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Finnish Cardiac Society

39th Progress Report Meeting

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Kittilä

Contents

History of the Progress Report Meetings 70

Young Investigators Award Competition abstracts
Abstracts in the order of presentation
39th Progress Report Meeting - Young Investigators Award Competition 71
Competition is supported by unrestricted educational grant from
Boehringer Ingelheim.

CHA2DS2VASc score and the thromboembolic risk of electrical cardioversion of acute atrial fibrillation.
(Clinical research).
Toni Grönberg (BM), Faculty of Medicine, University of Turku 71

Catheter ablation versus antiarrhythmic medication as first line therapy in paroxysmal atrial fibrillation; on treatment analysis of the MANTRA-PAF data.
(Clinical research).
Antti Hakalahti (M.D.), Department of Cardiology, University of Oulu 72

Gene transfer using vammin induces robust angiogenesis and improves left ventricular function.
(Basic science).
Paavo Halonen (BMed), A.I. Virtanen -institute, University of Eastern Finland 73

Comparison of 18F-fluoromethylcholine and 18F-fluorodeoxyglucose for imaging of atherosclerotic plaque inflammation in mice.
(Basic science).
Sanna Hellberg (M.Sc.), Turku PET Centre, University of Turku 74

Determinants of cardiovascular autonomic function in coronary artery disease patients with and without type 2 diabetes.
(Clinical research).
Jaana Karjalainen (Doctoral student, MSc), Institute of clinical medicine, University of Oulu 75

Detection of concurrent atrial ischemia with continuous monitoring of dynamic PR-segment changes in patients with acute myocardial infarction.
(Clinical research).
Pentti Korhonen (MD), TAYS Sydänkeskus Oy 76

The effects of vascular endothelial growth factor B and D -gene transfer on myocardial perfusion in healthy and ischemic porcine hearts.
(Basic science).
Jussi Nurro (Mr), A.I.V. -institute, University of Eastern Finland 77

Lymphangiogenesis in aortic valve stenosis - novel regulatory roles for valvular myofibroblasts and mast cells.
(Basic science).
Suvi Syväranta (MD), Wihuri Research Institute 78

Prevalence and long-term prognosis of fragmented QRS in standard 12-lead electrocardiogram in middle-aged subjects.
(Clinical research).
Henri Terho (Bachelor of Medicine), Department of Internal Medicine, University of Oulu 79

Acute effect of adjunctive breast cancer radiotherapy on right ventricular systolic and diastolic function.
(Clinical research).
Suvi Sirkku Tuohinen (MD), Heart Center Co, Tampere University Hospital 80

History of the Progress Report Meetings

Progress Report Meeting is organized by Finnish Cardiac Society to present opportunity for young investigators to report results of their studies. An important point is also training in presenting scientific papers to criticism of senior colleagues.

Boehringer Ingelheim has supported organizing the meeting from the beginning, 1975 by helping in practical matters and presenting grants to the best of speakers.

Winners of the Boehringer Ingelheim grants

From year 2007 onwards the competition has had two categories instead of 1st and 2nd prize. However, if less than three eligible abstracts has been received to either category, the organizers reserve the right to combine the categories.

