four of the cases during follow-up; one of the patients was referred from another medical center with the diagnosis of cardiac thrombus. With respect to the etiology of DCMP, 80 (96.4%) cases had idiopathic DCMP, two (2.4%) had chronic renal failure, and one (1.2%) had persistent junctional reciprocating tachycardia. Mitral insufficiency was mild in 31 (37.3%), moderate in 22 (26.3%), severe in 12 (14.5%), and ignorable in 5 (6%) cases. ICT was diagnosed in 5 (6%) patients via TTE. Twenty-nine (34.9%) of the patients treated with acetylsalicylic acid had left ventricular ejection fraction (LVEF) $\leq 30\%$ and 54 (65.1%) of them had LVEF $>30\%$. Antiaggregant treatment was only started in patients with LVEF $<30\%$. The mean follow-up period was 16 ± 9.5 months (3 days-25 months) in the cases with ICT and 34.7 ± 36.4 months (1-168 months) in the other cases. Table 2 demonstrates the general characteristics of the cases with ICT. Diagnosis was made via TTE in 5 (6%) cases with ICT. Mean diameter left ventricle end diastolic dimension (LVEDD) was 5.0 ± 0.7 (4-5.93

cm) in DCMP cases with thrombus, whereas it was 4.3 ± 1 (2.2-6.27 cm) in those without thrombus. No statistically significant difference was found between the groups ($p=0.117$). Mean LVEF was $35.2\pm 2.7\%$ (32-38%) in DCMP cases with thrombus, whereas it was $34.7\pm 11\%$ (10-55%) in those without thrombus. No statistically significant difference was found



Figure 1. In Case 1, thrombus is seen in the apical region of the left ventricle.

with LV thrombus. The incidence of ICT in DCMP ranges from 4-16%, whereas it increases to 43-57% in pediatric autopsy reports.^[10-17] The risk for ICT and systemic thromboembolus increases in case of a LVEF <20%.^[13,14,18,19] Günthard et al.^[13] detected ICT in 17 (14%) of 130 cases with DCMP. They found the left ventricle fractional shortening (LVFS) to be significantly lower in the cases with ICT (mean LVFS 10±3%) as compared to those without ICT (mean LVFS 17±6%). Falk et al.^[14] detected thrombus in the LVs of 11 (44%) of 25 adults with non-ischemic DCMP. In comparison with the group without thrombus (LVFS 11-25%), they found the LVFS to be significantly low in these cases (LVFS ≤10%). McCrindle et al.^[6] also found LVEF to be significantly low in DCMP cases with ICT (mean LVEF 21±9%) as compared to the other group (mean LVEF 28±15%). However, in line with the present study, Choi et al.^[18] found no difference between the groups with and without thrombus in terms of LVEF.

Transthoracic echocardiogram (TTE) does not offer satisfactory views of the left atrial appendage. However, transesophageal echocardiography (TEE) is a reliable technique that gives a clear view of the left atrial appendage (LAA), with sensitivity and specificity of 100% and 99%, respectively. TEE was not suitable because most of our patients were infants.^[20,21]

Patients with dilated LV and low EF seem to be at high risk for LV thrombus formation. In our study, although LVEDD was larger in the patients with ICTs, no statistically significant difference was found between the groups with and without ICT. Furthermore, LAA thrombosis should be considered when designing the treatment strategy for patients with DCMP at sinus rhythm.^[20,22]

Whereas ICT is usually seen in the LVs of the cases with DCMP, it is mostly seen in the atria of the cases with pulmonary hypertension, Eisenmenger syndrome or atrial fibrillation. In case of severe systolic and diastolic dysfunction of the LV, thrombus is more likely to occur in the LV and left atrium due to stasis of blood flow, whereas it is more likely to occur in the right atrium in case of pulmonary hypertension.^[6,13,18,21-24] However, the incidence of thrombus formation is decreased in LV dysfunction, since mitral insufficiency prevents stasis.^[2,25-30] In the present study, mild mitral regurgitation was detected in the cases having ICT in the LV. The second case with ICT in

the right atrium had moderate mitral insufficiency, secundum atrial septal defect and moderate pulmonary hypertension. We think that pulmonary hypertension might have contributed to the presence of the ICT in the right atrium.

There are different treatment options in DCMP patients with LV thrombus, like anticoagulation, antiplatelet therapy and surgery. Although it is difficult to recommend open-heart surgery to patients with a sole indication of LV thrombus, Lee et al.^[31] stated in their study that systemic thromboembolism incidence is lower in patients treated with surgery when compared with other treatment options. It is known that anticoagulation therapy with warfarin reduces both the risk of ICT and subsequent thromboembolism. Guidelines recommend anticoagulation with warfarin for patients with ICT or cardioembolic stroke.^[32,33] Günthard et al.^[13] recommended prophylactic anticoagulant therapy in DCMP cases with ICT if LVFS is <20%. Falk et al.^[14] emphasized that the risk for ICT and thromboembolism was high in the cases not using anticoagulant and with low LVFS. Prophylactic antiplatelet, antiaggregant or anticoagulant therapy is recommended in the cases with LVEF <30%.^[6,13,14,19,23] However, the present DCMP cases developed ICT and systemic thromboembolus even though their LVEFs ranged from 32-38%. It was clear that the cases with LVEF <30% who were receiving prophylactic antiaggregant therapy had no ICT. Therefore, it would be beneficial to review the criteria to commence prophylactic antiaggregant/antithrombotic therapy.

