Dijabetička kardiomiopatija — definira se kao disfunkcija ventrikula koja nastaje neovisno o koronarnoj bolesti i hipertenziji. Za razvoj klinički manifestne dijabetičke kardiomiopatije može biti potrebno i nekoliko godina. Dijastolička disfunkcija predstavlja najraniju predkliničku manifestaciju bolesti koja prethodi sistoličkoj disfunkciji koja se kasnije može razviti u simptomatsko zatajivanje srca. Ehokardiografski se mogu utvrditi tri različita stadija dijabetičke kardiomiopatije: 1. rani stadij - nalaz bez strukturnih promjena (normalne dimenzije LV), a kardijalna disfunkcija može se utvrditi samo senzitivnijim metodama kao što je tkivni dopler. 2. srednji stadij karakteriziran je manjim promjenama veličine i debljine stijenki LV, ali sa značajnim promjenama dijastoličke i sistoličke funkcije koje mogu biti utvrđene konvencionalnom ehokardiografijom. 3. kasni stadij obuhvaća značajne strukturne i funkcionalne promjene.

Zaključak — Ehokardiografija je dijagnostička metoda koju bi bilo opravdano učiniti kod svakog novootkrivenog dijabetičara, a preporuka učestalosti kontrolnih pregleda ovisila bi o samom nalazu ali i pratećim čimbenicima rizika. Međutim, "cost benefit" takvog pristupa za sada nije opravdan, a ehokardiografija je trenutno indicirana kod svih dijabetičara sa sumnjom na kardiovaskularnu bolest kao i kod onih koji imaju dodatne čimbenike rizika.

Received: 17th Mar 2008

E-mail: kresimir.stambuk@zg.t-com.hr

Diabetic cardiomyopathy — is defined as a dysfunctional ventricle resulting independently of coronary disease and hypertension. Development of clinical manifestation of diabetic cardiomyopathy may require a few years. Diastolic dysfunction represents the earliest preclinical manifestations of illness that precedes systolic dysfunction which may later develop into symptomatic heart failure. Echocardiographic determination of the three various stages of diabetic cardiomyopathy is possible: 1st early stage: - finding without structural changes (normal LV dimensions), while cardiac dysfunction can only be detected by using some more sensitive methods such as tissue Doppler. 2nd middle stage: is characterized by slight changes in the size and thickness of the LV walls, but with significant changes to the diastolic and systolic function which may be determined by conventional echocardiography. 3rd late stage: includes significant structural and functional changes.

Conclusion — the echocardiography is a diagnostic method that might be justified for each newly identified diabetic patients, while a recommendation for frequent control checkups would depend on the findings themselves but also on the associated risk factors. However, the cost-benefit of such an approach is not justified for the time being, while the echocardiography is currently used for all diabetics thought to suffer from cardiovascular disease as well as those who possess additional risk factors.

Ehokardiografija u plućnih bolesnika

Echocardiography in pulmonary patients

Ivo Darko Gabrić

Clinical Hospital "Sestre milosrdnice", Zagreb

U bolesnika s kroničnom plućnom bolesti i/ili zatajenjem desnog srca ehokardiografija omogućuje procjenu plućne hipertenzije, veličine i sistoličke funkcije desnog ventrikula (DV) te potvrdu kliničke dijagnoze plućnog srca. Kod bolesnika s primarnom plućnom hipertenzijom ehokadiografijom se isključuju drugi uzroci plućne hipertenzije kao što su atrijski septalni defekt ili mitralna regurgitacija, a serijskim određivanjem plućne hipertenzije može se pratiti učinkovitosti terapije. Kod manjeg dijela bolesnika s plućnom embolijom može se prikazati ostatak tromba koji potiče ili prolazi kroz desno srce, dok je ipak veća je korist u procijeni indirektnih znakova plućne embolije kao što su povišen tlak u plućnoj arteriji, akutno tlačno opterećenja desnog ventrikula, dilatacija i disfunkcija DV te trikuspidna regurgitacija (TR).

