CORRELATION BETWEEN CT GUIDED PULMONARY ARTERY DIAMETER AND RIGHT VENRICULAR FUNCTION BY ECHOCARDIOGRAPHY IN PULMONARY HYPERTENSION PATIENTS

Thesis

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LIST OF ABBREVIATIONS

AT …….Acceleration time

EF …… Ejection fraction

ET …… Ejection time

FAC ……... Fractional area change

IVA …… Isovolumic acceleration

IVC ……. Inferior vena cava

IVCT …….. Isovolumic contraction time

IVRT ……...Isovolumic relaxation time

MPI ……... Myocardial performance index

MRI ……. Magnetic resonance imaging

LV ……..…Left ventricle

PA ……..…Pulmonary artery

PADP …….. Pulmonary artery diastolic pressure

PH ……..…Pulmonary hypertension

PLAX …….. Parasternal long-axis

PSAX …….. Parasternal short-axis

PVR ……..….Pulmonary vascular resistance

RA ……..…...Right atrium

RIMP …….…. Right ventricular index of myocardial performance

RV …… Right ventricle

RVH ……. Right ventricular hypertrophy

RVOT ……... Right ventricular outflow tract

RVSP …… Right ventricular systolic pressure

SD ……. Standard deviation

SPAP …….Systolic pulmonary artery pressure

TAM ……. Tricuspid annular motion

TAPSE ……. Tricuspid annular plane systolic excursion

3D ……. Three-dimensional

TR ……. Tricuspid regurgitation

2D ……..Two-dimensional

P/A ……pulmonary artery diameter/aortic diameter

PAH……Pulmonary arterial hypertension

NO…..……nitric oxide

CTEPH ……..chronic thromboembolic PAH

INTRODUCTION

Introduction

Pulmonary hypertension is defined as a resting mean pulmonary arterial pressure of 25 mmHg or greater at right heart catheterization, this is a hemodynamic feature that is shared by all types of pulmonary hypertension in the Dana Point classification system. A resting mean pulmonary arterial pressure of 20mmHg or less is considered normal, while mean pulmonary arterial pressures ranging between 21-24 mmHg is considered abnormal requiring further investigation about the clinical course of disease (45).

Pulmonary hypertension may primarily affect either the arterial (precapillary) or the venous (postcapillary) pulmonary circulation. Pulmonary arterial hypertension may be idiopathic or arise in association with chronic pulmonary thromboembolism; pulmonary embolism caused by tumor cells, parasitic material, or foreign material; parenchymal lung disease; liver disease; vasculitis; human immunodeficiency virus infection; or a left-to-right cardiac shunt. Its histologic characteristics include vascular changes-medial hypertrophy, intimal cellular proliferation, intraluminal thrombosis, and the development of plexiform lesions-that manifest primarily in the muscular pulmonary arteries. Features of pulmonary arterial hypertension that may be seen at computed tomography (CT) are central pulmonary artery dilatation, abrupt narrowing or tapering of peripheral pulmonary vessels, right ventricular hypertrophy, right ventricular and atrial enlargement, dilated bronchial arteries, and a mosaic pattern of attenuation due to variable lung perfusion (12)

CT-determined mean pulmonary artery diameter has excellent diagnostic value for detection of PH in patients with advanced lung disease. Therefore, standard chest CT scans can be used to screen for PH as a cause of exertional limitation in patients with

parenchymal lung disease. Because CT is commonly used to evaluate parenchymal lung disease, this information is readily available (34).

In noncontrast multi-detector computed tomography (CT), both main pulmonary artery (mPA) diameter and ratio of the mPA to the ascending aorta (ratio PA) measurements are easily obtained and highly reproducible. This simple measurement of axial mPA diameter at the level of the bifurcation of the right pulmonary artery is easy to define anatomically. The ascending aorta can be simultaneously measured to provide a quick calculation of the ratio PA. The Framingham Heart Study showed that the normal referance sex-specific CT cutoff value for mPA for men is 29 mm and for women is 27 mm and for ratio PA is 0.9 for both men and women. These 2 simple measurements may hold some promise for the preclinical diagnosis for the "silent diseases" of the cardiopulmonary system (186).

Severity of symptoms and survival in patients with pulmonary artery hypertension are strongly associated with right ventricular function, and right heart failure is the main cause of death in patients with PAH. Echocardiography and cardiac magnetic resonance imaging allow noninvasive evaluation of right ventricular function and structure .Simple, reproducible, noninvasive measures of right ventricular function would help to improve the management of patients with PAH, and to provide tools with which to help establish the optimal therapeutic approach to manage not only the effects of the disease on the pulmonary vasculature, but also to support and improve right ventricular function (18).

AIM OF THE WORK

Aim of the work ;

To correlate between the pulmonary artery diameter or its ratio to aortic diameter in CT chest and right ventricular function measured by echocardiography in pulmonary hypertension patients.

REVIEW OF LITERATURE

Chapter one

Pulmonary hypertension

Pulmonary hypertension (PH) is defined as a hemodynamic and pathophysiological condition characterized by an increase in mean pulmonary arterial pressure (P_{pa}) to ≥25 mmHg at rest as measured by right heart catheterization (RHC).(1)

Because this definition is based on hemodynamic criteria, pulmonary hypertension can be the result of a variety of diseases of different causes. Pulmonary arterial hypertension (PAH), however, should be distinctly differentiated from pulmonary venous hypertension resulting from left heart disease. PAH is characterized by elevations in the pulmonary arterial pressure and pulmonary vascular resistance (PVR) leading to right ventricular failure and premature death. (2)

Thus, the definition of PAH also requires normal pulmonary artery occlusion (or wedge) pressure to exclude elevations of pulmonary artery pressure simply as a compensation for elevated pressures in the left heart. PAH is commonly caused by or associated with an underlying pulmonary, cardiac or systemic disease (associated PAH [APAH], previously known as secondary pulmonary hypertension). Rarely, PAH is present in the absence of an identifiable cause or associated underlying disease and is referred to as idiopathic PAH (IPAH) or primary PAH (PPH). A familial form of IPAH (FPAH) accounts for about 6% of cases.(2)

The small case series on elevated pulmonary artery pressures in otherwise healthy young individuals in the 1950s and 1960s and the epidemic of anorexigenic-associated PAH in Europe led to the first World Health Organization (WHO) conference on PPH in 1973.(3)