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Elevated LVEDP, Chronic Pulmonary Oedema and Valve Disease

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Highlights

As part of ageing and with increased longevity of older people there has been a rise of those with increased left ventricular end diastolic pressure (LVEDP) and left ventricular diastolic dysfunction (LVDD). Comorbidities like hypertension, diabetes, chronic kidney disease, coronary artery disease and others appear to be contributing to this. Chronic interstitial pulmonary oedema may be a part of the presentation of those with elevated LVEDP/LVDD. Progressive valvular heart disease may also complicate the picture and make clinical decision-making difficult. This case report discusses these issues.

Keywords: LVEDP, chronic pulmonary oedema, valve disease

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Introduction

Elevated left ventricular end diastolic pressure (LVEDP) is one of the main features in the diagnostic algorithm of heart failure with preserved ejection fraction (HFpEF) [1]. It is also a feature in those with pure left ventricular diastolic dysfunction (LVDD), (and preserved ejection fraction), who have never developed heart failure [2]. In fact, it has been proposed that left ventricular diastolic dysfunction may progress through stages with increasing LVEDP from a stage of asymptomatic pre-clinical diastolic dysfunction to a pre-HFPEF stage and finally to the HFPEF stage [2]. With the population of elderly people rising, increased LVEDP appears to be a common finding on echocardiography and during left ventricular angiography [2, 3]. An important cause of this may be increased vascular resistance associated with ageing coupled with common comorbidities present in the elderly such as hypertension, diabetes mellitus or chronic kidney disease [4] but other causes such as coronary artery disease [5] may also play a role. It has been speculated that in many cases the elevated LVEDP may be the end result of the increased effort that the LV muscle undertakes to force blood into a high resistance vascular circuit [4]. This world of elevated LVEDP related to HFPEF and left ventricular diastolic dysfunction appears to be bringing in some unique challenges that are worth highlighting. I would like to briefly describe a patient of mine to make my case.

Case Report

A 77 year old lady presented with recurrent pulmonary oedema, the cause of which was initially unclear. She had a background history of hypertension, Type 2 diabetes mellitus, chronic renal impairment and moderate aortic stenosis (AS) with mild to moderate aortic regurgitation (AR). None of these were clinically important at that stage. On echocardiography, her LV systolic function was well preserved but the E/e was elevated at >15 suggesting an elevated LVEDP but aortic valve Dopplers indicated moderate aortic stenosis (peak velocity 3.3m/s, peak aortic valve gradient 42.4 mm Hg and mean gradient 24.9 mm Hg) with mild moderate AR . Left and right heart catheterisation showed non obstructive coronary atheroma with an elevated LVEDP of 20mm Hg and a mean instantaneous gradient across the aortic valve (using a Langston catheter) of 25 mm Hg suggesting moderate AS. There was no evidence of a left to right shunt on oximetry but pulmonary artery mean pressure was mildly elevated

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at 31mm Hg in keeping with mild post capillary pulmonary hypertension. The main problem appeared to be left ventricular diastolic dysfunction leading to elevated LVEDP and after other investigations I felt that the recurrent pulmonary oedema was being triggered by paroxysmal atrial fibrillation; I was luckily able to control this with oral amiodarone. My thinking here was that the AF was easily triggering the pulmonary oedema since the LVEDP was chronically elevated due to LVDD. Interestingly, this lady was also persistently hypoxic with an oxygen saturation of 90% on air and a PO2 of between 8-9kPa. The cause of the hypoxia remained unclear as lung function testing and a respiratory assessment did not find any abnormalities. Chest X rays showed frank pulmonary oedema during admissions with acute worsening of breathlessness but at other times simply suggested mild pulmonary venous congestion (figure 1). A high resolution CT scan of the thorax did provide some clues showing no lung parenchymal abnormalities of significance but there was mild perihilar and lower zone ground glass changes seen on the CT suggestive of chronic pulmonary oedema at a time when the patient was clinically stable and living at home with NYHA class III to IV exertional breathlessness, not in acute heart failure (fig 2). My feeling about this was that she was suffering from chronic interstitial pulmonary oedema due to the persistently grossly elevated LVEDP. I would have liked to reduce her LVEDP with drugs like diuretics but I found it difficult to treat. High doses of diuretics affected her renal function markedly; her blood pressure was already well controlled and she was now in sinus rhythm, so there was no AF to cardiovert. I restricted her fluid intake and arranged home oxygen for her which did help. Despite this, she was subsequently admitted with congestive cardiac failure a few times. I looked carefully to find another cause for her breathlessness (including an opinion from a colleague in respiratory medicine) but couldn't find anything of significance. Over the next 4 years she remained very symptomatic with limiting breathlessness and her gradient across the aortic valve.

