Article/Review

Gipertrofik kardiomiopatiyaning obstruktiv shaklida yurakning remodellashuv xususiyatlari, Morrou bo'yicha mioektomiyaning samaradorligi

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Annotatsiya:
Obstruktiv gipertrofik kardiomiopatiya (OGKMP)– genetik omillar tufayli yurakning remodellashuvi bilan kechuvchi kasallikdir. OGKMP chap qorinchaning chiqish yolida dinamik obstruksiya, diastolik disfunksiya, mitral regurgitatsiya, miokard ishemiyasi va yurak ritmi buzilishlari rivojlanishiga sabab bolishi mumkin. Klinik jihatdan bemorlarda yurak yetishmovchiligi belgilari bilan namoyon boladi. OGKMP, ayniqsa, yoshlarda va sportchilar orasida to'satdan yurak o'limining eng keng tarqalgan sababi hisoblanadi. AQSh va Yevropa davlatlarida otkazilgan epidemiologik kuzatuv natijalariga ko'ra, 35 yoshgacha bo'lgan sportchilarda to'satdan yurak o'limi 30–40% holatlarda OGKMP bilan bog'liq. Bugungi kunda, kasallikni davolashning zamonaviy noinvaziv usullari mavjudligiga qaramay, xirurgik usul samaradorligi ustun kelmoqda. Maqolada ushbu kasallikda yurakning remodellashuv xusussiyatlari, ushbu yonalishda olib borilgan klinik tadqiqotlar natijalari va davo usullarining samaradorligi haqida adabiyotlarda keltirilgan ma'lumotlar tahlil qilinadi.

Kalit so'zlar: gipertrofik kardiomiopatiya, Morrou mioektomiyasi, diastolik disfunksiya, yurakning remodellashuvi.

Heart remodeling in the obstructive form of hypertrophic cardiomyopathy, efficacy of Morrow septal myectomy

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Abstract:
Obstructive hypertrophic cardiomyopathy (OHCMP) is a disease characterized by cardiac remodeling due to genetic factors. OHCMP can cause the development of dynamic obstruction in the outflow tract of the left ventricle, diastolic dysfunction, mitral regurgitation, myocardial ischemia, and cardiac arrhythmias. Clinically, patients are manifested by symptoms of heart failure. OHCMP is the most common cause of sudden cardiac death, especially in young people and among athletes. According to the results of epidemiological observations conducted in the USA and European countries, sudden cardiac death in athletes under 35 years of age is associated with OHCMP in 30–40% of cases. Today, despite the availability of modern non-invasive methods of treating the

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disease, the effectiveness of the surgical method prevails. The article analyzes the data presented in the literature on the features of cardiac remodeling in this disease, the results of clinical studies conducted in this area, and the effectiveness of treatment methods.

Keywords: hypertrophic cardiomyopathy, Morrow myectomy, diastolic dysfunction, cardiac remodeling.

Kirish

Gipertrofik kardiomiopatiya (GKMP) — bu yurak mushak qavatining, aksariyat holatlarda chap qorincha devorining (kopincha qorinchalararo tosiqning) qalinlashuvi (gipertrofiyasi) bilan tavfsiflanadigan genetik kasallikdir [21]. Bu holat yurakning qon bilan tolishi va qon haydashini qiyinlashtiradi, yurakning elektr va mexanik faoliyatini buzilishiga sabab boladi [1].

GKMP autosomal-dominant yol bilan irsiylanuvchi kasallik bolib, uchrash chastotasi 1:500 [13, 21]. Patogen ozgarishlar MYH7 va MYBPC3 kabi genlarda sodir bolib [11,16], sarkomer oqsillarning relaksatsiyasini ozgartiradi, kaltsiy almashinuviga ta'sir etadi va energiya talabini oshiradi [1]. Bemorlarning taxminan 60%da sarkomer ozgarishlar aniqlanmaydi, ba'zi bemorlarda oilaviy anamnez yoki poligen etiologiya mavjud [8]. GKMP erkaklar va ayollar ortasida deyarli teng darajada uchraydi [21]. Kasallik har qanday yoshda, shu jumladan bolalik davrida ham aniqlanishi mumkin. Biroq klinik simptomlar kopincha 15–35 yosh oraligida namoyon boladi [13]. Aynan shu yoshdagi bemorlarda yurak gipertrofiyasi klinik belgilari namoyon bola boshlaydi. Shuningdek, kasallik osmirlik davrida aniqlanmagan bolsa, yoshi ulgayganda tosatdan yurak olimi korinishida birinchi marta namoyon bolishi mumkin [5]. Bugungi kunda GKMP tashxisi uchun asosiy mezon bu- chap qorincha diastolik disfunksiya mavjud bolgan holda miokard qalinligi 1,5 sm va undan ortiq bolishidir [21].

