Sažetak sa skupa | Meeting abastract

Desnostrani srčani zalisci — morfološka i funkcijska procjena

Right-sided heart valves — morphological and functional assessment

Danijel Planinc*

Klinički bolnički centar "Sestre Milosrdnice", Zagreb, Hrvatska

University Hospital Centre "Sestre milosrdnice", Zagreb, Croatia

Trikuspidna valvula

Trikuspidni aparat sastoji se od trikuspidne valvule (TV), prstena, kordi, papilarnih mišića, desnog atrija i ventrikula. Disfunkcija TV može biti primarna (morfološki abnormalna valvula) ili sekundarna (valvulni aparat je morfološki uredan). Primarni patološki procesi dovode do stenoze i/ili regurtgitacije, a sekundarni, koji su najčešće posljedica miokardne i/ili valvulne bolesti lijevog srca, gotovo isključivo do trikuspidne regurgitacije (TR). Minimalna do blaga TR (fiziološka) nalazi se u oko 50-65% zdravih odraslih osoba. Fiziološka regurgitacija u pravilu ima male brzine što ukazuje na normalan tlak u DV.

Ehokardiografija je referentni dijagnostički standard za bolesti TV. Budući su anatomija i pokreti TV vrlo složeni, jednodimenzijska ehokardiografija ima u tome malu ulogu. Etiologija i stupanj bolesti procjenjuju se na temelju nalaza 2-D ehokardiografije s pulzirajućim, kontinuiranim te obojanim Dopplerom. Bitan je prikaz anatomije i dinamike, primjena kontinuiranog Dopplera za mjerenje brzina utoka kroz stenotičku valvulu te obojanog Dopplera za prikaz regurgitacije. Utvrđivanje etiologije zahtijeva pažljivu evaluaciju morfologije i pokretljivosti listića, dimenzija prstena, morfologije subvalvulnog aparata te veličine i funkcije DV. Septalni i prednji listić mogu se dobro prikazati u

Tricuspid valve

Tricuspid apparatus consists of the tricuspid valve (TV), a ring, chorda, papillary muscle, right atrium and ventricles. TV dysfunction may be primary (morphologically abnormal valve) or secondary (valvular apparatus is morphologically normal). Primary pathologic processes lead to stenosis and/or regurgitation, while the secondary processes, which are often the consequence of myocardial and/or valvular left heart disease, almost exclusively to tricuspid regurgitation (TR). Minimal to mild TR (physiological) is found in about 50-65% of healthy adults. Physiological regurgitation usually has low velocities, which indicates a normal pressure in the RV.

Echocardiography is the reference diagnostic standard for the TV diseases. Since the TV anatomy and movements are very complex, one-dimensional echocardiography has a small role in it. The etiology and degree of the disease are assessed on the basis of the findings of 2-D echocardiography with pulsating, continuous, and colored Doppler. The image of the anatomy and dynamics, the use of the continuous Doppler for the measurement of the velocities of inflow through the stenotic valve and colored Doppler for imaging regurgitation are important. Determining the etiology requires a careful evaluation of morphology and mobility of the leaflets, ring dimensions, morphology of subvalvular apparatus and the RV size and function. The sep-

parasternalnoj kratkoj osi na bazi srca, u subkostalnoj kao i u apikalnoj poziciji 4 šupljine kada se također vidi odnos s mitralnom valvulom. Parasternalno, iz prikaza ulaznog dijela DV vrlo dobro se vide stražnji i prednji listić. Posljednih nekoliko godina sve se više, posebice u velikim kardiokirurškim centrima, u evaluaciji TV primjenjuje 3-D ehokardiografija. Brzina ranog dijastoličkog transtrikuspidnog protoka normalno je manje od 1,0 m/sec, srednji dijastolički PG obično manji od 2 mmHg.

