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RESEARCH ARTICLE

RIGHT VENTRICULAR INFARCTION: FOCUS AND REVIEW

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ABSTRACT

Acute coronary artery disease is a real obsession to any practitioner because of its heavy complications. Right ventricular infarction is a pathological entity distinguished by several diagnostic, therapeutic and prognostic features. These differences can be explained by the anatomical structure of the right ventricle and the physiopathological peculiarities in the occurrence of an infarction. Recent literature has focused on the subject in its details with sometimes inconsistent results. The ECG (18 leads) echocardiography and especially coronary angiography contribute intensively to the diagnosis. Urgent management of ventricular infarction is based on urgent recanalization, maintenance of a good right ventricular pre-load, it tends to reduce the high intra-hospital mortality.

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INTRODUCTION

Coronary artery disease and its consequences on the heart pump largely predominate in the incidence of various heart diseases in developed countries and, presumably, more and more in developing countries; their impact on morbidity and mortality is subject to countless studies and researches. Unlike left ventricular infarction, which has been extensively described and studied since antiquity, right ventricular infarction has been described as a distinct clinical entity since 1974 (Cohn, 1974). The variability of clinical expression and the peculiarities of the management of right ventricular involvement during myocardial infarction are based on a pathophysiology that is gradually better and better understood.

History Epidemiology: The pioneering autopsy study published in 1986 accurately quantified the frequency and importance of the mural extension of right ventricular infarctions; 107 patients (out of 131) who died of coronary pathology in cardiology intensive care unit, in fourteen months, were autopsied; there were 211 aftermath of left ventricular infarction (85 anterior, 56 lateral, 70 posterior). The frequency of right ventricular involvement was the same (64 versus 66%) in anterior and posterior infarction (near zero frequency in lateral infarction), but the size of right ventricular necrosis was very different (11% of the right ventricle was infarcted in anterior infarction; 50% of infarcted right ventricle in posterior infarcts), which explains why right ventricular involvement goes unnoticed in anterior infarcts (Andersen, 1987). The distinction between bi-ventricular involvement (extension to the right ventricle of the myocardial infarction), which is generally quite frequent during inferior infarction, is of the order of 26 to 50% depending on the series and 6 % in anterior infarction; whereas isolated right ventricular infarction remains exceptional in the literature (Ondrus et al., 2013).

Physiopathology: The right coronary is dominant in 60 to 90% of human hearts, vascularizing the atrio-ventricular node and the posterior walls of both ventricles as well as the posterior third of the septum, the sinus node, and the tricuspid sub-valvular apparatus. The anterior ventricular artery (AIV) provides vascularization of the tip and anterior wall of the right ventricle. The classic right ventricular infarction (RV) complicating inferior infarction is therefore due to proximal occlusion of the right coronary artery; Occlusions of the anterior inter ventricular artery are often accompanied by a small right ventricular infarction that goes unnoticed and has no clinical consequences. The sensitivity of the right ventricle to ischemia is low compared with that of the left ventricle; this is explained by several factors: in particular its low muscular mass with reduced oxygen requirements, systolic-diastolic vascularization with numerous collateralities as well as oxygen

diffusion from the ventricular chamber directly to the parietal thickness (Ondrus et al., 2013). Overgaard et al. explained that the occurrence of hemodynamic complications are frequent during right infarctions, resulting from the confluence of several mechanisms (Christopher Overgaard, 2002):

- An immediate dilatation of the RV limited by the pericardial envelope.
- Acute ischemic dilatation of RV leading to a decrease in LV pre-load which will aggravate reduction of the ejection fraction (mimicking the consequences of tamponade).
- Paradoxical displacement of the inter ventricular septum and atrio-ventricular asynchrony actively contribute to the alteration of left ventricular preload and therefore to the collapse of the ejection fraction.
- The frequent occurrence of rhythmic and conductive complications aggravates left ventricular dysfunction.

Nevertheless, the CABIN and then the ISNER experimental models indicated that cardiac output depression associated with RV infarction is largely dependent on pericardial integrity, which means that inter-ventricular dependence rather than the systolic function of RV is the main mechanism of hemodynamic disorders (Figure 1) (Cabins, 1987; Isner, 1988; Wellens, 1993). As on (Figure 1): Two physiological concepts explaining the harmful effects of a volume overload. (a) Normal ventricle: at the end systole, the free wall of the right ventricle (RV) moves towards the septum. (b) Pericardial Restriction Effects (Above: Before Volume Overload; Below: After Excessive Volume Loading): Right Ventricular (RV) dilatation, due to volume overload, may cause elevation intra pericardial pressure, an increase in pericardial stress (red arrow) and a change in geometry because of the inter ventricular septum. These changes contribute to the reduction of ejection fraction by decreasing the distensibility of the left ventricle (LV), preload and ventricular elastance. (c, d) Role of the inter ventricular septum (c: pure infarction of RV, d: infarction of RV with septal ischemia). (c) at the end of the systole, the free wall of the RV moves towards the septum. At the end of the diastole, the RV dilates during the diastole and the septum is reversed with respect to the reduced volume of the left ventricle. At the end of the systole, the septum thickens but moves paradoxically into the right ventricle, displacing the volume of the right ventricle despite its parietal dyskinesia. (d) Septal ischemia depresses septal contraction and overall function of the left ventricle, resulting in dilatation of the left ventricle. Non-thickening of the septal wall is associated with further displacement to the right ventricle. Septal thinning Pan-systolic and paradoxical displacement extension are associated with additional depression of the right ventricle performance. All these abnormalities are aggravated by the frequent occurrence of conduction and rhythm disorders (Ondrus, 2013; Philippe Meurin, 1995).

