REVIEW

Pitfalls in the diagnosis of left ventricular hypertrabeculation/non-compaction

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Left ventricular hypertrabeculation/non-compaction (LVHT) is a cardiac abnormality, characterised by >3 trabeculations apically to the papillary muscles and intertrabecular spaces. LVHT may occur with other cardiac abnormalities, heart failure, electrocardiographic abnormalities and neuromuscular disorders. This study gives an overview about (1) patients with LVHT in whom LVHT was initially overlooked and (2) cardiac conditions that may lead to falsely diagnosed LVHT. In 50 reported cases, LVHT has been overlooked and misdiagnosed as dilated (n = 20), hypertrophic (n = 14) or restrictive cardiomyopathy (n = 2), endocardial fibroelastosis (n = 5), endomyocardial fibrosis (n = 1), myocarditis (n = 3), thrombus (n = 2), localised left ventricular hypertrophy (n = 1), left ventricular mass (n = 1) or myocardial/pericardial disease (n = 1). In 14 patients, LVHT was diagnosed only by transoesophageal echocardiography (n = 1), computed tomography (n = 2) ventriculography (n = 2), magnetic resonance imaging (n = 3) or pathoanatomic findings (n = 6). Falsely diagnosed LVHT comprises false tendons, aberrant bands, thrombi, apical hypertrophic cardiomyopathy, fibroma, obliterative processes, intramyocardial haematoma, cardiac metastases and intramyocardial abscesses. Echocardiographers should be more aware of LVHT and consider its differential diagnoses.

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Normally, the left ventricle is less trabeculated than the right ventricle. Sometimes, however, the left ventricle is heavily trabeculated. This cardiac abnormality has been primarily described by echocardiography and is termed left ventricular hypertrabeculation/non-compaction (LVHT). LVHT has an unknown aetiology and is echocardiographically characterised by >3 left ventricular trabeculations apically to the papillary muscles, visible in one echocardiographic image plane, and intertrabecular spaces perfused from the ventricular cavity, as visualised on colour Doppler imaging.[1] Initially described as an echocardiographic abnormality, LVHT can also be visualised by ventriculography, cardiac computed tomography, cardiac magnetic resonance imaging and pathoanatomic findings.[2–6] Owing to the rapid development of cardiac imaging techniques, unfortunately no uniformly accepted golden standard diagnostic test presently exists for LVHT, and only few studies have systematically compared echocardiography or cardiac magnetic resonance studies with pathoanatomic findings.[6–9]

LVHT occurs with or without other cardiac abnormalities, and is often associated with heart failure, and echocardiographic abnormalities.[1 2 10] LVHT occurs in dilated, poorly contracting leftventricles, as well as in well-contracting, normal-sized ventricles.[11] The prognosis of patients with LVHT has initially been thought to be poor, but recent follow-up studies show that the prognosis is not as poor as has been assumed, and that the rate of thromboembolic events is small.[2 12 13] It is unknown whether the prognosis of patients with LVHT can be improved by early diagnosis.

LVHT has been described as being associated with mutations in the following genes: CSX, DMPK, dystrophin, G4.5, mitochondrial DNA and α-dystrobrevin.[14–19] Although some authors postulate LVHT to be a distinct cardiomyopathy, there are several indications that LVHT has a heterogeneous aetiology.[6 20] Often believed to be a congenital cardiac abnormality, LVHT may occur as an acquired disease.[21 22]

LVHT is commonly associated with neuromuscular disorders such as myoadenylate deaminase deficiency, mitochondriopathy, myotonic dystrophy, Becker's muscular dystrophy, Barth syndrome, Friedreich's ataxia and Pompe's disease[1 18 23] (Robert Weintraub, personal communication, 2003). Because of its rarity, LVHT has been often overlooked. As a consequence of the increasing awareness of LVHT, however, LVHT is also falsely diagnosed. This review aims to give an overview about (1) patients with LVHT in whom LVHT was initially overlooked and (2) cardiac conditions that may lead to falsely diagnosed LVHT.

METHODS

A Medline search was carried out for publications on LVHT until 2003; keywords were ''noncompaction'', ''non-compaction'', ''hypertrabeculation'' and ''persisting sinusoids''. The articles were further searched for reports of cases of LVHT.