Chap qorincha chiqish yolida obstruksiya mavjudligiga kora GKMPning obstruktiv va noobstruktiv shakllari tafovut qilinadi [10]. Obstruktiv gipertrofik kardiomiopatiya (OGKMP)- obstruksiya natijasida chap va/yoki ong qorinchadan aortaga qon haydalishining buzilishi hisobiga intrakardial gemodinamik buzilishlar bilan kechuvchi, chap va/yoki ong qorinchaning chiqish yolida bosim gradientining ortishiga sabab boluvchi GKMP shakli hisoblanadi [11].

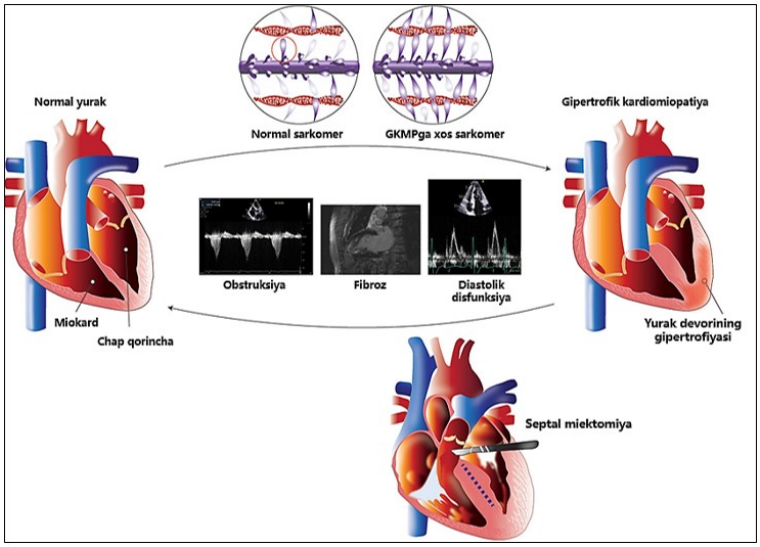

Figure 1. Cardiac Remodeling in Hypertrophic Cardiomyopathy
Rasm 1. Gipertrofik kardiyomiyopatiyada yurakning remodellanishi

Obstruktiv gipertrofik kardiomiopatiyada yurakning remodellashuv xususiyatlari. OGKMP yurakdagi sezilarli struktur va funksional ozgarishlar bilan tavsiflanib, ayniqsa chap qorinchaning chiqish yolidagi (ChQChY) ozgarishlar bilan kechadi. Aksariyat hollarda asimmetrik gipertrofiya kuzatilib, bazal tosiqning qalinlashuvi natijasida ChQChY torayadi, bu esa sistolik obstruksiyani yuzaga keltiradi [6]. Bazi bemorlarda esa gipertrofiya konsentrik korinishda bolib,

butun miokard devorining qalinlashishi va chap qorincha boshligining torayishi bilan namoyon boladi [8]. (1-rasm)

OGKMPda yurakning struktur remodellanishi quyidagi o‘zgarishlarni o‘z ichiga oladi: miokard gipertrofiyasi, kichik tomirlardagi ishemiya, hamda fibroz (to‘qima chandiqlanishi) [20]. Bu o‘zgarishlar nafaqat sarkomer oqsillarni kodlovchi genlardagi mutatsiyalar, balki ChQChYda yuzaga keladigan gemodinamik yuklama kabi ikkilamchi omillar bilan ham bog‘liq [1].

