HYPERTROPHIC CARDIOMYOPATHY. LITERATURE REVIEW

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Conflict of interest
The authors declare that they have no conflicts of interest

Abstract

Hypertrophic cardiomyopathy is a common hereditary heart disease with a heterogeneous clinical picture and a natural history. Recent advances in diagnosis and treatment methods have played an important role in reducing the incidence of adverse clinical events; however, the complete elimination of sudden cardiac death is still an unattainable achievement. Despite the heterogeneous clinical profile and complex pathophysiology, effective treatment strategies are available, including implantable defibrillators to prevent sudden death, medical and surgical myectomy (or, alternatively, alcohol ablation of the septum) to alleviate outflow obstruction and symptoms of heart failure, as well as pharmacological strategies (and possibly radiofrequency ablation) to control atrial fibrillation and prevent embolic stroke. Now, after more than 50 years, hypertrophic cardiomyopathy has been transformed from a rare and largely untreatable disorder to a common genetic disease with management strategies that permit realistic aspirations for restored quality of life and advanced longevity. This article discusses some aspects of this condition: epidemiology, clinic, diagnosis and surgery technique.

Objective.*Evaluate the effectiveness of surgical treatment of patients with hypertrophic cardiomyopathy.*

Material and methods.*This literature review was carried out in accordance with the PRISM statement. The databases searched in this review included Pubmed, Web of Science, Scopus, and Cochrane for systematic reviews.*

Conclusion. *The diagnosis of HCMP is based mainly on echocardiographic variables including the dynamic parameters of LV, LVOT the distribution of increased muscle thickness, the mechanism and severity of MR as well as the degree of diastolic dysfunction.*

Keywords
HCMP, echocardiography, left ventricle.

Гипертрофическая кардиомиопатия. Обзор литературы

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Конфликт интересов
Авторы заявляют об отсутствии конфликта интересов

Аннотация

Гипертрофическая кардиомиопатия - распространенное наследственное заболевание сердца с неоднородной клинической картиной и естественным анамнезом. Последние достижения в методах диагностики и лечения сыграли важную роль в снижении частоты неблагоприятных клинических проявлений; однако полное устранение внезапной сердечной смерти по-прежнему остается недостижимым достижением. Несмотря на неоднородный клинический профиль и сложную патофизиологию, доступны эффективные стратегии лечения, включая, имплантируемые дефибрилляторы для предотвращения внезапной смерти, медикаментозную и хирургическую миэктомию (или, альтернативно, алкогольную абляцию перегородки) для облегчения обструкции оттока и симптомов сердечной недостаточности. А также фармакологические стратегии (и, возможно, радиочастотная абляция) для контроля фибрилляции предсердий и предотвращения эмболического инсульта. Теперь, по прошествии более чем 50 лет, гипертрофическая кардиомиопатия превратилась из редкого и в значительной степени неизлечимого заболевания в распространенное генетическое заболевание со стратегиями лечения, которые позволяют реалистично стремиться к восстановлению качества жизни и увеличению продолжительности жизни. В этой статье рассматриваются некоторые аспекты этого состояния: эпидемиология, клиника, диагностика и методика хирургического вмешательства.

Цель исследования. *Оценить эффективность хирургического лечения пациентов с гипертрофической кардиомиопатией.*

Материал и методы. *Этот литературный обзор был выполнен в соответствии с заявлением PRISM. Базы данных, в которых проводился поиск в этом обзоре, включали базы данных Pubmed, Web of Science, Scopus и Cochrane для систематических обзоров.*

Заключение. *Диагноз ГКМП основывается преимущественно на эхокардиографических переменных, включая динамические показатели ЛЖ, ВОЛЖ, распределение увеличенной толщины мышц, механизм и тяжесть МН, а также степень диастолической дисфункции.*