OVERLOOKING LVHT

It has been repeatedly reported that LVHT was not diagnosed at the initial echocardiographic investigation but at one of the follow-up

Abbreviation: LVHT, left ventricular hypertrabeculation/non-compaction

investigations.[3 10 14 24–35] In most of these reports it is not mentioned after how many echocardiographic investigations LVHT was diagnosed. It is also not acknowledged how many different investigators were required to establish the diagnosis. Our own experience shows that between June 1995 and December 2001, LVHT was diagnosed at the first echocardiographic investigation in 26 of 55 (47%) patients.[1] In all but one of these cases, the investigations were carried out by one echocardiographer with a longstanding interest in LVHT (CS). LVHT was diagnosed at the second examination in 15 (27%) patients, at the third examination in 7 (13%), at the fourth examination in 3 (6%), at the fifth examination in 2 (4%) and in 1 (4%) patient each at the sixth and ninth examinations (fig 1). These experiences show that the most important prerequisite for the diagnosis of LVHT is the echocardiographer's awareness for this condition when examining the left ventricular cavity.[23] Data from another institution indicate that the prevalence of LVHT was 0.05% per year.[36] No data are available about the prevalence of LVHT among the normal population and from autopsy series.

Another point that makes diagnosing LVHT difficult is the quality of the echocardiographic machine, as the apical region of the left ventricle is sometimes difficult to visualise. When looking for LVHT, image resolution, contrast and positioning of the focus in the apical region are of crucial importance. Transducer frequency should be as high as possible, especially when using apical views to investigate the left ventricle. The introduction of second harmonic imaging was a great leap forward in this respect, which is substantiated by our experience. The echocardiographic machine used from June 1995 to April 1998 was an Aloka 870SSH with a 3.5-MHz transducer (Aloka, Tokyo, Japan). From May 1998, we used a Vingmed System Five with a 2.5–3.6-MHz transducer with second harmonic imaging (GE Vingmed Ultrasound, Horten, Norway). By applying the same diagnostic criteria, the prevalence of LVHT rose with the application of the improved echocardiographic equipment (table 1).

It has been shown that LVHT is sometimes not diagnosed by transthoracic echocardiography (table 2) but by transoesophageal echocardiography,[37] cardiac computed tomography,[4 38] ventriculography,[25 39] cardiac magnetic resonance imaging[27 38–41] or only pathoanatomically after cardiac transplantation or at autopsy.[7 18 42–45] The difficulties in visualising the trabeculations and deep intertrabecular recesses seem to be higher in small well-contracting left ventricles than in dilated poorly contracting left ventricles. This is substantiated by a report in which LVHT was diagnosed only after ventricular dilatation had occurred.[26] In another report, it is shown that LVHT was visible only in diastole.[40] The deep intertrabecular recesses may not be visible, either as a result of the cardiac contraction state or because of small thrombi within them.

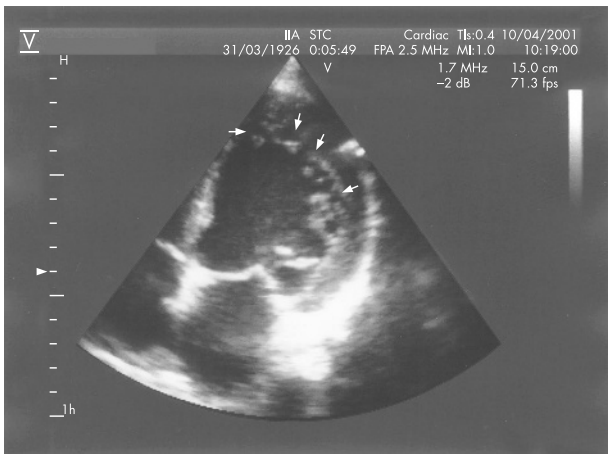

Figure 1 Echocardiographic apical four-chamber view showing left ventricular hypertrabeculation/non-compaction in the lateral and apical parts of the left ventricular wall. The trabeculations were misinterpreted as thrombi at two previous echocardiographic investigations.

Table 1 Prevalence of left ventricular hypertrabeculation/non-compaction diagnosed by echocardiography

Year	Echocardiographies (n)	LVHT (n)	Prevalence of LVHT (%)
1995*	1708	2	0.12
1996	3502	5	0.14
1997	3613	3	0.08
1998	3407	7	0.21
1999	3444	14	0.41
2000	3479	14	0.40
2001	3496	10	0.29
Total	22649	55	0.24

LVHT, left ventricular hypertrabeculation/non-compaction.
*From June to December 1995.