OGKMP mavjud bemorlarda miokard toqimasini mikroskopik organilganda, kardiomiotsitlarning tartibsiz joylashuvi (disarray syndrome) [6] va interstitsial fibroz aniqlandi. Ushbu ozgarishlar miokardning elastikligini kamaytirib, mushak toqimasining qattiqligini oshiradi, bu esa diastolik disfunksiya, aritmogen holatlar va boshqa funksional buzilishlar rivojlanishiga sabab boladi [10]. Shuningdek, miokard gipertrofiyasi darajasi ko‘pincha miokard devori zo‘riqishini va kasallik og‘irligini aks ettiruvchi biologik ko‘rsatkichlar — ya’ni miya natriyuretik peptidi (BNP) va N-terminalli pro-BNP (NT-proBNP) darajalarining oshishi bilan bog‘liqligi aniqlangan [7].

Miokardning gipertrofiyasi va fibrozlanishi yurakning diastolik disfunksiyasiga sabab boladi– qorincha diastola paytida tola kengaya olmaydi. Natijada chap qorinchaning qon bilan tolishi qiyinlashib, intraventrikulyar diastolik bosim oshadi [19]. Bu holat yurakning “gemodinamik siqilishiga” olib kelib, vaqt o tishi bilan chap bolmacha kengayadi [6]. OGKMP mavjud bemorlarida otkazilgan septal mioektomiya jarohatidan keyin ham chap bolmacha hajmi normal korsatkichlarga qaytmasligi kuzatilgan [21]– bu diastolik disfunktsiya natijasida bolmacha turgun kengayganini anglatadi. Deyarli barcha OGKMP mavjud bemorlarida malum darajada diastolik disfunksiya mavjud bolib, bu ularda yurak yetishmovchiligi (YY) alomatlarini keltirib chiqaradi [6].

OGKMPda yurakning anatomik tuzilishi ham ozgaradi. Gipertrofiyalashgan qorinchalararo tosiq va kichik hajmli chap qorincha boshligi “banansimon” geometrik shaklni egallaydi [18]. Shu bilan birga, mitral klapan apparatining anomaliyalari (qopqoqchalar elongatsiyasi yoki ortiqcha toqimalar, papillyar mushaklarning gipertrifikatsiyasi va notogri joylashishi) rivojlanadi [19]. Natijada sistola paytida mitral klapan oldingi varagining patologik oldinga siljishi – “sistolik oldingi harakat” (SAM fenomeni) yuzaga keladi [1]. SAM fenomeni [19] mitral qopqoq tabaqasi va qalinlashgan tosiqning kontaktiga sabab bolib, chiqish yolini yanada toraytiradi va ikkilamchi mitral yetishmovchilikni keltirib chiqaradi. Shunday qilib, OGKMPda anatomik (tosiq va qopqoqchalar tuzilishi) va funksional (dinamik SAM) ozgarishlar kombinatsiyasi yurakning patologik remodellashuvini belgilaydi [3].

Morrou boyicha mioektomiya samaradorligi. Morrou boyicha transaortal septal mioektomiya — OGKMda chap qorinchaning chiqish yo‘lida yuzaga kelgan obstruksiyani bartaraf etishda oltin standart hisoblanadi [21]. Ushbu operatsiya davomida qalinlashgan qorinchalararo tosiqning ma’lum bir qismi (odatda 5–10 gramm to‘qima) [10] rezeksiya qilinadi va natijada chiqish traktining fiziologik kengligi tiklanadi. Amaliy natijada ChQChY obstruksiyasi sezilarli darajada kamayadi, SAM-fenomen (mitral klapaning tosiqqa tortilishi) sabab yuzaga kelgan mitral regurgitatsiya darhol kamayadi [9,10]. Bu o‘zgarishlar yurak ichki bosimini me’yorlashuvi va bemorning gemodinamik holati yaxshilanishi, klinik simptomlarning kamayishi bilan namoyon boladi [2].