Diagnosis: Diagnosis is essentially based on clinical examination data, ECG, echocardiography and measurements of hemodynamic values (Boxt, 1999). According to many larges studies,Magnetic resonance imaging (MRI) is the gold standard for the exploration of RV dysfunction (Melissa Lyle, Ryan, 2016). Considering their indisputable contribution and their availability, ECG and echocardiography are the basic examinations for the diagnosis of RV. During a myocardial infarction of the inferior territory, the appearance of a triad, made of hypotension with increased jugular pressure and clarity on the chest X-ray, is considered as a base of orientation towards a right extension of the infarction with a sensitivity of 25% and a specificity of 96% (Dell'Italia, 1983); nevertheless, a Kussmaull sign or a paradoxical pulse can be revealing (Cintron, 1981). Physical examination may reveal signs of right heart failure, galloping noises, a functional or ischemic tricuspid regurgitation murmur or persistent hypoxia despite oxygenation (Takeuchi, 1989; Haji, 2000; Crawford, 2006).

The electrocardiogram: The new AHA / ESC recommendations call the completion of an 18-lead ECG as a recommendation I C (Patrick, 2013; Gabriel steg, 2012). On classical derivations, V1 and V2 can point to a straight extension (17). The ST elevation associated or not with Q waves on the V3R-V6R leads is strongly suggestive of the diagnosis of RV infarction (Ondrus, 2013; Erhardt, 1976; Braat, 1983; Yoshino, 1998). On 3 different studies, Braat et al. underlined the particular interest of V4R ST elevation as a diagnostic component of RV infarction by proximal coronary artery stenosis and as an indicator of poor prognosis (Braat, 1988; Braat, 1984; Braat, 1984). Indeed, it has been shown that ST elevation in V4R is strongly associated with RV dysfunction, hemodynamic impairment and elevated intra-hospital mortality (Braat, 1983; Braat, 1988; Braat, 1984; Braat, 1984; Candell-Riera, 1981; Klein, 1983); this elevation is generally absent after 12 hours of installation (Fijewski, 2002). It can occur during pulmonary embolism, antero-septal infarction, LV aneurysm, left ventricular hypertrophy and Brugada syndrome (Onur Baydar, 2016; Hsu, 2003). Other electrical manifestations related to RV infarction have been reported (Figure 2), higher D III than D II ST elevation, progressive V1 to V3 without necrosis and under-shift beyond 2 mm in DI and aVL with QRS widening in right leads (Turhan, 2003; Rashduni, 2003; Mittal, 1997; Verouden, 2009).

Echocardiography: Manyanatomical difficulties (complicated geometry and trabecular) limit the contribution of the 2D echocardiography during the RVinfarction (O'Rourke, 2004), it gives information about bi-ventricular functions, the cinetic disorder and associated valvularabnormalities. The presence of segmented cinetic disorders of the walls of the VD (hypokinesia or akinesia(that are quantified by a score index or echocardiography Strain) associated with its dilation defined more accurately the RV infarction than the only presence of cinetic disorders (Lebeau, 2004; Dell'Italia, 1984; Lopez-Sendon, 1983) Echocardiography is recommended to watch a paradoxical septum (Lopez-Sendon, 1983), a tricuspid regurgitation with right atrial dilatation (Yilmaz et al., 2003) and a deviation of the inter auricular septum in convexity towards the left auricle in more than 80% cases and even a re-permeabilization of the foramen oval seen with Doppler (Mattioli, 2000) .The measurement of the TAPSE (tricuspid annular plane systolic excursion) inTM mode informs on the systolic function of the RV (longitudinal shortening). On their study, Ghio and al have documented the prognostic impact of TAPSE impairment in cardiac failure regardless of etiology (Ghio, 2000), while, Engstromand al. (on a large scale) a TAPSE value less than 14 mm in patients admitted for myocardial infarction complicated by shock is correlated with significant long-term mortality (Engstrom, 2010). This echocardiographic parameter also informs about the systolic function of the LV and can be altered in case of impairment of just LV function (Kjaergaard et al., 2005; Kidawa, 2005; Lopez-Candales, 2006).