Year	1st Prize	2nd Prize
1975	Erkki Pesonen	–
1976	Heikki Karppanen	Markku S. Nieminen
1977	Matti Halinen	Ulla Korhonen
1978	Ilkka Torstila	Markku S. Nieminen
1979	Olli Meretoja	Aila Rissanen
1980	Jorma Viikari	Jouko Jalonen
1981	Markku Kupari	Irma Koivula
1982	Heikki Huikuri	Markku Kupari
1983	Seppo Hietakorpi	Kari Niemelä
1984	Markku Laakso	Heikki Huikuri
1985	Jukka Räisänen	Kari Niemelä
1986	Pekka Koskinen	Juha Mustonen
1987	Kimmo Mattila	Silja Majahalme
1988	Heikki Tikkanen	Paula Rämö
1989	Hannu Näveri	Keijo Peuhkurinen
1990	Markku Mäkijärvi	Juhani Valkama
1991	Eero Mervaala	Paavo Uusimaa
1992	Eero Mervaala	Anne Remes
1993	Juha Hartikainen	Helena Kovanen
1994	Kai Kiilavuori	Juha Perkiömäki
1995	Sirkku Pikkujämsä	Pasi Tavi
1996	Jorma Kokkonen	Timo Mäkikallio
1997	Pekka Raatikainen	Marja Laitinen
1998	Marja Laitinen	Antti Ylitalo, 3rd Prize Timo Mäkikallio
1999	Mika Laine	Timo Mäkikallio
2000	Saila Vikman	Antti Kivelä
2001	Jari Tapanainen	Pertti Jääskeläinen
2002	Tuomas Rissanen	Markku Pentikäinen
2003	Juhani Junttila	Markus Leskinen
2004	Jere Paavola	Tuomas Rissanen
2005	Mikko Mäyränpää	Satu Helske
2006	Olli Tenhunen	Johan Lassus

Year	Basic Science category	Clinical Research category
2007	Satu Helske	Ville Kytö
2008	Mirella Hietaniemi	Minna Kylmälä
2009	Johanna Lähteenvuo o.s. Markkanen	Annukka Marjamaa
2010	1st Prize Jani Tikkanen 2nd Prize Riina Kandolin	
2011	Markku Lähteenvuo	Aapo Aro
2012	1st Prize Kirsi Kujala 2nd Prize Maija Bry	
2013	Suvi Syväranta	Toni Grönberg

CHA2DS2VASc score and the thromboembolic risk of electrical cardioversion of acute atrial fibrillation

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Aim

Perioperative anticoagulation is recommended during cardioversion of acute atrial fibrillation (AF) in patients with risk factors for stroke. However, these recommendations are based on a few small retrospective studies. Hence, our aim was to determine the real-life risk related to electrical cardioversion of acute AF. We also assessed if CHA2DS2VASc score predicts embolic complications in this setting.

Methods

During 2003-2010 3143 patients underwent 7660 cardioversions to acute (< 48 hours) AF in three hospitals. Embolic and bleeding complications were evaluated after 6508 successful electrical cardioversions. There were 2020 cases with and 4488 without periprocedural anticoagulation.

Results

The success rate of electrical cardioversions was 94.2 %. A total of 38 definite thromboembolic complications were noted within 30 days (mean 5 days) after successful electrical cardioversion and 32 of those were strokes. In addition, 5 transient ischemic attacks and two pulmonary embolisms occurred. Incidence of definite embolic events was significantly higher in cardioversions without perioperative anticoagulation (0.8 % vs. 0.1 %, p = 0.001). No major bleeding events were registered. CHA2DS2VASc score predicted (p < 0.0001) embolic events in patients without anticoagulation (Figure).

Conclusions

High CHA2DS2VASc is a strong predictor of embolic events when no anticoagulation is used. Periprocedural anticoagulation is important also in cardioversions of acute AF in patients with risk factors for stroke.

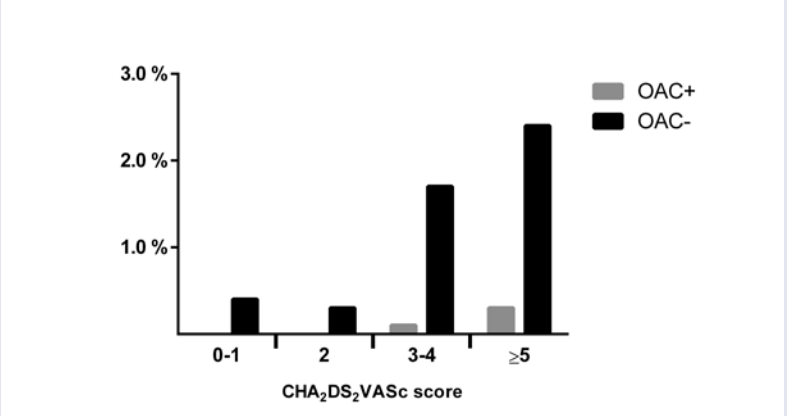

Figure.