Ключевые слова
ГКМП, эхокардиография, левый желудочек

Гипертрофиялық кардиомиопатия. Әдебиет шолу

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Аңдатпа

Гипертрофиялық кардиомиопатия-гетерогенді клиникалық көрінісі және табиғи тарихы бар жалпы тұқым қуалайтын жүрек ауруы. Диагностикалық және емдеу әдістеріндегі соңғы жетістіктер қолайсыз клиникалық көріністердің жиілігін төмендетуде маңызды рөл атқарды; алайда кенеттен жүрек өлімін толығымен жою әлі де қол жетпейтін жетістік болып қала береді. Біртекті емес клиникалық профильге және күрделі патофизиологияға қарамастан, тиімді емдеу стратегиялары бар, соның ішінде кенеттен өлімнің алдын-алу үшін имплантацияланатын дефибрилляторлар, ағып кетудің және жүрек жеткіліксіздігінің белгілерін жеңілдету үшін дәрі-дәрмектер мен хирургиялық миэктомия (немесе алкогольді септальды абляция). Сондай-ақ, атриальды фибрилляцияны бақылауға және эмболиялық инсульттің алдын алуға арналған фармакологиялық стратегиялар (және мүмкін радиожиілікті абляция). Енді 50 жылдан астам уақыттан кейін гипертрофиялық кардиомиопатия сирек кездесетін және көбінесе емделмейтін аурудан жалпы генетикалық ауруға айналды, бұл өмір сүру сапасын қалпына келтіруге және өмір сүру ұзақтығын арттыруға нақты ұмтылуға мүмкіндік беретін емдеу стратегиялары бар. Бұл мақалада осы жағдайдың кейбір аспектілері қарастырылады: эпидемиология, клиника, диагностика және хирургиялық әдіс.

Мақсаты. *Гипертрофиялық кардиомиопатиясы бар науқастарды хирургиялық емдеудің тиімділігін бағалау.*

Материал және әдістер. *Бұл әдеби шолу орындалды prism мәлімдемесіне сәйкес. Өткізілген деректер базасы бұл шолуда іздеу Pubmed, Web of Science, Scopus және жүйелі шолулар үшін Cochrane.*

Қорытынды. *Гипертрофиялық кардиомиопатия диагнозы негізінен өтірік динамикалық көрсеткіштерін қоса, эхокардиографиялық айнымалыларға негізделген, СҚ, СҚШБ бұлшықет қалыңдығының таралуы, механизмі мен ауырлығы, сондай-ақ диастолалық дисфункция дәрежесі.*

Түйін сөздер
ГКМП, эхокардиография, сол жақ қарынша

Relevance

Hypertrophic cardiomyopathy is a common hereditary cardiovascular disease occurring in one out of 500 people in the whole population [1-3]. It is caused by more than 1400 mutations in 11 or more genes [4-8] encoding cardiac sarcomere proteins. Although hypertrophic cardiomyopathy is the most common cause of sudden death in young people (including trained athletes) [9, 10] and can lead to functional disability as a result of heart failure and stroke, most affected people probably remain undiagnosed, and many do not have a significant reduction in life expectancy or significant symptoms. The diagnosis is most often made by echocardiographic assessment of left ventricular hypertrophy, gradients of the left ventricular outlet tract, systolic and diastolic function, as well as mitral valve anatomy and function. Magnetic resonance imaging of the heart also plays a diagnostic role, determining the degree and localization of left ventricular hypertrophy, and anatomical abnormalities of the mitral valve and papillary muscles.

