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HEMODYNAMICS IN HEART FAILURE – IS CLINICAL EXAMINATION RELEVANT?

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Elucidating hemodynamic parameters remains the hallmark of management in patients with acute or chronic heart failure. Demystification of assessment of filling pressures is a continual and vital pursuit for cardiologists. In the era of hand-held imaging devices, extensive diastolic guidelines and readily available emerging biomarkers ala, brain natriuretic peptide (BNP); is the role of clinical examination (CE) becoming obsolete? CE consists of carefully taken targeted history and meticulously conducted physical examination.A properly conducted clinical examination provides in depth insight into the hemodynamics - elevated ventricular filling pressures and cardiac output. This helps both in immediate clinical management and offers important prognostic information.[1] Though suggestions have been rife to do away with clinical examination and abandon stethoscope in clinical management,there is evidence to elucidate and emphasize the role of clinical examination especially in the setting of heart failure.[2] Recently, a lot of hue and cry has been raised about lack of clinical skills – 'hyposkilia' in the newer generation of doctors.[3] Most experienced clinicians still believe in clinical examination being an important tool as it has withstood the test of time in spite of innovations in biomarkers and technology. This article confines to offer objective evidence on the role of clinical examination in determining hemodynamics and prognosis in patients with HF.

To set things in perspective, it was Forrester who four decades ago demonstrated, that clinical examination classified patient after myocardial infarction into four clinical profiles that correlated well with invasively obtained data. The profile were based on the presence or absence of congestion or wetness by documenting pulmonary capillary wedge pressure more than or equal to 18 mmHg and adequacy of cardiac output determined by having cardiac index more than 2.2 l/min/m^2. Profile 1presented with no congestion and no hypoperfusion, profile 2 congestion without hypoperfusion, profile 3 hypoperfusion without congestion and profile 4 both congestion and hypoperfusion.[4] Both clinical and invasive markers predicted short-term markers with enhanced mortality within the presence of congestion and still worse with hypoperfusion.[4]

Patients of HF can be classified into wet or dry depending on the volume status and warm or cold based on perfusion status.After a thorough clinical examination HF patients can be confidently classified into one of the four quadrants as proposed by Stevenson. Profile A has dry patients with no hypoperfusion, profile B has patients who are wet but no hypoperfusion, profile C has dry patients with

hypoperfusion and profile D patients are wet with hypoperfusion.[5] It is clear that the ability to assess preload and congestion is far superior than to elucidate effects of cardiac output – perfusion. Elevated ventricular filling pressureis inferred usually by elevation of right atrial pressure. A patient is considered to be wet when the estimated pulmonary capillary wedge pressure is more than 22 mmHg and considered cold when cardiac index is less than 2.2 l/min/m^2 – invasive parameters correlate very well with clinical status.[6]

The determination of 'wet' is based on the presence of any sign or symptom that is associated with elevated ventricular filling pressures. Assessing volume status is critical in the management of HF, helpful in titrating the dosage of diuretic and agents used to modulate preload and afterload. Clinical findings that help to assess volume status are distention of jugular veins (JVD), hepatojugular reflux (HJR). Orthopnea, bendopnea and square-wave response of blood pressure during Valsalva maneuver.[7] Volume depletion can be detected by the presence of signs of dehydration, changes in blood pressure and heart rate with changes in posture.[7]

Of all the signs, elevated Jugular vein or JVD is perhaps the most reliable, useful and reproducible sign of elevated right atrial pressure reflecting the raised right ventricular filling or right ventricular end diastolic pressure.[8] JVD is an estimate of right atrial pressure and a visible JV reflects more than 10cm H2Oand this can be converted to mm Hg bearing in mind the relationship of 1 mmHg equal to 1.36 cm of H2O.[9,10] Internal jugular vein is usually employed for assessment, but external jugular vein may be used when in doubt.[11] Recognition and documentation of JVD is an integral art of clinical examination, and in a study cardiology faculty was found to be more accurate than doctors in training.[12] In a study in which patients were divided in three groups based on invasive assessment of JVP - less than 8 mmHg, more than 12 mmHg and the intermediate group assessment of JVD besides appreciation of orthopnea were most dependable markers.[13] Presence of either or together JVD and a third heart sound gallop were observed to increase the risk of hospitalization, composite risk of hospitalization and death and death alone in SOLVD study.[14] In a recent study employing more than 2000 patients admitted with acute HF presence of JVD was associated with increased risk of in-hospital adverse events and increased 30days, one year and ten years all-cause mortality.[15] An inspiratory increase in venous pressure like Kussmaul's sign was shown to have adverse effects in patients with advanced HF.[16]

Hepato Jugular reflux is considered to be positive if JVP increases by more than 3 cm lasting for more than 10 seconds after applying continuous pressure on abdomen and demonstrating a rapid fall on releasing the pressure.[17] This demonstrates improved detection of raised right heart filling pressure in view of increase venous return from inferior vena cava. This reliably predicts that pulmonary capillary pressure is equal to or more than 15mm Hg. This may be considered as a precursor of frank JVD.[18] In a study on patients being evaluated for cardiac transplantation, 42%had HJR and all these patients had PCWP more than 18 mmHg.[6] Besides diagnostic it also offers prognostic information. In ESCAPE study patients with persistent HJR as against in those with resolved had higher 6 months morbidity and mortality.[14] Moreover in patients with persistent JVD as against only HJR had higher 6 months mortality.[19] Presence of dyspnea in supine position is known as orthopnea is association with increase in pulmonary capillary wedge pressure being more than 28 mm Hg.[20] Presence of JVD and orthopnea indicate increase in left heart filling pressure, both offering additive information in patients with HF. A new term is being coined as 'orthodema' taking into account both orthopnea and edema. Worsening of score of orthodema is associated with worse outcome.[20]