MISINTERPRETATION OF LVHT

Owing to these imponderabilities, LVHT has been misdiagnosed as hypertrophic cardiomyopathy,[10 24 26–29 33 34 40 43 45] dilated cardiomyopathy,[10 14 18 25 27 29–31] endocardial fibroelastosis,[3 10 42] endomyocardial fibrosis,[39] restrictive cardiomyopathy,[10 44] myocarditis,[10 32 35] thrombus,[7 38] localised left ventricular hypertrophy,[37] left ventricular mass[4] or myocardial/pericardial disease.[32]

Misinterpretation as hypertrophic cardiomyopathy may result because LVHT itself may manifest with thickened left ventricular walls, is preferably located in the apical region of the left ventricle and is also regarded as a subtype of the apical type of hypertrophic cardiomyopathy. LVHT is misinterpreted as dilated cardiomyopathy because the left ventricles of >50% of patients with LVHT are dilated and have poor systolic function.[11]

LVHT may be misinterpreted as endocardial fibroelastosis or endomyocardial fibrosis, as this abnormality is often located in the apical region and goes along with considerable endocardial thickening. LVHT was mainly misinterpreted as endocardial fibroelastosis or endomyocardial fibrosis in times when the transducer quality was poorer than it is today and the intertrabecular spaces could not be visualised adequately (table 2). On the other hand, the coincidence of LVHT and endocardial fibrosis has been reported.[44] In a further study, applying electron-beam computed tomography, prominent trabeculations are described as a manifestation of endomyocardial fibrosis.[46] Restrictive filling patterns have been reported in association with LVHT, endomyocardial fibrosis and endocardial fibroelastosis. This may account for establishing the diagnosis of restrictive cardiomyopathy in patients with LVHT.[10 25 44 47 48] Possibly, an overlap exists between these cardiac abnormalities, which might be mixed up or associated with LVHT. Cardiac phenotypes that occur as a result of G4.5 mutations may include dilated cardiomyopathy, endocardial fibroelastosis, LVHT and dilated hypertrophic cardiomyopathy.[19]

LVHT can be misinterpreted as a thrombus or left ventricular mass because of its location in the apical region and because echocardiographers are more familiar with this entity. LVHT may be misinterpreted as myocarditis because in myocarditis, reduced systolic function and thickening due to oedema may occur in different locations of the myocardium.

Table 2 Misinterpretations of left ventricular hypertrabeculation/non-compaction

Investigations before diagnosing LVHT (n)	LVHT diagnosed by	Reference number	Year
Dilated cardiomyopathy			
1	Repeated TTE	30	1985
1	Autopsy	18	1997
Several	Repeated TTE	10	1999
NI	Repeated TTE	14	1999
1	Repeated TTE	29	2001
1	Repeated TTE, MRI	27	2001
Several	Repeated TTE	31	2002
NI	Repeated TTE, ventriculography	25	2002
Endocardial fibroelastosis			
Several	Repeated TTE	3	1986
NI	Autopsy	42	1988
1	MRI, ventriculography	39	1995
Several	Repeated TTE	10	1999
Hypertrophic cardiomyopathy			
1	Autopsy	43	1993
2	Autopsy	45	1996
Several	Repeated TTE	24	1999
Several	Repeated TTE	10	1999
1	Repeated TTE	29	2001
5	Repeated TTE	26	2002
1	Repeated TTE	34	2002
Apical hypertrophic cardiomyopathy			
Several	Repeated TTE	10	1999
Several	Repeated TTE	28	2000
1	MRI	40	2002
NI	Repeated TTE	33	2002
Several	Repeated TTE	10	1999
2	TEE	37	2000
Restrictive cardiomyopathy			
1	Pathoanatomy	44	1996
Several	Repeated TTE	10	1999
Myocarditis			
1	Repeated TTE	35	1999
Several	Repeated TTE	10	1999
Several	Repeated TTE	32	2002
1	MRI	41	2002
Thrombus or ventricular mass			
NI	Pathoanatomy	7	1999
1	CT	4	1991
1	CT, MRI	38	2002

CT, computed tomography; LVHT, left ventricular hypertrabeculation; MRI, magnetic resonance imaging; NI, not indicated; TEE, transoesophageal echocardiography; TTE, transthoracic echocardiography.

FALSELY DIAGNOSED LVHT

Apart from overlooking LVHT, several cardiac abnormalities, including false tendons, aberrant bands, thrombi, the apical type of hypertrophic cardiomyopathy, fibromas, obliterative processes of the left ventricular cavity, intramyocardial haematomas, cardiac metastases and intramyocardial abscesses, may be falsely diagnosed as LVHT.