Catheter ablation versus antiarrhythmic medication as first line therapy in paroxysmal atrial fibrillation; on treatment analysis of the MANTRA-PAF data

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Background

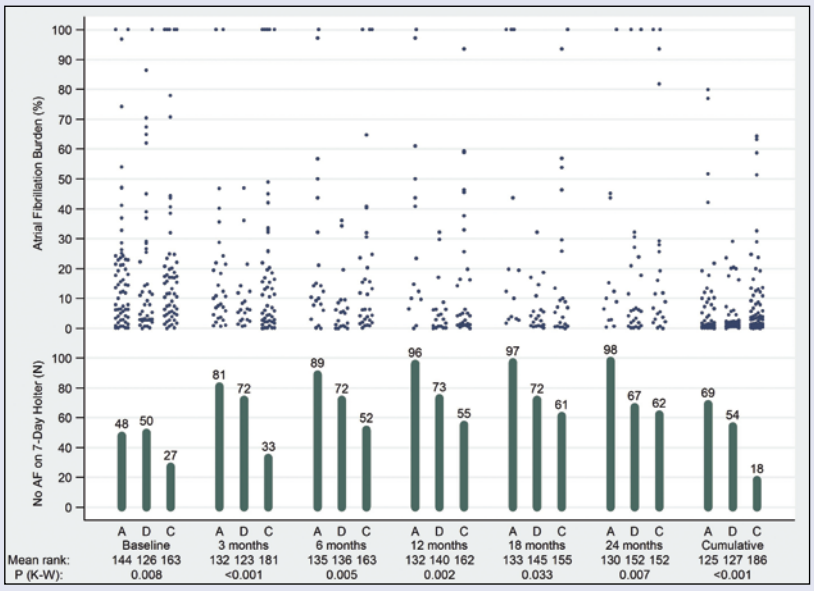
The MANTRA-PAF is the largest randomized trial comparing radiofrequency catheter ablation (RFA) and antiarrhythmic drug (AAD) therapy in treatment of paroxysmal atrial fibrillation (PAF). Intention-to-treat analysis of the data showed that at 24 months AF burden was significantly lower in the RFA than in the AAD group. Because many patients received both therapies, we provide here data on those who received only the prescribed treatment (on treatment analysis).

Methods

We randomly assigned 294 AAD naive patients with PAF to RFA (146 patients) or class IC or class III AAD therapy (148 patients). Follow-up included 7-day Holter recording at 3, 6, 12, 18, and 24 months. In the on treatment analysis the primary end points were AF burden (i.e., percentage of time in AF during the recording) at each Holter recording and cumulative AF burden and freedom of any AF lasting for more than 60 s. These data were compared also to those who had supplementary treatment with RFA or AAD (cross-over group).

Results

In the AAD group, 54 patients (36%) underwent supplementary ablation and in the RFA group 30 patients (21%) received antiarrhythmic medication after the 3 months blanking period (at 24 months 9% were on AAD). Eight patients were excluded from the analysis because they did not receive the index treatment. At 24 months, AF burden was significantly lower and more patients in the RFA than in AAD group and the cross-over group were free from any AF (89% vs. 73% vs. 74%, P<0.001). The cumulative AF burden was lowest in the RFA and highest in the cross-over group (P<0.001). There were no significant differences in the number of severe complications between the groups.



Conclusions

These data provide further support to the current guidelines indicating that RFA can be used as initial treatment in highly symptomatic and relatively healthy patients with paroxysmal AF. The results should not be extrapolated to broader population of patients with AF most of whom are elderly with comorbidities.

Gene transfer using vammin induces robust angiogenesis and improves left ventricular function

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Aim

Novel therapies are needed for patients who suffer from chronic myocardial ischemia but are not suitable for conventional treatments. Vammin is a snake venom derived vascular endothelial growth factor (VEGF) and a selective VEGF receptor-2 ligand. The purpose of this study was to determine whether an adenoviral vammin gene transfer induces angiogenesis or affects the perfusion or function of porcine myocardium during normoxia and chronic myocardial ischemia.