Glavno obilježje trikuspidne stenoze (vrlo je rijetka i gotovo uvijek uzrokovana reumatskom vrućicom) je mehanička opstrukcija utoku krvi u DV tijekom dijastole. Evaluacija Doplerom slična je evaluaciji mitralne stenoze. Stenoza se smatra uskom kada je srednji dijastolički gradijent veći od 7 mmHg, a T1/2 duže od 190 ms, odnosno površina ušća manja od 1,0 cm^2.

TR može biti uzrokovna primarnom bolešću valvule (npr. prolaps u sklopu miksomatoznog sy., ruptura korde, endokarditis) ili nastaje sekundarno (funkcijska TR) zbog dilatacije trikuspidnog prstena (posljedica plućne hipertenzije bilo kojeg uzroka) iako je valvula morfološki uredna. Sekundarna TR je znatno češće (75-80% bolesnika). Kada sistolički tlak u plućnoj arteriji poraste na više od 55 mmHg, javlja se određeni stupanj TR; ako je tlak niži od 40 mmHg prisustvo više nego blage TR upućuje na strukturne abnormnalnosti listića ili subvalvulnog aparata. TR se javlja u 8-35% bolesnika sa stečenim valvulnim bolestima, posebice reumatskim, rjeđe degenerativnim, znatno češće uz bolesti mitralne nego aortne valvule. Akutna teška TR javlja se uglavnom kao sekundarna funkcijska regurgitacija, najčešće u sklopu akutnog tlačnog opterećenja DV (plućna embolija) ili rjeđe volumskog opterećenja (akutna ruptura iv septuma kao komplikacija infarkta miokarda) s posljedičnim akutnim zatajivanjem i dilatacijom DV. TR se kvalitativno procjenjuje kao minimalna, blaga, umjerena, ili jaka (teška) regurgitaciju. Pomoću obojanog Dopplera gradira se ljestvicom 1-4+, ovisno o proširenosti sistoličkog protoka u desnom atriju. Trajanje duže od l00 msec, označavaju jaku TR kao i vena kontrakta ≥0,65 cm. Gusti se signal vidi u jakoj TR, ali treba imati na umu da je brzina odraz gradijenta tlaka između desnog ventrikula i atrija, a ne stupnja regurgitacije. Efektivna površina regurgitirajućeg otvora (ERO), kao i regurgituirajući volumen izračunavaju se metodom PISA kao i kod mitralne regurgitacije. ERO ≥40 mm^2 ukazuje na jaku TR, kao i regurgitirajući volumen od 45 ml (za razliku od 60ml u mitralnoj regurgitaciji). Osim toga, pokazatelji teške TR su: 1. površina mlaza obojenim doplerom veća od 30% površine desnog atrija, 2. gusti signal kontinuiranog doplera, 3. dilatacija prstena ≥4cm ili nepotpuna koaptacija kuspisa, 4. konkavni kasnosistolički oblik signala kontinuiranog doplera, 5. brzina utoka krvi ≥1m/s, 6. dilatacija desnog atrija i ventrikula i 7. sistolički reverzni protok u donju šuplju i hepatalne vene. Volumsko opterećenje DV povezano je s abnormalnim gibanjem interventrikulskog septuma koji se u sistoli pomiče prema sredini DV, a u dijastoli brzo vraća straga, pa lijevi ventrikul u dijastoli poprima oblik slova "D". Brzina mlaza TR utvrđena Dopplerom koristi se za procjenu sistoličkog tlaka DV, koji u odsustvu opstrukcije izlaznog trakta odgovara sistoličkom tlaku u plućnoj arteriji.

Rijetki uzroci TR su: prirođeni prolaps (npr. u Marfanovom sindromu), displazija TV, Ebsteinova anomalija, karcital and anterior leaflet may well be shown in the short parasternal axis on the heart base, in subcostal and in the apical position of 4 chambers when the relation with mitral valve is also shown. Parasternally, from the image of the entry part of the RV we can well see the posterior and anterior leaflet. During the past few years, 3-D echocardiography has been increasingly used in the evaluation of TV especially in high volume surgical centers. The velocity of the early diastolic transtricuspid flow is normally below 1.0 m/sec, while the mean diastolic PG is usually below 2 mmHg.