Morrou boyicha mioektomiya chiqish yo‘lidagi obstruksiyani bartaraf etib, subaortal gradientni deyarli nol darajasiga tushiradi [10]. Keng ko‘lamli tadqiqotlarda operatsiyadan so‘ng ChQChY gradienti o‘rtacha 50–90 mm.sim.ust. dan 5–10 mm.sim.ust. gacha kamaygani aniqlangan [12,14]. Masalan, otkazilgan markaziy tadqiqotda mioektomiya natijasida ChQChY bosim gradienti 89 mm mm.sim.ust.dan 16 mm.sim.ust. pasaygan; mitral regurgitatsiya og‘irligi esa bemorlarning 78%ida mo‘tadil- og‘ir darajadan atigi 7%gacha tushgan [12].

Morrou boyicha septal mioektomiya ochiq yurak operatsiyasi bo‘lib, uning oziga xos ayrim asoratlar mavjud. Biroq tajribali markazlarda 30 kunlik o‘lim darajasi atigi 0,5–1% ni tashkil etadi [5]. Eng keng tarqalgan asorat - atrioventrikulyar blokada bo‘lib [8] bu holat Giss tutami zararlanishi bilan bog‘liq va bemorlarning 2–5%ida doimiy elektrokardiostimulyator o‘rnatishni talab qiladi [22]. Qorinchalararo to‘siqning chuqur rezeksiyasi natijasida tosiq defekti rivojlanishi (1%) mumkin [15]. Operatsiyadan keyin yurak ritm buzilishlari, xususan bolmachalar fibrillyatsiyasi 20% gacha bemorlarda kuzatilgan [17,22]. Kam hollarda esa insult (<1%) yoki boshqa tromboembolik asoratlar qayd etiladi [15]. Reabilitatsiya davri o‘rtacha 6–9 kun davom etadi [10]. Shunga qaramay, tajribali jamoalar ishtirokida bu asoratlarning chastotasi minimal darajada bo‘ladi.

2014 yilda bir guruh amerikalik mualliflar 1998-2010 yillar oraligida 665 bemorda ChQChY obstruktsiyasini jarrohlik yoli bilan davolashning uzoq muddatli natijalarini tavsiflovchi kop markazli tadqiqot natijalarini nashr etdilar. Umumiy shifoxona olimi 5,9% ni tashkil etdi. 8,7% hollarda toliq AV blokadasi tufayli doimiy yurak stimulyatori implantatsiyasi talab qilingan [15].

Klinik jihatdan, 94% ga yaqin bemorlarda YY simptomlari NYHA bo‘yicha I–II funksional sinfgacha yaxshilanishga erishilishi kuzatilgan [2]. Ya’ni hansirash, ko‘krak sohasida og‘riq, sinkope kabi simptomlar sezilarli darajada kamaygan. Septal mioektomiya nafaqat simptomlarni bartaraf etadi, balki bemorlarning uzoq muddatli hayot davomiyligini oshiradi [2]. Tadqiqotlarga ko‘ra [9]: 5 yillik tirik qolish – 94%; 10 yillik tirik qolish – 90%ni tashkil etadi. Bu ko‘rsatkichlar sog‘lom populyatsiya bilan taqqoslaganda ahamiyatli farq qilmaydi. Bemorlarning 90% dan ortig‘i yillar davomida simptomsiz hayot kechirishga muvaffaq bo‘lgan [2].

To‘satdan yurak o‘limi va og‘ir aritmiyalar chastotasi mioektomiya o‘tkazilgan bemorlarda pasayadi yoki o‘zgarishsiz qoladi [3]. Shu bilan birga, genetik va miokardial substrat saqlanib qolishi mumkinligi sababli, ayrim holatlarda implantatsiyalanuvchi defibrillyator (IDF) tavsiya qilinadi [22]. Mioektomiya aritmogenezni to‘liq bartaraf qilmasada, yurakning nasos funksiyasini yaxshilaydi va yurak yetishmovchiligi xavfini kamaytiradi [2].

Toronto klinikasida 649 nafar OGKMP bemorlari ustida olib borilgan tadqiqotda invaziv muolaja olgan bemorlarning 10 yillik tirik qolish ko‘rsatkichi – 87,8%, faqat dori bilan davolanganlarda esa 75,8% (p=0.008) bo‘lgan [9]. Shu bois, simptomatik OGKMPda medikamentoz terapiya yetarli bo‘lmasa, invaziv muolajalar — mioektomiya yoki alkogol ablatsiyasi tavsiya etiladi [23]. Invaziv usullar simptomlarni kamaytiradi va hayot davomiyligini uzaytiradi, biroq OGKMPning genetik-asosiy xavflarini- masalan, aritmiyalarni- to‘liq yo‘q qila olmaydi.