In patients with chronic pulmonary disease and/or failure of the right heart, echocardiography allows assessment of pulmonary hypertension, the size and systolic function of the right ventricle (RV) and confirmation of the clinical diagnosis of the pulmonary heart. For patients with primary pulmonary hypertension, the echocardiography excludes other causes of pulmonary hypertension such as atrial septal defects or mitral regurgitation, and serial determination of pulmonary hypertension allows assessment of treatment efficacy. In a smaller percentage of patients with pulmonary embolism, the remaining thrombus may be indicated which affects or passes through the right heart, whereas the greater use in assessment of indirect signs of pulmonary embolisms such as increased blood pressure in the pulmonary artery, acute pressure exerting pressure on

Kronična plućna hipertenzija bilo da je nastala kao posljedica primarne plućne bolesti, zbog ponavljajućih plućnih embolija ili se radi o primarnoj plućnoj hipertenziji rezultira grupom kliničkih simptoma i znakova koje nazivamo plućno srce ili cor pulmonale. Osnovni patofiziološki mehanizam tog kliničkog sindroma je kronično tlačno opterećenje DV pri ejekciji u plućni krvotok povišene rezistencije. U početku nastaje kompenzatorna hipertrofija DV uz očuvanu sistoličku funkciju, s vremenom se pogoršava kontraktilnost uz dilataciju DV te nastaje umjerena do teška TR s posljedičnom dilatacijom desnog atrija (DA). Akutna plućna hipertenzija nastaje u masivna plućnoj emboliji kada dolazi do naglo nastale visoke plućne vaskularne rezistencije i razvoja akutnog plućnog srca.

Najpouzdaniji način ehokardiografske procjene plućne hipertenzije je putem mjerenja maksimalne brzine mlaza TR koja nastaje kao posljedica razlike sistoličkog tlaka DV i DA. Tlak u plućnoj arteriji (AP) se dobije uz pomoć Bernullijeve jednadžbe: $TAP=4(vTR)^2+TDA$. Za tu metodu potreban je nalaz trikuspidne regurgitacije što je rijetko ograničavajući faktor budući da se ona u određenom stupnju nalazi u 90% zdravih pojedinaca. Tlak u DA procjenjuje se pak prema veličini donje šuplje vene i stupnju njenog respiratornog kolapsa. Dijastolički tlak u AP se pak može procijeniti putem doplerske krivulje pulmonalne regurgitacije odnosno njene brzine na kraju dijastole nastale kao posljedice gradijenta tlaka između AP i DV.

Druga, iako manje precizna metoda procjene stupnja plućne hipertenzije je analiza doplerske krivulje brzine protoka kroz pulmonalno ušće (PV AT), pri čemu je u višoj plućnoj hipertenziji odnosno rezistenciji vrijeme postizanja maksimalne brzine ejekcije kraće odnosno krivulja poprima strmiji oblik (PV AT < 100 ms). Metoda je ograničena jer se mjeri vrlo mali period vremena tako da su česte greške u procjeni.

Ostali indirektni znakovi plućne hipertenzije ukazuju na njeno prisustvo, ali ne i na težinu. To je u prvom redu hipertrofija i dilatacija DV za čiju je procjenu najbolji subkostalni prikaz. U određenim slučajevima DV poprima oblik slova D u prikazu iz parasternalne kratke osi a i.v. septum se giba paradoksno. Najracionalnije objašnjenje za tu pojavu je da se septum giba prema centru mase srca tijekom sistole, a uz hipertrofični desni ventrikul centar mase pomiče se anteriorno te se septum giba prema sredini desnog ventrikula umjesto prema lijevom. Jednodimenzijskim prikazom (M-mod) pulmonalne valvule se pri plućnoj hipertenziji nalazi smanjena amplituda "a" vala uz ranije zatvaranje valvule (u sredini sistole). Iako visoko specifičan (>90%) taj znak je nisko osjetljiv (30-60%), ako istovremeno gledamo protok kroz pulmonalno ušće na dopler krivulji protoka nalazi se nagli pad protoka u sredini sistole. Još jedan znak plućne hipertenzije je produljeno vrijeme izovolumne relaksacije DV koje se može izračunati mjerenjem intervala između zatvaranja pulmonalnog i otvaranja trikuspidnog zalistka (na jednodimenzijskom M-modu ili putem doplera).