The main characteristic of tricuspid stenosis (it is very rare and almost always caused by rheumatic fever) is a mechanical obstruction to the blood flow into the RV during diastole. Doppler evaluation is similar to the mitral stenosis evaluation. The stenosis is considered to be narrow when the mean diastolic gradient is greater than 7 mm Hg, and T1/2 longer than 190 ms, or the orifice area is less than 1.0 cm^2.

TR can be caused by primary valve disease (e.g. prolapse within the myxomatous sy., chorda rupture, endocarditis), or occurs as secondary (functional TR) due to the tricuspid ring dilatation (a consequence of pulmonary hypertension from any cause), although the valve morphology is normal. Secondary TR is much more frequent (75-80% of patients). When the systolic pressure in the pulmonary artery rises to more than 55 mmHg, a certain degree of TR occurs; if the pressure is lower than 40 mmHg, the presence of more than mild TR indicates the structural abnormalities of the leaflets or subvalvular apparatus. TR occurs in 8-35% of patients with acquired valvular diseases, especially rheumatic, more rarely degenerative diseases, more frequently in conjunction with mitral than with aortic valve disease. Acute severe TR mainly occurs as a secondary functional regurgitation, most commonly within the RV acute pressure load (pulmonary embolism) or less frequently within volumetric load (acute rupture of the iv septum as a complication of the myocardial infarction) with consequential acute RV failure and RV dilation. TR is qualitatively assessed as minimal, mild, moderate, or severe regurgitation. By using the colored Doppler it is graded by the scale 1-4+, depending on the extent of systolic flow in the right atrium. The duration longer than 100 msec means severe TR as well as the vena contracta ≥0.65 cm. The strong signal is seen in severe TR, but we should bear in mind that the velocity is a reflection of the pressure gradient between the right ventricle and atrium, not the degree of regurgitation. The effective regurgitant orifice area (ERO) and the regurgitant volume are calculated by using the PISA method the same as in case of mitral regurgitation. ERO ≥40 mm^2 indicates a strong TR and regurgitant volume of 45 ml (unlike 60ml in mitral regurgitation). In addition, the indicators of severe TR are: 1. the color Doppler jet area greater than 30% of the right atrial area, 2. continuous Doppler dense signal, 3. ring dilatation ≥4cm or incomplete cusp coaptation, 4. late systolic concave configuration of the continuous Doppler signal, 5. blood flow velocity ≥1m/s, 6. right atrial and ventricular dilatation and 7. systolic and reversal flow in the inferior vena cava and hepatic veins. RV volumetric load is associated with abnormal motion of the interventricular septum which in systole moves towards the RV center, and in diastole it quickly returns backwards, so the left ventricle in diastole takes the shape of the letter "D". TR jet velocity determined by Doppler is used for the assessment of the RV systolic pressure,

noidna bolest srca, trauma trikuspidne valvule, tumori, endokarditis, endokardna fibroelastoza, i jatrogena oštećenja. U odsutnosti plućne hipertenzije TR se uglavnom dobro podnosi.