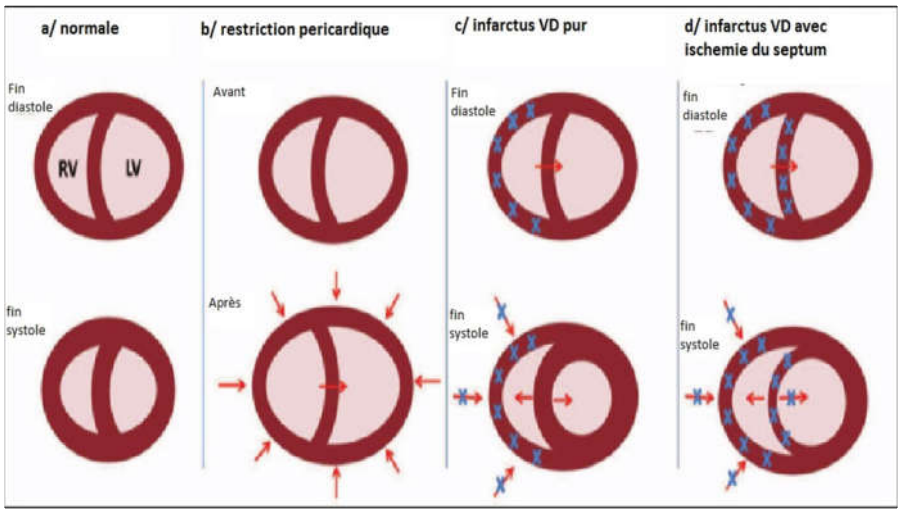

Figure 1. Pathophysiology of pressure effects during VCD infarction compared with other situations (5).

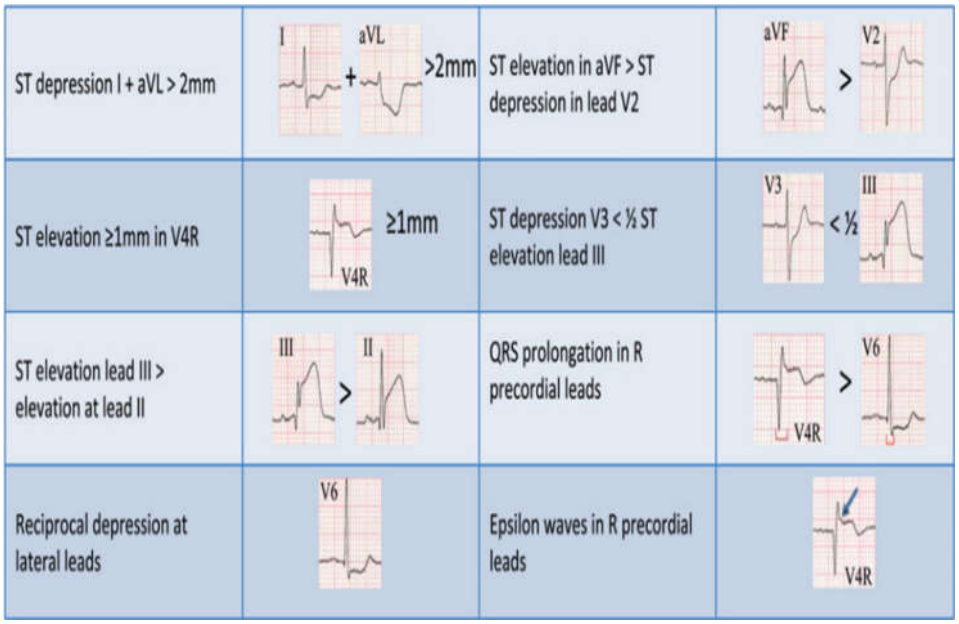

Figure 2. Different manifestations on the ECG of RV infarction (28).

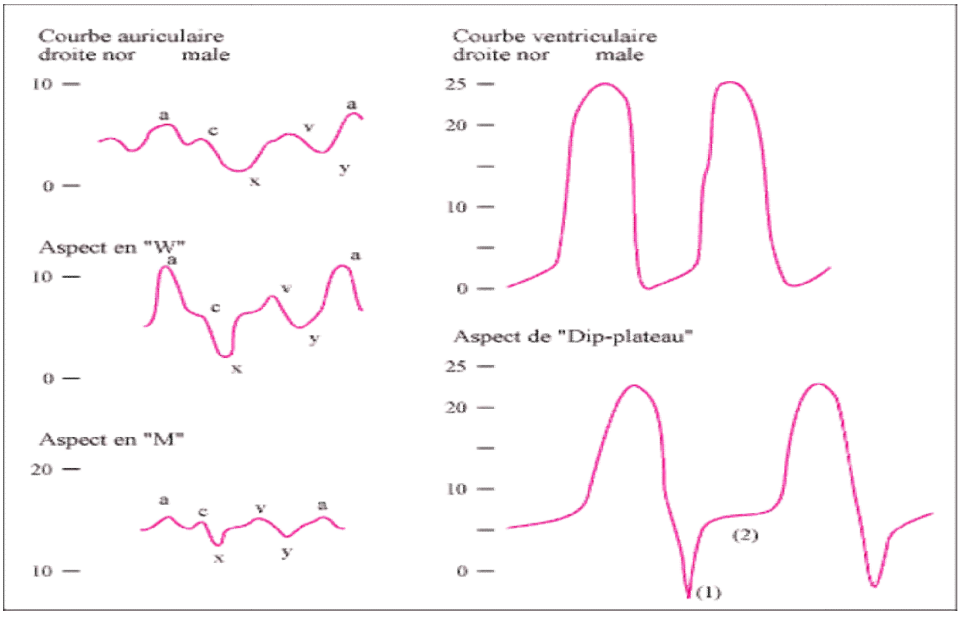

Figure 3. Various hemodynamic aspects possible during right ventricular infarction (5).