Pathophysiology in HCMP

HCMP is defined as an abnormal thickening of the LV without expansion of the chamber, which is usually asymmetric, develops in the absence of an identifiable cause (for example, aortic valve stenosis, hypertension) and is associated with a violation of myocardial fibers [11, 12]. The main structural anomalies underlying HCMP are [1] disorder of myocardial cells when the cells are in an unorganized state, in contrast to the normal parallel arrangement of myocytes; [2] dysfunction of the coronary microcirculatory bed due to an increase in the wall/lumen ratio; and [3]remodeling changes [13, 14]. In intramiocardial arterioles <80 microns, studies have shown a 2-fold increase in the wall-to-lumen ratio, predisposing patients with silent myocardial ischemia, ongoing myocardial damage and fibrosis. Moreover, these changes are not limited to the areas of LVH and myocardial remodeling, which occur as a compensatory mechanism and may include changes in myocytes, fibroblasts and interstitials. These changes develop over many years before symptoms appear. Disorganized pattern of myocytes, increased wall/lumen ratio of coronary arteries and remodeling changes in patients with HCMP lead to impaired coronary reserve, diastolic dysfunction, supraventricular and ventricular rhythm disturbances, and sudden death. LV remodeling may include fibrosis, diffuse, asymmetric, focal or concentric hypertrophy, as well as a decrease in the size of the cavity [15, 16]. Obstruction of the excretory tract of the left ventricle occurs with HCMP, and it was initially thought that basal septum hypertrophy invading the LVOT caused the obstruction. However, later studies have shown that during ventricular systole, the flow against the incorrectly positioned mitral valve apparatus (MV) leads to the appearance of resistance forces on part of the valves, which are then pushed into LVOT [17-21] anomalias of the MK apparatus may include displacement of papillary muscles in front, hypertrophied papillary muscles in contact with the septum, elongated mitral flaps or abnormal insertion of the papillary muscle into the anterior mitral flap [18, 21, 22].

The enlargement of the left ventricle may be accelerated or aggravated by a decrease in the final diastolic volume or systemic arterial resistance or an increase in contractility or heart rate [23].

Modern classification of diseases:

Idiopathic hypertrophic subaortic stenosis.

Asymmetric hypertrophy of the septum without changes from the aortic and mitral valves, without obstruction of the LV exit tract.

Apical HCMP with restriction of the hypertrophy zone to the apical region. Symmetrical HCMP with concentric LV myocardial hypertrophy.

The last 3 forms are rare and are not accompanied by the development of obstruction of the LV outflow tract.

Classification of the New York Heart Association's HCMP:

- I degree - pressure gradient not higher than 25 mm Hg.
- II degree - pressure gradient from 25 to 36 mm Hg.
- III degree - pressure gradient from 36 to 44 mm Hg.
- IV degree - pressure gradient 45 mm Hg.

Classification by degree of hypertrophy:

* moderate - the thickness of hypertrophy is 15-20 mm;
* average - hypertrophy thickness of 21-25 mm;
* pronounced - the thickness of hypertrophy is more than 25 mm.

Table 1.
Electro-and Echocardiographic examinations at HCMP

Nonspecific Electrocardiogram changes associated with hypertrophic cardiomyopathy
- Hypertrophy of the left ventricle (S-shaped wave in V1 ≥35 mm; R-shaped wave in V5 >35 mm)
- Left axis deviation/left front hemiblock
- Intraventricular conduction delay (QRS >0.12 ms)
- Enlargement of the left atrium (wide toothed wave P in lead II; deeply inverted wave P in V1)
- Pathological Q-waves
- Poor progression of the R wave in precordial leads
- Supraventricular arrhythmias (most often atrial fibrillation)
- Full block of package branches
- ST segment depression
- Inverted T-waves in ≥2 consecutive leads