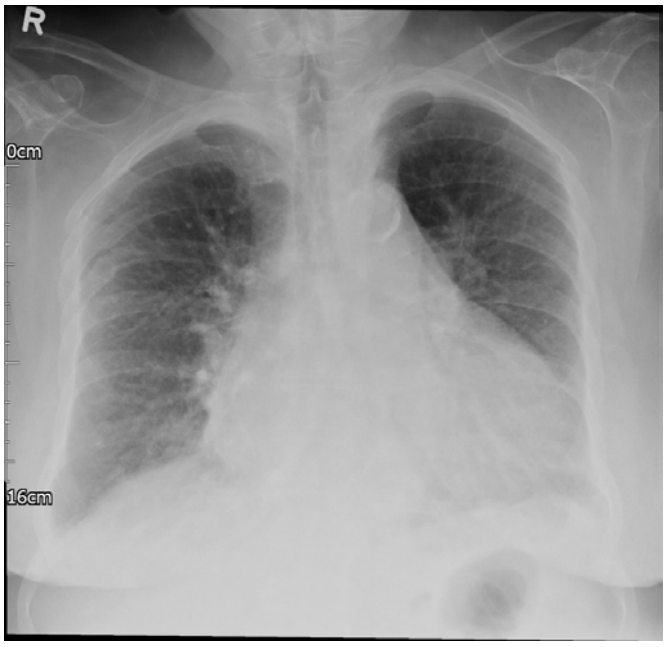

Figure 1. Chest X ray at a time when the patient was clinically stable and not in acute pulmonary oedema. A small left pleural effusion with pulmonary venous congestion is seen.

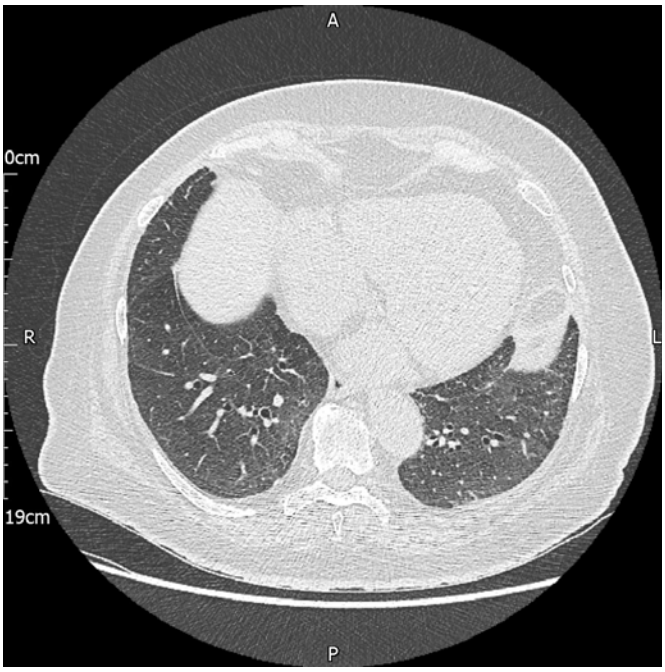

Figure 2. Mild ground glass changes in the lower lobes with septal thickening is seen.

slowly climbed but the aortic stenosis remained in the moderate range until right at the end of this period .

Discussion

Now here is a dilemma in such cases with moderate valve disease and pre-existing marked elevation of LVEDP due to LVDD: should we consider valve surgery early even when the valve disease is moderate rather than severe? The argument would be that moderate AS in this case would be very likely to be contributing to the high LVEDP that almost invariably is the reason for her chronic pulmonary oedema and her disabling symptoms (as her aortic valve gradient climbed in the next few years this became more likely and more relevant). Correcting the valve problem may well help symptoms by reducing the chronically elevated LVEDP. She is not my only patient with this problem (another one has moderate mitral regurgitation with pre-existing high LVEDP of >20 mm Hg) and I am sure that many other cardiologists are asking this question. If we think that the question is valid then we might need to review the indications for valve intervention in the light of HFPEF as well as in those with pre-existing high LVEDP due to LVDD. In fact, my lady described above, eventually developed severe aortic stenosis (peak velocity 4.2m/s, peak gradient 69mm Hg, mean gradient 41mm Hg, and valve area 1 square cm with moderate to severe AR on echocardiography) for which she underwent a TAVI (Transcatheter Aortic Valve Implantation) procedure. This helped both her hypoxia and her symptoms quite dramatically and rapidly post procedure making me wonder why I had to wait that long to intervene on her valve disease!

Conclusion

Ground glass changes on chest CT scans (in non acute pulmonary oedema patients) have been described mainly in patients with Pulmonary Hypertension due to left heart disease (WHO Group 2 Pulmonary Hypertension) [6, 7]. Bilateral septal thickening often accompanies patchy ground glass changes predominantly in the dependant lower lobes, sometimes accompanied by small

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unilateral or bilateral pleural effusions. However, the changes can be subtle and intermittent and recognition that this represents chronic interstitial pulmonary oedema leading to hypoxia in HFPEF/LVDD patients is limited, often leading to an intensive hunt for other (mainly respiratory) causes of hypoxia without success, causing confusion. With an ever increasing burden of LV diastolic dysfunction, HFPEF and pulmonary hypertension secondary to HFPEF, being familiar with all the expected clinical presentations (including the unusual ones) of this syndrome will help to arrive at a diagnosis early and to intervene in a timely and appropriate manner. For chronic pulmonary oedema related to HFPEF/LVDD the mainstay remains diuretics but a common sense treatment approach for HFPEF seems sensible. [8]

Declarations of Interest

The author declares no conflict of interest.

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The author states that they abide by the authors' responsibilities and ethical publishing guidelines of the International Cardiovascular Forum Journal.[9]

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