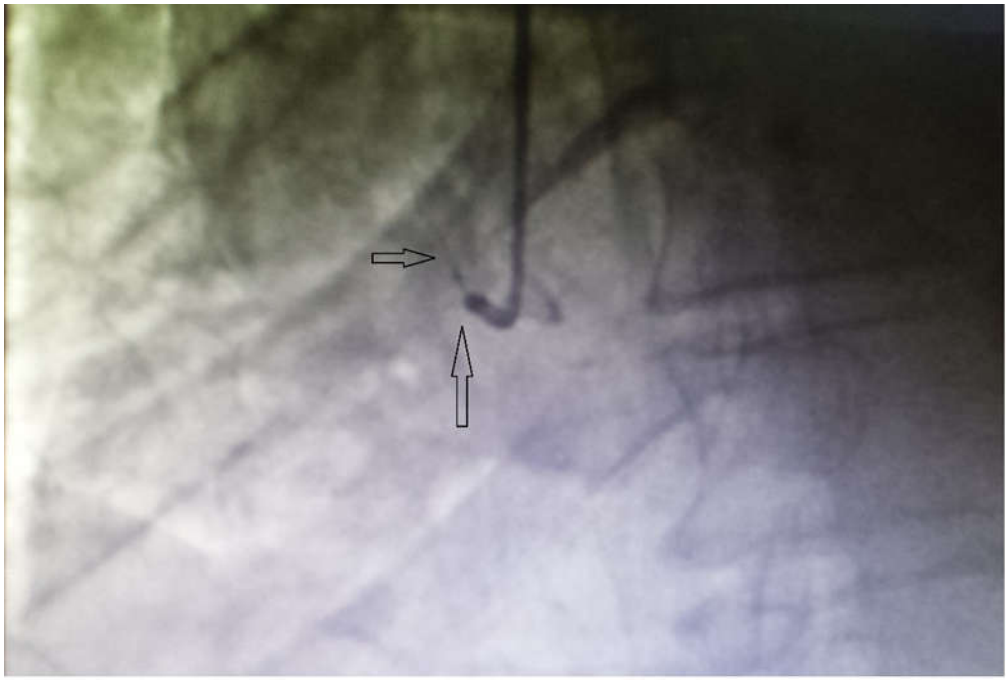

Figure 4. Proximal obstruction of the right coronary (with upwards the departure of the artery of the conus) (one of our patients)

Tissue Doppler provides objective information on the overall function of the right ventricle (Ho, 2006; Meluzin, 2001; Vogel, 2002). Several studies have highlighted the reduction in lateral systolic velocities of the tricuspid ring following RV infarction, which is correlated with RV dysfunction (Oguzhan et al., 2003; Alam et al., 2008; Ozdemir et al., 2003). The reduction in diastolic velocity is also related to the onset of RV dysfunction (49).

The alteration of the myocardial perfusion index (Tei index) is highly sensitive and specific for the extension of the inferior infarction to the RV (Ozdemir, et al., 2003; Kakouros, 2009). This index is considered by El Sebaie as the most significant echocardiographic index of proximal coronary involvement in the case of inferior infarction (Maha, 2016). 3D echocardiography has no additional contribution compared to conventional imaging techniques (Tamborini, 2008), however its results are in good agreement with those of cardiac MRI (Leibundgut, 2010); its necessity in case of RV infarction remains to be proven. Echocardiography helps to eliminate differential diagnosis in the presence of adiastolia, such as constrictive pericarditis and tamponade; and guides to diagnosis if right ventricular dysfunction.

The right catheterization (figure 3): is justified only if signs of bad tolerance, it can objectify (Philippe Meurin, 1995):

- **Adiastolia:** high right ventricular pressure with pressure of the right atrium (RAP) often greater than or equal to capillary pressure (in fact the elevation of the right atrium pressure which becomes close to the capillary pressure with a difference of less than 5 mm Hg is already a diagnostic criteria) and elevation of the diastolic right ventricle pressure (DRVP), a diagnostic criteria being the DRVP/ Cp ratio <0.8. We note the tendency to equalize diastolic pressures without pulmonary arterial hypertension.
- We will also look for the dip-tray aspect. The search for these signs of adiastolia can be sensitized by a filling test.
- On the right atrial pressure curve, a prominent "x" depression and a blunted "y" descent reflect a right ventricular filling disorder; the appearance of the wave "a" (atrial contraction) which is either increased (the atrial pressure curve thus taking on a "W" appearance) is lowered (the curve thus taking on the appearance of "M") makes it possible to differentiate two populations: when atrial systole is very effective ("W") the prognosis is better than when it is not very effective ("M"), which underlines the importance of atrial systole in this pathology.
- A decrease in cardiac output (colorimetric or thermo dilution) is often associated.
- At the same time, right ventriculography can be performed for the study of the segmental cinetic of the right ventricle and the search for tricuspid insufficiency.

The radionuclear techniques were considered as the basic for evaluation of the different volumes and the systolic function of the RV, currently they are overlaid by the cardiac MRI which is considered "the gold standard", its different acquisition techniques associated Gadolinium injection can detect early or late (aftermath of necrosis) ischemic lesions of RV and any associated lesions (Kjaer A, Lebech , 2005; Kumar, 2006; Younger, 2006). Chest tomography is mediocre in terms of RV infarction outside congenital coronary anomalies (He, 2006; Saremi, 2009).