While prophylactic anticoagulants have been used in adults with DCMP to prevent ICT and systemic thromboembolus,^[33] studies performed with children are limited. In recent years, thrombolytic agents such as rt-PA, streptokinase and urokinase have been used for ICT.^[15,34,35] Although antiplatelet and antiaggregant agents are effective in preventing ICT, it is known that they have no effect on thrombus resolution. Heparin or oral anticoagulant should be used in the event of ICT.^[13,36] Nevertheless, there are authors that recommend prophylactic anticoagulant use when LVFS is decreased <20%.^[6,13,23]

The limitations of our study are its design as a single institution retrospective study, the underestimation of small thrombi with echocardiogram, and the inability to perform TEE in our patients, as most of them were infants.

In conclusion, ICT development in the cases with DCMP despite having LVEF >30% led us to believe that the criteria to commence prophylactic therapy with antiplatelet/antiaggregant agents should be reviewed and that multicenter prospective studies on the prophylactic use of anticoagulants are needed.

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REFERENCES

- Towbin JA, Lowe AM, Colan SD, Sleeper LA, Orav EJ, Clunie S, et al. Incidence, causes, and outcomes of dilated cardiomyopathy in children. *JAMA* 2006;296:1867-76. [CrossRef](#)
- Kalaria VG, Passannante MR, Shah T, Modi K, Weisse AB. Effect of mitral regurgitation on left ventricular thrombus formation in dilated cardiomyopathy. *Am Heart J* 1998;135:215-20. [CrossRef](#)
- Kaplan SD, Chartash EK, Pizzarello RA, Furie RA. Cardiac manifestations of the antiphospholipid syndrome. *Am Heart J* 1992;124:1331-8. [CrossRef](#)
- Yamamoto K, Ikeda U, Furuhashi K, Irokawa M, Nakayama T, Shimada K. The coagulation system is activated in idiopathic cardiomyopathy. *J Am Coll Cardiol* 1995;25:1634-40.
- Yokota Y, Kawanishi H, Hayakawa M, Kumaki T, Takarada A, Nakanishi O, et al. Cardiac thrombus in dilated cardiomyopathy. Relationship between left ventricular pathophysiology and left ventricular thrombus. *Jpn Heart J* 1989;30:1-11.
- McCrinkle BW, Karamlou T, Wong H, Gangam N, Trivedi KR, Lee KJ, et al. Presentation, management and outcomes of thrombosis for children with cardiomyopathy. *Can J Cardiol* 2006;22:685-90. [CrossRef](#)
- Ahnert AM, Freudenberger RS. What do we know about anticoagulation in patients with heart failure? *Curr Opin Cardiol* 2008;23:228-32. [CrossRef](#)
- Giglia TM, Witmer C. Hematologic aspects of pediatric and adolescent heart disease: bleeding, clotting, and blood component abnormalities. In: Allen HD, Driscoll DJ, Shaddy RE, Feltes TF, editors. *Moss and Adams' heart disease in infants, children, and adolescents: including the fetus and young adult*. 8th ed. Volume 2. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2013. p. 1589-607.
- Maron BJ, Towbin JA, Thiene G, Antzelevitch C, Corrado D, Arnett D, et al. Contemporary definitions and classification of the cardiomyopathies: an American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. *Circulation* 2006;113:1807-16. [CrossRef](#)
- Waller BF, Grider L, Rohr TM, McLaughlin T, Taliercio CP, Fetters J. Intracardiac thrombi: frequency, location, etiology, and complications: a morphologic review-Part I. *Clin Cardiol* 1995;18:477-9. [CrossRef](#)
- Waller BF, Rohr TM, McLaughlin T, Grider L, Taliercio CP, Fetters J. Intracardiac thrombi: frequency, location, etiology, and complications: a morphologic review-Part II. *Clin Cardiol* 1995;18:530-4. [CrossRef](#)
- Chang YC, Wu JM, Wu MH, Wang JK, Lue HC. Left ventricular thrombi in children with dilated cardiomyopathy. *J Formos Med Assoc* 1995;94:469-73.
- Günthard J, Stocker F, Bolz D, Jäggi E, Ghisla R, Oberhänsli I, et al. Dilated cardiomyopathy and thrombo-embolism. *Eur J Pediatr* 1997;156:3-6. [CrossRef](#)
- Falk RH, Foster E, Coats MH. Ventricular thrombi and thromboembolism in dilated cardiomyopathy: a prospective follow-up study. *Am Heart J* 1992;123:136-42. [CrossRef](#)
- Herron SB, Lax D, Zamora R. Successful thrombolysis of acute left atrial thrombi in two pediatric patients following interventional cardiac catheterization. *J Invasive Cardiol* 2004;16:35-9.
- Reeder GS, Tajik AJ, Seward JB. Left ventricular mural thrombus: two-dimensional echocardiographic diagnosis. *Mayo Clin Proc* 1981;56:82-6.
- Rey C, Piot JD, Bourlon F, Cloez JL, Fermont L. 2-dimensional echocardiographic diagnosis of an intracardiac thrombus in children. [Article in French] *Arch Mal Coeur Vaiss* 1984;77:503-9. [Abstract]
- Choi SH, Jeong SI, Yang JH, Kang IS, Jun TG, Lee HJ, et al. A single-center experience with intracardiac thrombosis in children with dilated cardiomyopathy. *Pediatr Cardiol* 2010;31:264-9. [CrossRef](#)
- Gibelin P. Anticoagulant treatment and dilated cardiomyopathy. [Article in French] *Arch Mal Coeur Vaiss* 1995;88(4 Suppl):617-21. [Abstract]
- Manning WJ, Weintraub RM, Waksmonski CA, Haering JM, Rooney PS, Maslow AD, et al. Accuracy of transesophageal echocardiography for identifying left atrial thrombi. A prospective, intraoperative study. *Ann Intern Med* 1995;123:817-22. [CrossRef](#)
- Bakalli A, Kamberi L, Pllana E, Zahiti B, Dragusha G, Brovina A. The influence of left ventricular diameter on left atrial appendage size and thrombus formation in patients with dilated cardiomyopathy. *Türk Kardiyol Dern Ars* 2010;38:90-4.
- Bakalli A, Georgievska-Ismael L, Koçinaj D, Musliu N, Krasniqi A, Pllana E. Prevalence of left chamber cardiac thrombi in patients with dilated left ventricle at sinus rhythm: the role of transesophageal echocardiography. *J Clin Ultrasound* 2013;41:38-45. [CrossRef](#)
- John JB, Cron SG, Kung GC, Mott AR. Intracardiac thrombi in pediatric patients: presentation profiles and clinical outcomes. *Pediatr Cardiol* 2007;28:213-20. [CrossRef](#)
- Sethi KK, Kulshreshtha A, Gupta MP. Recurrent paradoxi-