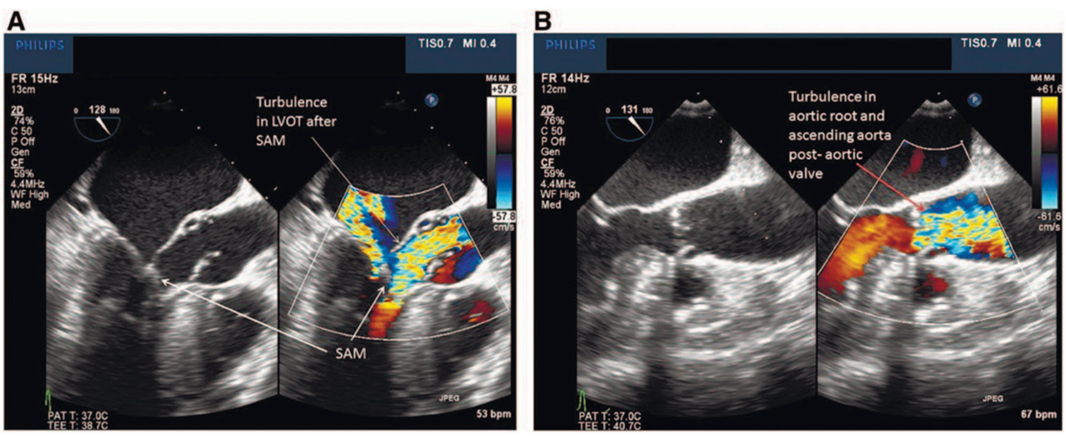

Figure 1.
A, image of transesophageal echocardiography (TEE) -color flow Dopplerography (CFD) images of the middle long axis of the esophagus (ME-LAX) in a patient with hypertrophic cardiomyopathy with turbulence in the excretory tract of the left ventricle (LVOT) at the level of systolic anterior movement of the mitral valve (SAM) (proximal to the aortic valve). B, TEE-CFD image of the ME-LAX image in a patient with valvular aortic stenosis showing laminar flow in the LVOT and turbulence distal to the affected aortic valve. (Hypertrophic cardiomyopathy: a review Nadia Hensley 1, Jennifer Dietrich, Daniel Nyhan, Nanhi Mitter, May-Sann Yee, MaryBeth Brady)

Table 2.
Echocardiographic focus in hypertrophic cardiomyopathy

Echocardiographic focus in hypertrophic cardiomyopathy
1. The presence of hypertrophy and its distribution; report measurements of the size of the left ventricle, wall thickness (septum, posterior, maximum)
2. Left ventricular ejection fraction
3. Pancreatic hypertrophy and the presence of dynamic pancreatic obstruction
4. The volume of the left ventricle, indexed by body surface area
5. Diastolic function of the left ventricle (pressure of relaxation and filling)
6. Systolic pressure in the pulmonary artery
7. Dynamic obstruction at rest and with Valsalva, the place of obstruction and the slope
8. Evaluation of the mitral valve and apparatus, details of mitral regurgitation (i.e. mechanism, severity);

Surgical technique: In this article we will carefully focus on the technique: transaortic myoectomy. An attempt at a basal septum myectomy using transaortic access was originally described by Morrow in 1961 [25-31], but it was first performed in 1958 and subsequently described by Kleland in 1963 [32-37]. The initial report described a limited myectomy without a specific anatomical resection. The technique of formal basal myectomy was later published in 1975. Initially, this method involved excision of a rectangular segment of the septum myocardium under the flap of the right coronary aortic valve which extended apically to the point of contact of the septum of the anterior flap of the mitral valve. This point is usually delimited by a fibrous scar which develops a second time due to the constant contact of the valve leaf with the septum myocardium during systole. The total myocardial sample excised during Morrow's myectomy is approximately 3-4 cm long, 1 cm wide and 1.5 cm deep [27]. More recently the standard transaortic procedure has turned into an extended septal myectomy. This procedure creates a longer myocardial excision and opens the LVOT more apically than the Morrow procedure. Following the initiation of artificial circulation (CPB), the exposure of the left ventricle is achieved by an oblique aortotomy performed through the midpoint of the non-coronary sinus of the aorta and ending about 1 cm above the aortic ring. Polypropylene seams remain or not. The Ross retractor keeps the aorta open, and the suction tip for cardiotomy is used to retract and protect the anterior flap of the mitral valve. Depending on the surgeon's preferences, scalpel No. 10 or 11 is used to cut the septum, starting directly under the nadir of the right aortic valve leaf and directed to the left. to the anterior flap of the mitral valve, removing the basal part of the hypertrophied septum. The incision in this area is carefully marked, because a tissue rupture further to the right of the midpoint of the right valve leaf will increase the risk of damage to the membranous septum and disruption of the conductive tissue, thereby significantly increasing the likelihood of complete heart block. Then, starting again from the area of the initial incision, the area of the cut-out septum is lengthened to the apex of the heart, making sure that the excision is performed outside the endocardial fibrous scar and in the apical trabeculations. The completed myectomy extends from the subaortic level, about 5 mm below the aortic ring, to the level of the middle ventricle, opposite the base of the anterior papillary muscle of the mitral valve, with a total length of about 7 cm.