Methods

Gene transfers were done percutaneously with rising doses of adenoviral vammin (AdVammin) using AdCMV as a control. Angiography and positron emission tomography (PET) imaging were used to measure the effects on angiogenesis and myocardial perfusion, and cardiac ultrasound and modified Miles assay to measure tissue edema and permeability.

Myocardial ischemia was induced using a novel bottleneck stent model of chronic myocardial ischemia. Left ventricular function was analyzed before and after the gene transfer using LV cine angiography and by measuring cardiac output. Infarction area was also measured.

Results

AdVammin induced robust angiogenesis and tissue permeability also visible in the angiograms (arrowheads in fig.1) at day 6. Mean capillary size was increased by 429-737% when compared to that of the control group (429%, 737%, and 720% with doses of 1e9, 5e9 and 1e10 vp/ml, respectively) and relative tissue permeability was increased up to 3.34. In ischemic myocardium, left ventricular ejection fraction was increased from 43% to 51% six days after the gene transfer in AdVammin treated animals.

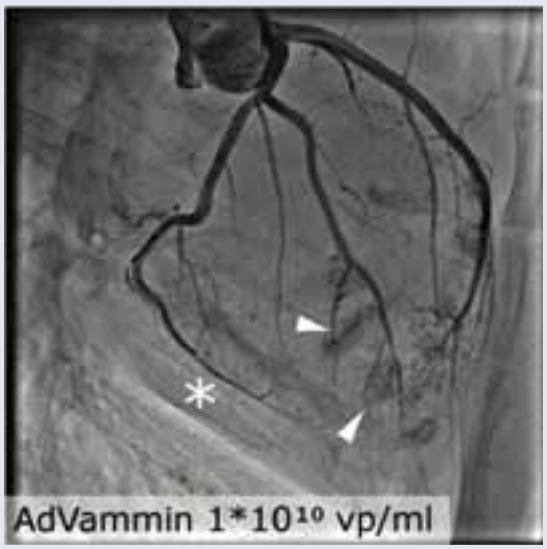

Figure 1.

Conclusions

In this study we have shown that vammin gene transfer induces robust angiogenesis in porcine myocardium as well as increases tissue permeability in a dose dependent manner. We have also demonstrated that the gene transfer improves left ventricular function and could thus be beneficial in treatment of chronic myocardial ischemia.

Comparison of 18F-fluoromethylcholine and 18F-fluorodeoxyglucose for imaging of atherosclerotic plaque inflammation in mice

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Aim

Molecular imaging of inflammation with positron emission tomography (PET) is a potential method to detect vulnerable atherosclerotic plaques. However, there is a need for more specific PET tracers than fluorine-18 labelled fluorodeoxyglucose (18F-FDG), a marker of glucose metabolism, which has a high physiological uptake in the myocardium. Therefore, we studied whether 18F-fluoromethylcholine (18F-FMCH), which is taken up in cells with high phospholipid metabolism, could be used for PET imaging of atherosclerotic plaque inflammation.

Methods

We studied 16 hypercholesterolemic and diabetic mice (IGF-II/LDLR-/-ApoB100/100) fed with high-fat diet for 4 months to induce atherosclerosis. The mice were injected with 18F-FMCH or 18F-FDG, and 20 or 90 minutes later, uptake of tracer was measured in the plaques and normal vessel wall by digital autoradiography of aortic sections. Tracer uptake was measured in the aorta, myocardium and blood by gamma counter. Mac-3 antibody was used to detect macrophages in plaques.