- cal embolism in Eisenmenger syndrome. *Indian Heart J* 1984;36:121-3.
25. Virchow R. Phlogose und thrombose in geffasssystem. In: *Gesammelte Abhandlungen zur wissen schaftlichen medicin*. Frankfurt: Meidinger Sohn; 1856.
 26. Van Dantzig JM, Delemarre BJ, Bot H, Koster RW, Visser CA. Usefulness of mitral regurgitation in protecting against left ventricular thrombus after acute myocardial infarction. *Am J Cardiol* 1995;75:1270-2. [CrossRef](#)
 27. Blondheim DS, Jacobs LE, Kotler MN, Costacurta GA, Parry WR. Dilated cardiomyopathy with mitral regurgitation: decreased survival despite a low frequency of left ventricular thrombus. *Am Heart J* 1991;122:763-71. [CrossRef](#)
 28. Maze SS, Kotler MN, Parry WR. Flow characteristics in the dilated left ventricle with thrombus: qualitative and quantitative Doppler analysis. *J Am Coll Cardiol* 1989;13:873-81. [CrossRef](#)
 29. Delemarre BJ, Visser CA, Bot H, Dunning AJ. Prediction of apical thrombus formation in acute myocardial infarction based on left ventricular spatial flow pattern. *J Am Coll Cardiol* 1990;15:355-60. [CrossRef](#)
 30. Jacobs LE, Kotler MN, Parry WR. Flow patterns in dilated cardiomyopathy: a pulsed-wave and color flow Doppler study. *J Am Soc Echocardiogr* 1990;3:294-302.
 31. Lee JM, Park JJ, Jung HW, Cho YS, Oh IY, Yoon CH, et al. Left ventricular thrombus and subsequent thromboembolism, comparison of anticoagulation, surgical removal, and antiplatelet agents. *J Atheroscler Thromb* 2013;20:73-93. [CrossRef](#)
 32. Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction-executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction). *Circulation* 2004;110:588-636. [CrossRef](#)
 33. Ripley TL, Nutescu E. Anticoagulation in patients with heart failure and normal sinus rhythm. *Am J Health Syst Pharm* 2009;66:134-41. [CrossRef](#)
 34. Aspesberro F, Beghetti M, Oberhänsli I, Ozsahin H, Humbert J, Rimensberger PC. Local low-dose urokinase treatment of acquired intracardiac thrombi in preterm infants. *Eur J Pediatr* 1999;158:698-701. [CrossRef](#)
 35. Ferrari F, Vagnarelli F, Gargano G, Roversi MF, Biagioni O, Ranzi A, et al. Early intracardiac thrombosis in preterm infants and thrombolysis with recombinant tissue type plasminogen activator. *Arch Dis Child Fetal Neonatal Ed* 2001;85:F66-9.
 36. Kupferschmid C, Schmaltz AA, Tacke E, Apitz J, Lang D. Left ventricular thrombi in three children with dilated cardiomyopathy: diagnostic procedure and clinical course. *Pediatr Cardiol* 1984;5:65-9. [CrossRef](#)
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