Mioektomiya va alkogol septal ablatsiyasi (ASA) [4]- chap qorincha chiqish yoli obstruktsiyasini bartaraf etishga qaratilgan ikki asosiy invaziv muolajadir.

ASA [17]: kam invaziv, ammo ablatsiya orqali sun’iy chuqurcha hosil qilinadi. Davolash samarasi uchun 3–6 oy vaqt talab qilinadi [4]. Klinik samaradorlik jihatidan ikkala usul ham NYHA bo‘yicha simptomlarni yaxshilaydi [2]. 10 yillik tirik qolish: mioektomiya – 85%, ASA – 82% (farq ahamiyatsiz) [2,17].

Asoratlar farqi:

- ASA: AV-qamal xavfi yuqoriroq- 10–20% bemorlarga implantatsiyalanuvchi defibrillyator (IDF) kerak bo‘ladi [17].
- Mioektomiya: doimiy implantatsiyalanuvchi defibrillyator (IDF) zarurati 2–4% atrofida [22].
- Takroriy aralashuv ehtiyoji: ASA – 10–15%, mioektomiyada- kam (<5%) [2,17].

Umuman olganda, mioektomiya ko‘pincha bir martalik samarali yechim hisoblanadi, ASA esa muayyan guruh bemorlar (yoshi katta, operatsion xavfi yuqori) uchun tavsiya etiladi.

Xulosa

Yuqoridagi malumotlar OGKMP holatlarida septal mioektomiya hozircha eng yuqori klinik samaradorlikka ega usul bo‘lib qolayotganini ko‘rsatadi: u ChQChY obstruktsiyasini deyarli to‘liq bartaraf etadi, transaortal bosim gradientini keskin pasaytiradi va uzoq muddatli omon qolish hamda hayot sifatini yaxshilaydi. ASA esa kamroq invaziv alternativ sifatida tanlanishi mumkin; u ham simptomlarni yengillashtiradi va ayrim kogort tadqiqotlarda uzoq muddatli natijalari jarrohlik aralashuviga yaqinlashadi. Optimal strategiyani tanlashda markazning tajribasi, bemorning yoshi, yondosh kasalliklar, qorinchalararo tosiqning qalinlashuv darajasi, mitral klapan va papillyar mushaklarning anatomik xususiyatlari, shuningdek koronar qon tomirlarning xusussiyatlari hisobga olinadi.

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Authors' contribution.

Conceptualization, T.A.; methodology, N.R.; software, N.R.; validation, T.A.; formal analysis, T.A.; investigation, N.R.; resources, N.R.; data curation, T.A.; writing — original draft preparation, N.R.; writing — review and editing, N.R., Hakimov A.A.; visualization, N.R., Hakimov A.A.; supervision, T.A.; project administration, T.A.; funding acquisition, T.A. All authors have read and agreed to the published version of the manuscript.

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Ethics approval.

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Republican Specialized Scientific and Practical Medical Center of Cardiology.

Nashrga xabardor qilingan rozilik.

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Consent for publication.

Informed consent was obtained from all participants involved in the study.

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Tadqiqotda keltirilgan barcha ma'lumotlar adabiyotlar ro‘yxatida ko‘rsatilgan manbalardan olingan.

Data Availability Statement

All data presented in the study are derived from the sources listed in the reference section.

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Conflict of interest

The authors declare no conflicts of interest.

Qisqartmalar

ASA alkogol	septal ablyatsiya
ChQChY	chap qorincha chiqish yo‘li
GKMP	gipertrofik kardiomiopatiya
IKD	implantatsion kardioverter defibrillyator
IVT	interventrikulyar tosiq
MR	mitral regurgitatsiya
NYHA	Nyu York Yurak Assotsiatsiyasi
OGKMP	obstruktiv gipertrofik kardiomiopatiya
TYO	to‘satdan yurak o‘limi
YY	yurak yetishmovchiligi

Adabiyot

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