Results

The aortas of all mice showed large fibroatheroma-type plaques containing high density of macrophages. Autoradiography of aortic sections showed strong focal accumulation of both 18F-FMCH (Figure) and 18F-FDG in the atherosclerotic plaques (plaque-to-normal vessel wall ratios 2.7±0.3 vs. 2.4±0.4, p=0.06 between tracers). The highest tracer uptake, up to 4 times higher than in the normal vessel wall, was associated with high macrophage density. Aorta-to-blood ratios of 18F-FMCH and 18F-FDG were similar (4.6±2.2 vs. 3.2±0.8, p=0.13), but uptake of 18F-FMCH was 88 % lower in the myocardium (% of injected radioactivity /gram of tissue 8.2±3.1 vs. 68±28, p<0.001)

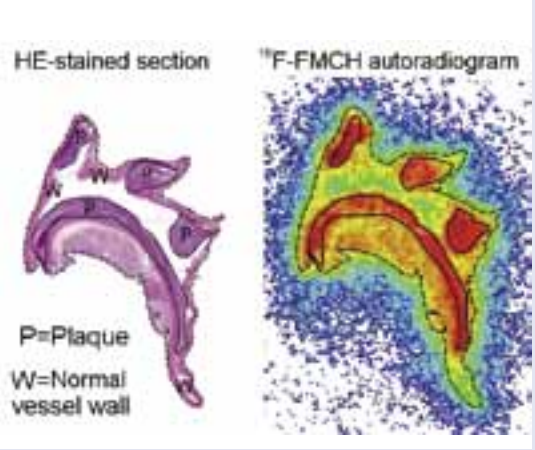

Figure.

Conclusions

The PET tracer 18F-FMCH is taken up in inflamed atherosclerotic plaques in the mouse aorta. The uptake of 18F-FMCH in plaques is comparable with that of 18F-FDG, but its lower uptake in the myocardium could be beneficial for imaging of inflammation in the coronary arteries.

Determinants of cardiovascular autonomic function in coronary artery disease patients with and without type 2 diabetes

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Aim

Cardiovascular autonomic dysfunction, which is a well-known risk factor of mortality, is a common finding among the patients with coronary artery disease (CAD) and diabetes. However, the reasons of autonomic dysfunction in CAD patients are not well known.

Methods

We examined the associations between autonomic function, assessed by three different methods, such as heart rate (HR) recovery after exercise, 24 hour HR variability (SDNN) and HR turbulence, and other potential factors, such as demographic characteristics, history of myocardial infarction and revascularization, medication, metabolic, inflammatory and coronary risk variables, echocardiographic parameters, exercise capacity, and the presence of type 2 diabetes (T2D) among 1060 CAD patients (age 67 ± 8 years, 69% males, 50 % patients with T2D).

Results

In univariate analysis, all three indexes of autonomic function were related to several demographic, metabolic, inflammatory and coronary risk markers, echocardiographic and exercise variables as well as features of T2D. In multiple linear regression model, exercise capacity ($R = 0.34$, $p < 0.001$) was the strongest predictor of HR recovery followed by age ($R = -0.24$, $p < 0.001$). SDNN also had the strongest relationship with exercise capacity ($R = 0.33$, $p < 0.001$) followed by presence of T2D ($R = 0.16$, $p < 0.001$). HR turbulence slope was most closely related to left ventricular ejection fraction ($R = 0.18$, $p < 0.001$) and exercise capacity ($R = 0.13$, $p = 0.001$)

Conclusions

Low exercise capacity is the most important determinants of impaired autonomic regulation of HR, suggesting to major importance of physical fitness in prevention of autonomic dysfunction and its clinical consequences in patients with CAD.

Detection of concurrent atrial ischemia with continuous monitoring of dynamic PR-segment changes in patients with acute myocardial infarction

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Aim

Despite its potential prognostic value concurrent atrial ischemia is often overlooked in patients with acute myocardial infarction (AMI). There is no study concerning the relationship between atrial coronary circulation and dynamic PR-segment changes in patients with AMI.

Methods

We evaluated dynamic PR-segment changes in 37 patients with AMI. In all patients ECG was monitored continuously up to 4 hours prior to the coronary angiogram. The PR-segment changes were analysed using a custom-made software, and changes in the PR-segment were correlated with the angiographic findings. The flow in the principal atrial coronary branches (i.e., sinoatrial nodal, atrioventricular nodal, left atrial circumflex artery) was assessed according to the location of the culprit lesion relative to the origin of the atrial branches.