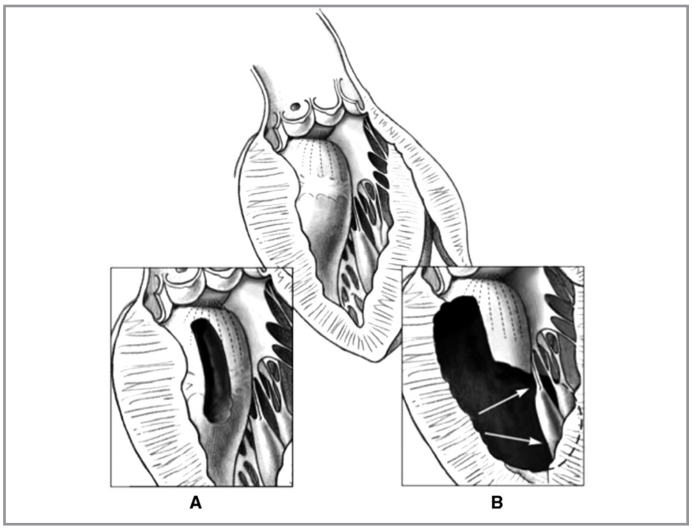

Figure 2.
Comparison of the classic Morrow procedure (A) with the modification of the extended septal myectomy (B). The resection of the septum wall expands to the top, to the free wall on the left side of the image, and then to the right, as indicated by the white arrows. The dotted lines in the basal septum represent the bundles of the left bundle emanating from the membranous septum

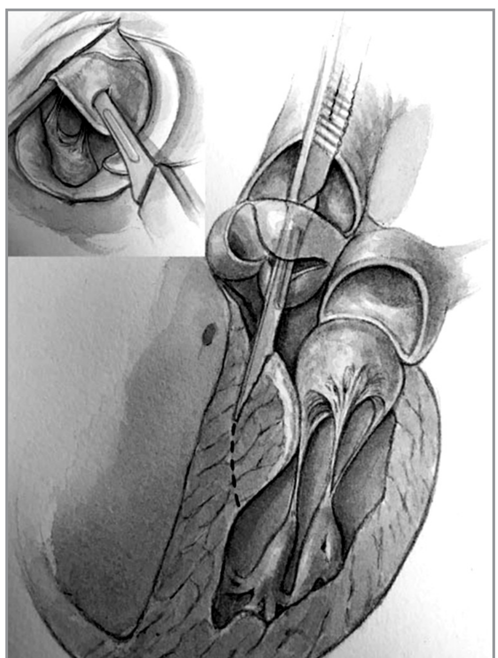

Figure 3.
(a) Extended thymectomy of the basal septum using supravalvular aortotomy. (b) The surgeon's view through the aortotomy, determining the hypertrophied septum directly below the right coronary aortic valve leaf

Conclusion. The diagnosis of HCMP is based mainly on echocardiographic variables including the dynamic parameters of LV, LVOT the distribution of increased muscle thickness, the mechanism and severity of MR as well as the degree of diastolic dysfunction. Current indications for surgical intervention include patients with symptoms that are immune to drug therapy who can tolerate the risk of surgical intervention and patients with pronounced outflow gradients, even if they are asymptomatic.

Despite the ambiguity the mechanism underlying the improvement of symptoms, LV condition and long-term survival after myectomy is at least partially due to LV regression. It is extremely important for cardiac surgeons to understand the mechanisms of this disease in order to best manage these patients in perioperative conditions. It is very important to diagnose these HCMP patients in time, provide the necessary therapy and hospitalization for surgical treatment.

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