Pulmonalna valvula

Pulmonalna valvula ima tri listića i nalazi se na spoju izlaznog trakta DV i trunkusa plućne arterije. Abnormalna pulmonalna valvula može biti akomisuralna s izraženim kupolastim izbočenjem u sistoli i ekscentričnim otvorom, unikomisuralna s jednom asimetričnom komisurom, bikuspisna sa spojenim komisurama ili displastična, s jače zadebljanim i deformiranim listićima. Stenoza pulmonalne valvule (PS) je u oko 95% bolesnika prirođena, a u oko 80% se javlja izolirano. Može biti valvulna, supravalvulna (periferna) ili subvalvulna (infundibularna). 2-D ehokardiografijom mogu se prikazati zadebljani, fibrozni ili kalcificirani listići ograničenih pokreta, poststenotička dilatacija plućne arterije ili njenih ogranaka, kao i hipertrofija i/ili dilatacija DV zbog tlačnog opterećenja. Normalno maksimalna brzina krvi kroz pulmonalno ušće iznosi 1,0 m/s. Supravalvulna PS može biti posljedica stenoze trunkusa plućne arterije ili jedne od grana distalno od zaliska, kada se ehokardiografijom mogu vidjeti jedan ili više pregradnih tračaka u arteriji. Povećanje brzine krvi primjenom Dopplera nalazi se distalno, a ne u razini same valvule. Subvalvulna PS je većinom prirođena, rijetko izolirana, te obično udružena s valvulnom stenozom, VSD, tetralogijom Fallot, transpozicijom velikih arterija ili rijetko hipertrofijskom kardiomiopatijom. Mišićni tračak ili suženje subvalvulnog područja, obično bez poststenotičke dilatacije vidi se na 2-D ehokardiogramu. Povećanje brzine otkriva se primjenom Dopplera u izlaznom traktu DV, tj. ispod razine pulmonalne valvule. Težina PS procjenjuje se mjerenjem maksimalnih brzina transpulmonalnog protoka kontinuiranim Dopplerom odnosno izračunavanjem transvalvulnog gradijenta tlaka: maksimalni gradijent jednak ili veći od 60mmHg označava tešku PS. Sistolički tlak u desnoj klijetki može se izračunati na temelju brzine, mlaza TR koristeći modificiranu Bernoulijevu jednadžbu.

Trivijalni ili blagi stupanj pulmonalne regurgitacije (PR) može se utvrditi Dopplerom u većine zdravih osoba. Patološka PR u odraslih obično je posljedica ranijih intervencija zbog prirođenih srčanih grešaka (npr. tetralogije Fallot). U bolesnika s dugotrajnom teškom PR može doći do progresivne dilatacije DV s redukcijom sistoličke funkcije i nemogućnošću povećanja minutnog volumena u opterećenju te razvoja kongestivnog zatajivanja srca. Patološka PR razlikuje se od normalnog regurgitirajućeg mlaza duljim trajanjem (holodijastolička) i širim mlazom. PR može također biti uzrokovana reumatskom ili karcinoidnom bolešću, traumom, endokarditisom, plućnom hipertenzijom ili idiopatskom dilatacijom plućne arterije i prstena. Stupanj PR se ehodoplerkardiografski procjenjuje na temelju kvalitativne skale zasnovane uglavnom na jačini signala kontinuiranog Dopplera i veličini mlaza dobivenog obojenim Dopplerom. Važno je detaljno analizirati valvulu jer se relativno često i teška PR može previdjeti. Jednodimenzijska i dvodimenzijska ehokardiografija ne mogu otkriti direktno PR, ali mogu ukazati na osnovni uzrok., otkrivajući dilataciju DV, dilataciju plućne arterije, abnormalne pokrete in-

which in the absence of obstruction of the outflow tract equals the systolic pressure in the pulmonary artery.

Some rare causes of TR include: congenital prolapse (e.g. Marfan syndrome), TV dysplasia, Ebstein's anomaly, carcinoid heart disease, tricuspid valve trauma, tumors, endocarditis, endocardial fibroelastosis and iatrogenic damage. In the absence of pulmonary hypertension, TR is generally well tolerated.