U bolesnika s akutnom plućnom hipertenzijom kao posljedicom plućne embolije može nastati sistolička disfunkcija DV sa sekundarnom dilatacijom kao kompenzator-

the right ventricle, dilatation and dysfunction of the RV and tricuspid regurgitation (TR).

Chronic pulmonary hypertension whether occurring as a result of primary pulmonary disease due to recurring pulmonary embolism or whether it relates to primary pulmonary hypertension results in a group of clinical symptoms and signs which we call pulmonary heart or cor pulmonale. The basic pathophysiological mechanism of this clinical syndrome is chronic pressure to the RV during ejection to the pulmonary bloodstream with increased resistance. At first, there occurs compensating hypertrophy of the RV with preserved systolic functioning, but with time also deterioration of contractility along with dilatation of the RV and occurrence of mild to severe TR resulting in dilatation of the right atrium (RA). Acute pulmonary hypertension occurs in massive pulmonary embolism with the sudden occurrence of high pulmonary vascular resistance and development of acute pulmonary heart.

The most reliable manner of echocardiographic assessment for pulmonary hypertension is by measuring maximal jet speed of the TR occurring as a result of the difference in systolic pressure RV and RA. The pressure in the pulmonary artery (PAP) is gained with the assistance of Bernouilli's equation: $PAP=4(vTR)^2+RAP$. This method requires a tricuspid regurgitation finding which is rarely limiting factor as it at a certain stage is to be found in 90% of healthy patients. The pressure in the RA is determined however on the size of the inferior vena cava and the level of its respiratory collapse. Diastolic pressure in the pulmonary artery however may be determined by using a Doppler graph of the pulmonary regurgitation and its speed at the end of the diastole occurring as a result of the pressure gradient between pulmonary artery and RV.

Secondly, even though a less precise method of determining the condition of pulmonary hypertension is the analysis of using the Doppler curve to measure the speed of flow through the pulmonary valve (PV AT), whereby in more intensive pulmonary hypertension and resistance, the time of achieving the maximum speed of ejection is shorter and the curve gains a steeper shape (PV AT < 100 ms). The method is limited since it measures a very small period of time leading to frequent errors in assessments.

Other indirect signs of pulmonary hypertension suggest its presence, but not it seriousness. This is foremost hypertrophy and dilatation of the RV with a best assessment made on the basis of the subcostal view. In certain situations, the RV gains the shape of the letter D in the parasternal short axis view whereas the i.v. septum moves paradoxically. The most rational explanation for this phenomenon is that the septum moves towards the heart mass centre during systole, and along with hypertrophic right ventricular mass centre it moves anteriorly with the septum moving towards the middle of the right ventricle instead to the left. The one-dimensional view (M-mode) of the pulmonary valve during pulmonary hypertension suggests reduced amplitude for the "a" wave with earlier closing of the valve (in the middle of the systole). Even though highly specific (>90%), this sign is not very sensitive (30-60%), if we take into consideration at the same time the flow

nim mehanizmom kako bi se održao udarni volumen. Dilatacija DV dovodi do trikuspidne regurgitacije zbog zbog dilatacije prstena i promjene položaja papilarnih mišića. To dodatno volumno opterećenje dovodi do daljnje dilatacije desnog ventrikula i dodatne plućne hipertenzije. Dilatacija desnog atrija nastaje kao posljedica i tlačnog i volumnog opterećenja desnog atrija.