Results

Among the study population flow in the principal atrial coronary branches was diminished in 14 (38%) patients. Dynamic changes in the PR-segment level (0.041 ± 0.018 mV vs. 0.024 ± 0.008 mV, P=0.005) and PR-segment area (2.911 ± 1.365 nVs vs. 1.644 ± 0.791 nVs, P=0.005) were significantly greater in patients with compromised than in those with normal atrial circulation. There were no statistical differences in the absolute values of the PR-segment level and area between the groups.

Conclusion

According to our data, monitoring of the dynamic changes in the PR-segment in patients with AMI reliably identified those with concurrent atrial ischemia. Given the potential prognostic value of atrial ischemia these data may have important clinical implications in evaluation of patient with AMI.

The effects of vascular endothelial growth factor B and D -gene transfer on myocardial perfusion in healthy and ischemic porcine hearts

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Aim

The aim of this study was to evaluate the effect of adenoviral (Ad) gene transfer of vascular endothelial growth factor (VEGF) B186 and DNA on perfusion and capillary growth in a healthy and in an ischemic porcine heart.

Methods

Positron emission tomography (PET) perfusion scan of the heart was done six days after an AdVEGF-B, AdVEGF-D or control AdCMV intramyocardial gene transfer to the posterolateral wall of the left ventricle. The ischemic animals had their coronary blood flow restricted in the left circumflex artery one week prior to the gene transfer. Myocardial capillary density, mean capillary area and total microvascular area were calculated from the histological samples taken six days after the gene transfer.

Results

Preliminary results show that in healthy porcine heart, both VEGF-B and VEGF-D increase average capillary size in the gene transfer area and decrease perfusion in the anterior myocardium when compared to the gene transfer area. This effect did not cause any significant differences between groups in the contractility of the heart measured by ejection fraction and cardiac output. In the ischemic animals VEGF-B increased capillary number in areas near the infarction site (border zone) but decreased capillary number in the myocardium with normal histology. VEGF-D increased capillary size and number in the border zone and the same trend was visible in the normal myocardium. Neither of these had detectable effect on myocardial perfusion.

Conclusions

VEGF-B and VEGF-D myocardial gene transfers seem to increase the myocardial perfusion in healthy hearts in the expense of decreased myocardial perfusion in the non-treaded area. This might be due to the bsteal" effect of enlarged capillaries in the treated myocardium. In the ischemia model, increased capillary size and number do not seem to be enough for effective increase in myocardial perfusion.

Lymphangiogenesis in aortic valve stenosis – novel regulatory roles for valvular myofibroblasts and mast cells

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Aim

Aortic valve stenosis (AVS) is an active atheroinflammatory disease. Angiogenesis and infiltration of inflammatory cells, among them mast cells, belong to its central pathological features. Lymphatic vessels are present in aortic valves, but the involvement of lymphangiogenic factors and their regulation in the pathogenesis of AVS have not been studied. Thus, our aim was to investigate mechanisms of lymphangiogenesis in AVS.

Methods

Lymphatic vessels were visualized with LYVE-1 staining in 20 control, 5 sclerotic, and 40 stenotic human aortic valves. Vascular endothelial growth factors (VEGFs) VEGF-C and VEGF-D, and their lymphangiogenic receptor VEGFR-3, and the angiogenic VEGFR-2 were analyzed by quantitative real-time PCR and immunohistochemistry. Cultured myofibroblasts derived from human stenotic aortic valves, and cultured human mast cells were used to study VEGF-C regulation, and VEGF-C and VEGF-A were quantified from cell culture media by enzyme immunoassays.

Results

Lymphatic vessels, VEGF-C, VEGF-D, VEGFR-3 and VEGFR-2 were all present in the aortic valves. In AVS, the number of lymphatic vessels and the expression of VEGF-D, VEGFR-3, and VEGFR-2 were increased. Moreover, the numbers of lymphatic vessels correlated positively with those of neovessels ($r=0.525$, $p=0.001$) and mast cells ($r=0.374$, $p=0.017$). Cultured valvular myofibroblasts produced VEGF-C, and addition of tumor necrosis factor alpha (TNF-Δ to the cells augmented its secretion. In contrast, proteases released by activated human mast cells degraded VEGF-C.