Pulmonic valve

Pulmonic valve has three leaflets and is positioned at the junction of the RV outflow tract and truncus of the pulmonary artery. Abnormal pulmonic valve can be acommissural with prominent systolic doming of the valve cusps and an eccentric orifice, unicommissural with a single asymmetric commissure, bicuspid with fused commissures or dysplastic with severely thickened and deformed valve cusps. Pulmonic valve stenosis (PS) is congenital in around 95% of patients and it occurs in isolation in around 80% of patients. It can be valvular, supravalvular (peripheral) or subvalvular (infundibular). 2-D echocardiography may show thickened, fibrotic or calcified leaflets with limited motions, poststenotic dilatation of the pulmonary artery or its branches, as well as RV hypertrophy and/or dilatation due to pressure load. Normally, maximal blood velocity through the pulmonary orifice is 1.0 m/s. Supravalvular PS may be the consequence of pulmonary artery truncus stenosis or one of the branches distally from the valves, when echocardiography can show one or more septal track marks in arteries. The increase in the blood velocity by using Doppler is located distally, not at the level of the valve itself. Subvalvular PS is mostly congenital, rarely isolated, and usually in conjunction with valve stenosis, VSD, tetralogy of Fallot, transposition of large arteries, or rarely hypertrophic cardiomyopathy. Muscle track mark or narrowing of subvalvular area, usually without poststenotic dilatation is seen on 2-D echocardiogram. Increase in the velocity is detected by using Doppler in the RV outflow tract, that is, below the level of the pulmonary valve. The PS severity is assessed by measuring maximal velocities of transpulmonary flow by continuous Doppler or calculation of transvalvular pressure gradient: maximal gradient equaling or exceeding 60 mmHg indicates severe PS. Systolic blood pressure in the right ventricle can be calculated on the basis of velocity, TR jet by using the modified Bernoulli's equation.

Trivial or mild degree of pulmonary regurgitation (PR) can be determined by Doppler in most healthy persons. Pathological PR in adults is usually the consequence of earlier interventions due to congenital heart defects (e.g. tetralogy of Fallot). In patients with severe long-time PR, RV progressive dilatation can occur with reduction of systolic function and inability of increasing minute volume in the condition of load and development of congestive heart failure. Pathological PR differs from the normal regurgitant jet with longer duration (holodiastolic) and wider jet. PR can also be caused by rheumatic or carcinoid disease, trauma, endocarditis, pulmonary hypertension or idiopathic pulmonary artery and ring dilatation. The PR degree is echodopplercardiographically assessed on the basis of a qualitative scale mainly based on continuous Doppler sig-

terventrikulskog septuma, ev. vegetacije, debele imobilne listiće u reumatskoj bolesti ili karcinoidu, ili pak odsustvo listića u prirođenoj malformaciji valvule. Pokazatelji težine PR su: veliki retrogradni mlaz krvi obojanim Dopplerom, velika širina mlaza na razini valvule, širenje regurgitirajućeg mlaza krvi ispod donjeg dijela infundibularne regije DV, gusti i pojačani signal te povećani nagib signala (skraćeno vrijeme deceleracije) kao i povećana brzina sistoličkog protoka kroz pulmonalno ušće.

Received: 12th Oct 2011

*Address for correspondence: Klinički bolnički centar "Sestre milosrdnice", Vinogradska cesta 29, HR-10000 Zagreb, Croatia;

E-mail: danijelplaninc@gmail.com

nal strength and size of the jet obtained by colored Doppler. It is important to analyze the valve in details because even severe PR can relatively frequently be predicted. One-dimensional and two-dimensional echocardiography cannot directly detect PR, but can indicate the underlying cause thereby detecting the RV dilatation, pulmonary artery dilatation, abnormal movements of interventricular septum, potential vegetation, thick immobile leaflets in rheumatic disease or carcinoid, or the lack of leaflets in the congenital valve malformation. Indicators of PR severity are: large retrograde blood jet by colored Doppler, large jet width at the level of the valve, expansion of regurgitant blood jet below the lower part of RV infundibular region, dense and increased signal and increased signal slope (shortened deceleration time) and the increased velocity of systolic flow through pulmonary orifice.