Iako je kod plućnih bolesnika često otežan prikaz srčanih struktura zbog neadekvatnog ehokardiografskog prozora uslijed hiperinflacije pluća ehokardiografija je i dalje nezamjenjiva metoda u neinvazivnoj procjeni plućne hipertenzije i funkcije desnog srca.

Received: 17th Mar 2008
E-mail: idgabric@kbsm.hr

through the pulmonary mouth on the Doppler curve for flow the findings suggest a sudden fall in flow in the middle systole. Another sign of pulmonary hypertension is an extended time of iso volume relaxation of the RV which can be calculated by measuring the interval between the closing of the pulmonary and opening of the tricuspid valve (on the one-dimensional M-mode or by using the Doppler).

In patients with acute pulmonary hypertension resulting from pulmonary embolism there may occur a systolic dysfunction of the RV with secondary dilatation as a compensatory mechanism in order to maintain the stroke volume. Dilatation of the RV leads to tricuspid regurgitation due to dilatation of the ring and change in the position of the papillary muscles. This additional volume preload leads to further dilatation of the right ventricle and additional pulmonary hypertension. Dilatation of the right atrium occurs as a result of pressure and volume preload of the right atrium.

Even though lung patients often prove more difficult in showing heart structures due to an inadequate echocardiographic window caused by hyperinflation of the lungs, the echocardiograph continues to be an irreplaceable method in non-invasive assessment of pulmonary hypertension and the functioning of the right heart.

Ehokardiografska evaluacija atrijske morfologije i funkcije u aritmijama

Echocardiographic assessment of atrial anatomy and function in arrhythmias

Anton Šmalcelj

Clinical Hospital Centre Zagreb, Zagreb

Predodžbe kliničara o obliku, volumenu i funkciji oba atrija su često nedorečene. Premda je korelacija standardne M-mode dimenzija i volumena lijevog atrija u parasternalnim prikazima obično zadovoljavajuća, pogrešna je kvantifikacija trodimenzionalne strukture asimetrično proširenih, ili deformiranih atrija jednom jedinom veličinom. Izložene su temeljne značajke anatomije atrija uz pregled 2-D formula za računanje njihovog volumena.

Ukazuje se na moguće pogreške analize funkcije lijevog atrija Doppler prikazom transmitralnog dijastoličkog protoka kao i na pokušaje da se kvantificira do sada slabo istražena funkcija desnog atrija. Navode se podaci za Doppler analizu funkcije obje aurikule.

Ehokardiografska analiza kontrakcije i gibanja stijenke atrija je korisna dopuna elektrokardiografiji u prepoznavanju atrijskih aritmija. Analiza veličine, oblika i funkcije atrija je korisna za procjenu rizika atrijskih aritmija, osobito atrijske fibrilacije i undulacije. Važna je i pri izboru strategije liječenja. Transezofagijska ehokardiografija s prikazom lijeve aurikule je bitna u procjeni rizika tromboembolijskih komplikacija.

Clinician's perceptions on atrial shapes, volumes and functions are often vague. In spite of satisfactory correlation between left atrial M-mode dimension and its volume, the quantification of three dimensional structures of asymmetrically dilated and deformed atria by a single measurement value is erroneous. The key features of atrial anatomy and an overview of 2-D equations for volume calculations are reviewed.

Possible pitfalls in left atrial function analysis by transmitral Doppler flow are highlighted, as well as the attempts aiming at the quantification of still poorly defined right atrial function. The data on function of both atrial appendages are reviewed.

Echocardiographic analysis of atrial contraction and wall motion is a useful tool in recognizing atrial arrhythmias, adjunctive to electrocardiography. The analysis of atrial size, shape and function is useful for the risk assessment in various atrial arrhythmias and in atrial fibrillation and flutter in particular. It is also important for the choice of therapy strategy. Transesophageal echocardiography