Therapeutic particularities

Revascularization: Urgent revascularization by primary angioplasty improves the early normalization of ventricular function and reduces intra-hospital mortality (Hanzel, 2006); it contributes to the improvement of LV ejection fraction (Goldstein, 2002). At the same time, successful thrombolysis has a positive impact on the recovery of RV function and overall survival (Ramzy, 2009), but is frequently unsuccessful especially in cases of associated hypotension or proximal right

coronary involvement. (which is the case quite often (Figure 3). Drug dissolution in case of right ventricular MI is described as providing of several cases of reocclusion (Zehender, 1994). Thrombolysis, thereby, finds its place electively in case undoable primary angioplasty. Although the occurrence of shock state is quite common in RV infarctions compared to LV, cardiogenic shock mortality remains equivalent in both cases according to the Pfister and Jacobs studies (Pfisterer, 2003; Jacobs, 2003), but the occurrence of other potentially lethal complications (rhythmic, conductive and hemodynamic) further aggravates the prognosis (Mehta, 2001). Many studies have reported the progressive recovery of right ventricular function after 03 to 12 months, even without revascularization (Jacobs, 2003; Yasuda, 1990).

Other treatments: Given the physiopathological features of RV infarction, therapeutic behavior and prognosis differ from those of LV infarction, so, the use of diuretics, nitrates, angiotensin-converting enzyme inhibitors and opiates reduce RV preload of and may compromise the hemodynamic state (Ondrus, 2003). In a series of 300 cases,Ternacle states that the use of diuretics may be beneficial in case of RV infarction especially associated with oliguria and exceeds the filling effect alone (Hein, 2009), it remains of remarkable clinical interest as soon as the blood pressure begins to fall and it depends on the afterload of the RV and the fluid load status of the patient (Goldstein, 2002). Dobutamine is the drug of choice for cardiogenic shock in RV infarction, it improves the performance of RV and afterload with reduction of pulmonary vascular resistance and improves of atrio-ventricular conduction (Ferrario, 1994). Milrinone provides significant cardio-protection but may exacerbate hypotension (Hein, 2009; Ternacle, 2013); In this case the introduction of Dopamine sustains good coronary perfusion (Ferrario, 1994). Concomitant use of Levosimendan appears to improve LV function, decrease post-loading RV, and improve coronary perfusion (Ikonomidis et al., 2007; Follath, 2002), and these findings have been endorsed by the results of Russ and al (Russ, 2002). The intra-aortic balloon pump helps, among other things, to better infuse coronary arteries and contributes to the success of thrombolysis in RV infarction complicated by hypotension or shock (Kiernan, 2010; Giesler et al., 2006). The recovery of the physiological rhythm in case of conductive disorders after a RV infarction is essential. Bradycardia can be temporarily treated with atropine (Altun, 1998), especially associated with aminophylline or even the use of a stimulation probe or pace maker (Strasberg, 1991; Topol, 1982).

Prognosis

Short term: Right ventricular extension during myocardial infarction significantly increases intra-hospital mortality; a meta-analysis deduced that short-term mortality increased from 6.3% in the absence of right ventricular involvement to 17% in the presence of a right extension with an increased relative risk of mortality of 2.6 (Hamon, 2008). The frequent occurrence of refractory cardiogenic shock and conduction disorders explains this aggravation relatively. Primary angioplasty has improved short-term prognosis compared to fibrinolysis, although mortality remains high, cited in many publications in diverse populations (Keeley, 2003; TakuInohara, ?).

Long term: Counter to the short-term prognosis, the long-term one seems not to be influenced by the extension of MI to the right ventricle (Gumina, 2006). An average survival of 9 deaths per 100 patient-years after revascularization of the right ventricle MI; results comparable to those of infarction without RV extension (Philippe Meurin, 1995).

Conclusion

The right ventricle is distinguished by its anatomo-physiological peculiarities responsible for its physiopathological singularity explaining the advent of various complications. this implies an adequate diagnostic procedure and requires urgent and specific management with the validation of more and more new therapeutic methods to combat short-term prognosis.

REFERENCES

Alam M., Wardell J., Andersson E. et al., 2000. Right ventricular function in patients with first inferior myocardial infarction: assessment by tricuspid annular motion and tricuspid annular velocity. *Am Heart J.,* 139:710-15.

Altun A., Kirdar C., Ozbay G. 1998. Effect of aminophylline in patients with atropine-resistant late advanced atrioventricular block during acute inferior myocardial infarction. *Clin Cardiol*., 21:759-62.

Andersen HR., Falk, Nielsen D. 1987. Right ventricular infarction : frequency, size and topography in coronary heart disease : a prospective study comprising 107 consecutive autopsies from a coronary care unit. *J Am CollCardiol*., 10 : 1223-32.

Boxt LM. Radiology of the right ventricle. RadiolClin North Am 1999;37:379-400.

Braat SH., Brugada P., de ZC. et al., 1983. Value of electrocardiogram in diagnosing right ventricular involvement in patients with an acute inferior wall myocardial infarction. *Br Heart J.,* 49:368-72.

Braat SH., Brugada P., de ZC. et al., 1984. Right and left ventricular ejection fraction in acute inferior wall infarction with or without ST segment elevation in lead V4R. J Am CollCardiol., 4:940-4.

Braat SH., Brugada P., Den DK. et al., 1984. Value of lead V4R for recognition of the infarct coronary artery in acute inferior myocardial infarction. *Am J Cardiol.,* 53:1538-41.