False tendons and aberrant bands are often found in the left ventricle. They present echocardiographically as myocardial structures, communicating on both ends with the myocardium. These communications can be visualised echocardiographically by using different imaging planes.[49–51] Thrombi can be differentiated from LVHT by echocardiography and, possibly, by magnetic resonance imaging. On echocardiography, thrombi present with an echodensity different from that of the myocardium and they do not move synchronously with the ventricular contractions. Thrombi are most often located in areas with impaired contractility or aneurysms.[52] First experiences in cardiac magnetic resonance imaging report that ventricular thrombi are visualised as structures with homogeneously low signals and without gadolinium enhancement.[53] The apical type of

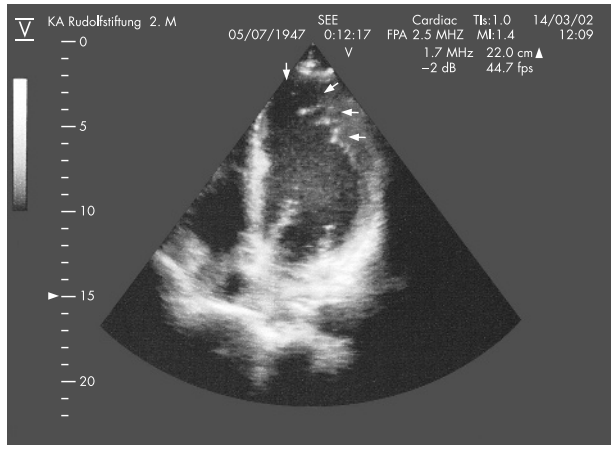

Figure 2 Left ventricular hypertrabeculation/non-compaction in a patient who had *Candida* sepsis. He eventually died 15 days after echocardiography. At autopsy, no hypertrabeculation was detected, but histologically multiple microabscesses were found in the myocardium.

hypertrophic cardiomyopathy is characterised by thickening of the apical myocardium but a smooth endocardial surface without trabeculations.[54] Cardiac fibromas have a homogeneous appearance, are well demarcated and often distort the cardiac anatomy.[55] Obliterative processes of the left ventricular cavity, caused by eosinophilic heart disease or endomyocardial fibrosis, often begin in the apex. The echocardiographic picture of these disorders is characterised by total obliteration of the ventricular apex, variable reduction in myocardial contractility, and hypoechogenic or hyperechogenic appearance of the obliterated region.[56 57] Cardiac metastases lead to a heterogeneously appearing myocardial structure, with irregular areas of low and high echodensity.[58] Intramyocardial haematoma, a rare complication of myocardial infarction, and cardiac microabscesses in *Candida* sepsis may result in an echocardiographic picture similar to that of LVHT,[59 60] which can be distinguished from LVHT only by surgery or autopsy (fig 2).

PRACTICAL IMPLICATIONS

To rule out overlooking and overdiagnosing LVHT, it is necessary that echocardiographers are more trained regarding the echocardiographic awareness of LVHT, the echocardiographic equipment is of a high standard, differential diagnoses of LVHT are considered if LVHT is suspected and, in cases with poor image quality, cardiac computed tomography or magnetic resonance imaging is carried out as an additional investigation to visualise the left ventricular apical region.[4 5 25 27 38 39 41 47 61]

Detection of LVHT would be facilitated by uniformly accepted, anatomically controlled diagnostic criteria for LVHT, which should be easily applicable even by less experienced echocardiographers. Owing to the lack of commonly accepted pathoanatomic diagnostic criteria, different echocardiographic criteria for LVHT are currently used. One group defines LVHT as ''numerous, excessively prominent trabeculations and deep intertrabecular recesses''.[6] In our experience, this definition is not clearcut, as it does not give a number above which the phenomenon is abnormal. On the contrary, we suggest the anatomically confirmed definition of >3 trabeculations in one imaging plane, apically from the insertion of the papillary muscles, as a practically useful diagnostic criterion when carrying out echocardiography, but also magnetic resonance imaging or computed tomography.[1 62]

Why LVHT is often associated with neuromuscular disorders is unknown. LVHT could be a cardiac manifestation of neuromuscular disorders. The prevalence of neuromuscular disorders was 82% in 49 patients with LVHT who were investigated neurologically.[1] In some of these cases, the neuromuscular disorder had not clinically manifested yet. In such cases, the diagnosis of LVHT may lead to the neurological diagnosis. Hence, it is recommended that patients with LVHT undergo a neurological investigation, irrespective of whether or not they have neurological symptoms.

If all these measures are realised, the incidence of LVHT may probably increase and thus more mild neuromuscular disorders can be detected earlier. This assumption is substantiated by a recent study on the epidemiology of childhood cardiomyopathy, in which a single cardiologist systematically reviewed all imaging information and found a prevalence of 9.2% of LVHT among all patients with cardiomyopathy.[23]

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