Conclusions

These results show that lymphangiogenesis is induced in advancing AS. Furthermore, valvular myofibroblasts and activated mast cells were identified as novel regulators of lymphangiogenesis in human aortic valves.

Prevalence and long-term prognosis of fragmented QRS in standard 12-lead electrocardiogram in middle-aged subjects

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Aim

QRS-complex fragmentation (fQRS), defined as changes in QRS-morphology with various RSR'-patterns, has been recently associated with increased mortality and arrhythmic events in various cardiac conditions. However, the prevalence and prognostic significance of fQRS in general population are not known.

Methods

We evaluated the 12-lead ECGs of 10736 Finnish middle-aged subjects (52% males, mean age 44±8.5 years) drawn from general population and followed for 30±11 years. fQRS was defined as various RSR'-patterns in two consecutive leads within the same territory (inferior II, III, aVF; lateral I, aVL, V4-V6; anterior V1-V3). Primary endpoints were death from any cause, cardiac and arrhythmic death.

Results

fQRS was present in 18.4% (n=1979) of subjects, including 16 % (n=1714) in inferior leads, 0.8 % (n=84) in lateral leads, and 2.9 % (n=316) in anterior leads. A total of 8 (0.07%) subjects had fQRS in all territories. fQRS was more common in males than in females ($p<0.001$), and subjects with fQRS were somewhat older than those without ($p<0.001$). fQRS in lateral leads predicted all-cause mortality (adjusted-HR 1.34, 95%CI 1.02-1.76, p=0.03) and cardiac death (adjusted-HR 1.67, 95%CI 1.08-2.57, p=0.02) even after multivariant adjustments. fQRS overall (in any leads), fQRS in inferior leads and fQRS in anterior leads were not associated with increased mortality rates. Furthermore, none of the fQRS subgroups or fQRS overall predicted arrhythmic death.

Conclusion

fQRS is a common finding especially in the inferior leads of a standard 12-lead ECG in middle-aged subjects. The presence of lateral fQRS is associated with increased risk of cardiovascular mortality in otherwise healthy population.

Acute effect of adjunctive breast cancer radiotherapy on right ventricular systolic and diastolic function

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Aim

Breast cancer radiotherapy has been shown to depress left ventricular (LV) function, but its effects of right ventricular (RV) function are largely unknown. Reduced RV systolic performance correlates with poor prognosis across broad spectrum of diseases. The aim of this prospective single center study was to investigate whether conformal 3D breast cancer radiotherapy has negative effect on RV systolic and diastolic function in the acute phase.

Method

Forty consecutive patients with early left-side breast cancer were evaluated before and immediately after radiotherapy. A comprehensive 2D echocardiographic examination was performed at each visit. Several measurements of RV function were performed including tricuspidal annular plane systolic excursion (TAPSE), pulsed tissue Doppler peak velocity at the lateral RV wall (S'), RV inflow and outflow analysis, valvular assessment and venous flow analysis.

Results

Radiotherapy reduced TAPSE from a baseline value of 24.9±4.1 mm to 22.7±4.0 mm ($p< 0.001$). In keeping with this S' declined from 13.0±5.0 m/s to 12.3 ±4.4 m/s ($p=0.086$) and pulmonary flow velocity time integral (VTI) from 16.7±3.3 to15.9±2.5 ($p=0.089$), respectively. These changes were not related to LV systolic or diastolic changes. According to multivariate analysis the use of angiotensin-converting enzyme inhibitor or angiotensin II receptor blocker may protect against these changes ($p= 0.097$).

Conclusions

Modern 3D radiotherapy reduced RV systolic function. These early changes might progress in time, and continuous follow-up of cardiac function is warranted in radiotherapy patients. As a readily available and sensitive measurement TAPSE is as a practical tool for this purpose.