Braat SH., Gorgels AP., Bar FW. et al., 1988. Value of the ST-T segment in lead V4R in inferior wall acute myocardial infarction to predict the site of coronary arterial occlusion. *Am J Cardiol.,* 62:140-2.

Cabins HS., Clubb KS., Wackers FJT., Zaret BL. 1987. Right ventricular infarction with anterior wall left ventricular infarction : an autopsy study. *Am Heart J* ., 113 : 16-23.

Candell-Riera J., Figueras J., Valle V. et al., 1981. Right ventricular infarction: relationships between ST segment elevation in V4R and hemodynamic, scintigraphic, and echocardiographic findings in patients with acute inferior myocardial infarction. *Am Heart J.,* 101:281-7.

Christopher Overgaard, M.D. et David Fitchett, M.D. Le choc cardiogénique dû à un infarctus du ventricule droit. Cardiologie Conférences scientifiques 2002. Volume VII, numéro 8.www.cardiologieconferences.ca

Cintron GB., Hernandez E., Linares E. et al., 1981. Bedside recognition, incidence and clinical course of right ventricular infarction. *Am J Cardiol.,* 47:224-7.

Cohn JN., Guiha NH., Broder MI., Limas CJ. 1974. Right ventricular infarction. Clinical and hemodynamic features. *Am J Cardiol.,* 33: 209-14.

Crawford LC., Panda M., Enjeti S. 2006. Refractory hypoxemia in right ventricular infarction: a case report. *South Med J.,* 99:79-81.

Dell'Italia LJ., Starling MR., Crawford MH. et al., 1984. Right ventricular infarction: identification by hemodynamic measurements before and after volume loading and correlation with noninvasive techniques. *J Am Coll Cardiol.*, 4:931-9.

Dell'Italia LJ., Starling MR., O'Rourke RA. 1983. Physical examination for exclusion of hemodynamically important right ventricular infarction. *Ann Intern Med.,* 99:608-11.

Engstrom AE., Vis MM., Bouma BJ. et al., 2010. Right ventricular dysfunction is an independent predictor for mortality in ST-elevation myocardial infarction patients presenting with cardiogenic shock on admission. *Eur J Heart Fail.,* 12:276-82.

Erhardt LR., Sjogren A., Wahlberg I. 1976. Single right-sided precordial lead in the diagnosis of right ventricular involvement in inferior myocardial infarction. *Am Heart J.,* 91:571-6.

Ferrario M., Poli A., Previtali M. et al., 1994. Hemodynamics of volume loading compared with dobutamine in severe right ventricular infarction. *Am J Cardiol.,* 74:329-33.

Fijewski TR., Pollack ML., Chan TC. et al., 2002. Electrocardiographic manifestations: right ventricular infarction. *J Emerg Med.,* 22:189-94.

Follath F., Cleland JG., Just H. et al., 2002. Efficacy and safety of intravenous levosimendan compared with dobutamine in severe low-output heart failure (the LIDO study): a randomised double-blind trial. Lancet360:196-202.

Gabriel Steg. 2012. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation European Heart Journal 33, 2569–2619 doi:10.1093/eurheartj/ehs215

Ghio S, Recusani F, Klersy C, et al. Prognostic usefulness of the tricuspid annular plane systolic excursion in patients with congestive heart failure secondary to idiopathic or ischemic dilated cardiomyopathy. Am J Cardiol 2000;85:837-42.

Giesler GM., Gomez JS., Letsou G. et al., 2006. Initial report of percutaneous right ventricular assist for right ventricular shock secondary to right ventricular infarction. *Catheter Cardiovasc Interv.,* 68:263-6.

Goldstein JA. 2002. Pathophysiology and management of right heart ischemia. *J Am CollCardiol.,* 40:841-53.

Gumina RJ, Murphy JG, Rihal CS, et al. Long-term survival after right ventricular infarction. *Am J Cardiol* 2006; 98:1571–1573.

Haji SA., Movahed A. 2000. Right ventricular infarctionediagnosis and treatment. *Clin Cardiol.,* 23:473-82.

Hamon M., Agostini D., Le Page O. et al., 2008. Prognostic impact of right ventricular involvement in patients with acute myocardial infarction: meta-analysis. *Crit Care Med.,* 36: 2023–2033.

Hanzel GS., Merhi WM., O'Neill WW. et al., 2006. Impact of mechanical reperfusion on clinical outcome in elderly patients with right ventricular infarction. *Coron Artery Dis.,* 17:517-21.

He H., Stein MW., Zalta B. et al., 2006. Computed tomography evaluation of right heart dysfunction in patients with acute pulmonary embolism. *J Comput Assist Tomogr.,* 30:262-6.

Hein M., Roehl AB., Baumert JH. et al., 2009. Anti-ischemic effects of inotropic agents in experimental right ventricular infarction. *Acta Anaesthesiol Scand*., 53:941-8.

Ho SY., Nihoyannopoulos P. 2006. Anatomy, echocardiography, and normal right ventricular dimensions. Heart., 92(Suppl 1):i2-13.

Hochman J., Sleeper L., Webb J., et al. 2006. SHOCK Investigators, Early revascularization and long-term survival in cardiogenic shock complicating acute myocardial infarction. *JAMA*295: 2511–2515.

Hsu LF., Ding ZP., Kam R. et al., 2003. Brugada-type ECG with polymorphic ventricular tachycardia: a red herring for isolated right ventricular infarction. Int J Cardiol 2003;91:255-7.

Ikonomidis I., Parissis JT., Paraskevaidis I. et al., 2007. Effects of levosimendan on coronary artery flow and cardiac performance in patients with advanced heart failure. *Eur J Heart Fail.,* 9:1172-7.

Isner JM. 1988. Right ventricular myocardial infarction. *JAMA* 259 : 712-8.

Jacobs AK., Leopold JA., Bates E. et al., 2003. Cardiogenic shock caused by right ventricular infarction: a report from the SHOCK registry. *J Am CollCardiol..,* 41:1273-9.

Kakouros N., Kakouros S., Cokkinos DV. 2009. Evaluating the results of right coronary artery angioplasty by using the echocardiographic marker of Tei index. Mediterranean Cardiology Meeting. *Taormina.,* 44.

Keeley EC., Boura JA. and Grines CL. 2003. Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomisedtrials.*Lancet.,* 361: 13–20.

Kidawa M., Kasprzak JD., Peruga J.Z., et al., 2005. Assessment of right ventricular systolic and diastolic function by Doppler tissue imaging in patients with first myocardial infarction and occluded right coronary artery. *Eur Heart J.,* 26(Abstract Sup):640.

Kiernan MS., Krishnamurthy B., Kapur NK. 2010. Percutaneous right ventricular assist via the internal jugular vein in cardiogenic shock complicating an acute inferior myocardial infarction. *J Invasive Cardiol.,* 22:E23-6.

Kjaer A., Lebech AM., Hesse B. et al., 2005. Right-sided cardiac function in healthy volunteers measured by first-pass radionuclide ventriculography and gated blood-pool SPECT: comparison with cine MRI. *Clin Physiol Funct Imaging.,* 25:344-9.

Kjaergaard J., Petersen CL., Kjaer A. et al., 2005. Evaluation of right ventricular volume and function by 2D and 3D echocardiography compared to MRI. *Eur J Echocardiogr.,* 7:430-8.

Klein HO., Tordjman T., Ninio R. et al., 1983. The early recognition of right ventricular infarction: diagnostic accuracy of the electrocardiographic V4R lead. *Circulation.,* 67:558-65.

Kumar A., Abdel-Aty H., Kriedemann I. et al., 2006. Contrast-enhanced cardiovascular magnetic resonance imaging of right ventricular infarction. *J Am CollCardiol.,* 48:1969-76.

Lebeau R., Di LM., Sauve C. et al., 2004. Two-dimensional echocardiography estimation of right ventricular ejection fraction by wall motion score index. *Can J Cardiol.,* 20:169-76.

Leibundgut G., Rohner A., Grize L. et al., 2010. Dynamic assessment of right ventricular volumes and function by real-time three-dimensional echocardiography:a

comparison study with magnetic resonance imaging in 100 adult patients. *J Am Soc Echocardiogr*., 23:116-26.

Lopez-Candales A., Rajagopalan N., Saxena N. et al., 2006. Right ventricular systolic function is not the sole determinant of tricuspid annular motion. *Am J Cardiol.,* 98:973-7.

Lopez-Sendon J., Garcia-Fernandez MA., Coma-Canella I., et al., 1983. Segmental right ventricular function after acute myocardial infarction: two-dimensional echocardiographic study in 63 patients. *Am J Cardiol.,* 51:390-6.

Maha H., El SebaieOsama El Khateeb. 2016. Right ventricular echocardiographic parameters for prediction of proximal right coronary artery lesion in patients with inferior wall myocardial infarction. *J Saudi Heart Assoc.,* 28:73–80.

Mattioli AV, Vandelli R, Mattioli G. Doppler echocardiographic evaluation of right ventricular function in patients with right ventricular infarction. J Ultrasound Med 2000;19:831-6.

Mehta SR., Eikelboom JW., Natarajan MK. et al., 2001. Impact of right ventricular involvement on mortality and morbidity in patients with inferior myocardial infarction. *J Am CollCardiol.,* 37:37-43.

Melissa Lyle, Ryan C. V. 2016. Conus artery occlusion causing isolated right ventricular outflowtract infarction: novel application of cardiac magnetic resonancein anterior STEMI. *Cardiovasc Diagn Ther*., 6(3):262-266.

Meluzin J, Spinarova L, Bakala J, et al. Pulsed Doppler tissue imaging of the velocity of tricuspid annular systolic motion; a new, rapid, and non-invasive method of evaluating right ventricular systolic function. Eur Heart J 2001;22:340-8.

Minamishima T., Sakata K., Mizuno Y., Sato K. et al., 2013. Usefulness of right ventricular tissue Doppler imaging for diagnosis of right ventricular myocardial infarction. *J Echocardiogr.* Sep;11(3):89-96.

Mittal SR., Jain SK. 1997. Electrocardiographic criteria for the diagnosis of right ventricular involvement in the setting of acute inferior infarction. *Int J Cardiol.,* 60:321-3.

O'Rourke RA., Dell'Italia LJ. 2004. Diagnosis and management of right ventricular myocardial infarction. *Curr Probl Cardiol*., 29:6-47.

Oguzhan A., Abaci A., Eryol NK. et al., 2003. Colour tissue Doppler echocardiographic evaluation of right ventricular function in patients with right ventricular infarction. *Cardiology.,* 100:41-6.

Ondrus T., Kanovsky J., Novotny T., Andrsova I ., Spinar J and Kala P. 2013. Right ventricular myocardial infarction: From pathophysiology to prognosis. Winter;18(1):27-30.

Onur Baydar, Veysel Oktay, Ugur Coskun, 3Ahmet Yildiz and Tevfik Gurmen. 2016. Isolated right ventricular infarction mimicking anterior ST-segment elevation. *Journal of Clinical and Diagnostic Research.* Apr, Vol-10(4): OD08-OD09.

Ozdemir K., Altunkeser BB., Icli A. et al., 2003. New parameters in identification of right ventricular myocardial infarction and proximal right coronary artery lesion. Chest., 124:219-26.

Patrick T. 2013. ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction. Circulation. 2013;128:e481.

Pfisterer M. Right ventricular involvement in myocardial infarction and cardiogenic shock. Lancet 2003;362:392-4.

Philippe Meurin, 1995. Gilles Montalescot. Infarctus du ventricule droit. John Libbey E ; vol 4 n 7 avril; 233-40.

Ramzy IS., 2009. O'Sullivan CA, Lam YY, et al.Right ventricular stunning in inferior myocardial infarction. *Int J Cardiol.,* 136:294-9.

Rashduni DL., Tannenbaum AK. 2003. Utility of ST segment depression in lead AVL in the diagnosis of right ventricular infarction. *N J Med.,* 100:35-7.

Russ MA., Prondzinsky R., Carter JM. et al., 2009. Right ventricular function in myocardial infarction complicated by cardiogenic shock: improvement with levosimendan. *Crit Care Med.,* 37:3017-23.

Saremi F., Gurudevan SV., Harrison AT. 2009. Isolated right ventricular infarction owing to anomalous origin of right coronary artery: role of MR and CT in diagnosis. *J Thorac Imaging.,* 24:34-7.

Strasberg B., Bassevich R., Mager A. et al., 1991. Effects of aminophylline on atrioventricular conduction in patients with late atrioventricular block during inferior wall acute myocardial infarction. *Am J Cardiol*., 67:527-8.

Takeuchi M, Minamiji K, Fujino M, et al. Role of right ventricular asynergy and tricuspid regurgitation in hemodynamic alterations during acute inferior myocardial infarction. JpnHeart J 1989;30:615-25.

TakuInohara, Shun Kohsaka, Keiichi Fukuda. The challenges in the management of right ventricular infarction.EuropeanHeart Journal: Acute Cardiovascular Care 2(3) 226 –234. DOI: 10.1177/2048872613490122.

Tamborini G., Brusoni D., Torres Molina JE. et al., 2008. Feasibility of a new generation three-dimensional echocardiography for right ventricular volumetric and functional measurements. *Am J Cardiol.,* 102:499-505.

Ternacle J. et al., 2013. should furosemide be avoided in acute right ventricular myocardial infarction? Annales de Cardiologie et d'Angéiologie 62 95–100.

Topol EJ., Goldschlager N., Ports TA. et al., 1982. Hemodynamic benefit of atrial pacing in right ventricular myocardial infarction. *Ann Intern Med.,* 96:594-7.

Turhan H., Yilmaz MB., Yetkin E. et al., 2003. Diagnostic value of aVL derivation for right ventricular involvement in patients with acute inferior myocardial infarction. *Ann Noninvasive Electrocardiol.,* 8:185-8.

Verouden NJ., Barwari K., Koch KT, et al. Distinguishing the right coronary artery from the left circumflex coronary artery as the infarct-related artery in patients undergoing primary percutaneous coronary intervention for acute inferior myocardial infarction. Europace 2009;11:1517-21.

Vogel M., Schmidt MR., Kristiansen SB.et al., 2002. Validation of myocardial acceleration during isovolumic contraction as a novel noninvasive index of right ventricular contractility: comparison with ventricular pressure-volume relations in an animal model. *Circulation.,* 105:1693-9.

Wellens HJJ. Right ventricular infarction. *N Engl J Med* 1993 ; 330 : 1036-8.

Yasuda T., Okada RD., Leinbach RC. et al., 1990. Serial evaluation of right ventricular dysfunction associated with acute inferior myocardial infarction. *Am Heart J.,* 119:816-22.

Yilmaz M., Erol MK., Acikel M. et al., 2003. Pulsed Doppler tissue imaging can help to identify patients with right ventricular infarction. *Heart Vessels.,* 18:112-16.

Yoshino H., Udagawa H., Shimizu H. et al., 1998. ST-segment elevation in right precordial leads implies depressed right ventricular function after acute inferior myocardial infarction. *Am Heart J.,* 135:689-95.

Younger J., Plein S., Greenwood JP. 2006. Isolated right ventricular infarct demonstrated by cardiac MRI. *Int J Cardiol.,* 113:e62-3.

Zehender M., Kasper W., Kauder E. et al., 1994. Eligibility for and benefit of thrombolytic therapy in inferior myocardial infarction: focus on the prognostic importance of right ventricular infarction. *J Am Coll Cardiol*., 24:362-9.

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