There has been renewed interest in square wave response of blood pressure to Valsalva maneuver in patients with heart failure. This was described more than four decades ago.[21] In normal individuals arterial blood pressure registers a fall during strain phase of Valsalva maneuver as the venous return decreases. As against that, blood pressure in patients of HF with elevated left heart filling pressure remains higher as the venous return stays elevated.[21,22] This reflects increase in pulmonary capillary artery pressure measured invasively. This does not occur in patients with isolated right heart pressure.[23.24]

A new term has been introduced in the world of cardiology called 'bendopnea' observed in patients with advanced HF. Patients develop dyspnea on bending forward at the waist and it is assessed in patients while sitting in a chair and bending forward touching their feet with hands.[25] It is deemed to be present if a patient develops dyspnea within 30 seconds of bending forward.[25] Bendopnea was seen in a minority of patients with elevated filling pressure with low cardiac output.[26] Recent studies have documented the presence of bendopnea in one fifth to half the number of patients presenting for evaluation for heart failure and its presence has been seen to increase worse outcomes in six months.[26-29]

As against assessment of elevated filling pressure, adequacy of perfusion is more challenging. Helpful clinical indicators being narrow pulse pressure, cool extremities, global assessment of cold and recently added bendopnea. Pulse pressure, difference between systolic and diastolic pressure and not systolic blood pressure signifies low cardiac output. Proportional pulse pressure correlated well with invasively determined cardiac output in a study on patients with HF.[30] However in another study it correlated in one quarter of patients.[11] In the same study it was shown that physicians' assessment of cold profile was associated with lower cardiac output.[11] Low cardiac output may be considered in differential diagnosis in a patient of HF whose

renal functions deteriorate during diuresis.

'Hemodynamic congestion' is described as a clinical condition in which a patient may have elevated left heart filling pressure but no apparent clinical abnormal findings. This may be diagnosed by estimating raised natriuretic peptides as a response to stretching of ventricular walls.[31] Natriuretic peptide measurements may help to add information for the diagnosis and risk stratification. However GUIDE-IT study failed to show superiority of natriuretic-guided therapy as against serial clinical assessments.[32]

In a recentprospective analysis on 452 patients admitted to cardiomyopathy services showed that clinical profiles helped in determining the outcomes. In profiles B and C risk of death and urgent transplantation increased as determined by univariate (HR 1.83 p=.02) and multivariate analysis (HR 2.48, p=0.003). Clinical profiles offered prognostic information even in patients in NYHA class III/IV symptoms (profile B: HR 2.23, p = 0.026; profile C: HR 2.73, p = 0.009). Stevenson clinical classification is helpful in defining profiles and these profiles predict outcomes and may be used to guide therapy and identify populations for future investigation.[3] The four clinical profiles offer, besides diagnostic insights, important therapeutic information. In a study warm and wet profile and cold and wet profile proved to be an independent risk factor for mortality and urgent transplant at one year.[4,11] Profile of wet and cold versus warm and dry was associated with 50% increased risk of re-hospitalization and death.[11] The profiles proposed by Stevenson are helpful to chalk out a therapeutic course. Vasodilators and inotropes may be added to parenteral diuretics in patients with decompensated heart failure. In profile B only diuretics may suffice, as perfusion is normal. In profile C vasodilators and inotropes may be helpful.[33]

Determining right versus left elevated filling pressure may pose a huge challenge. Most clinical markers like JVD, peripheral edema and ascites signify elevated right sided filling pressures whereas HJR, bendopnea, orthopnea and square-wave response point to left sided elevated filling pressures. In most patients, more than three fourths, elevated right sided pressure reflect elevated left sided filling pressure. In one fourth of cases there may exist discordance between left and right sided filling pressures with isolated increase of either side.[34-37] Based on above features patients can be classified to have elevated isolated right sided, left side or both. A disproportionate increase in right-sided pressure as compared to left sided filling pressure has different response to treatment and prognosis. This is termed as right-left equalizer with elevated right atrial pressure to pulmonary capillary wedge pressure more than .[67] is seen to be associated with impaired renal function and worse outcome.[38]

A physician cannot depend on biomarkers in the management of HF. There is a lack of reliablepredictors to document efficacy of drugs and devices in heart failure, and this is considered as an impediment to the development and evaluation of novel therapies. Recentlya study sought to determine whether treatment-related changes in natriuretic peptides (NPs) predict longer-term therapeutic effects in clinical trials of heart failure (HF). It was observed that treatment-related changes in NPs did not correlate with longer-term treatment effects on all-cause mortality (r = 0.12; p = 0.63), but correlated with HF hospitalization (r = 0.63; p = 0.008).[39]

To conclude, meticulously conducted clinical examination is of immense importance in the management of patients with HF. It offers both diagnostic and prognostic information and is helpful in guiding the therapy. Clinical examination is more accurate in assessing elevated ventricular filling pressure inferred from signs of elevated right atrial pressure. Detection of low cardiac output remains challenging. In patients of HF with deteriorating renal function, low cardiac output and right-left equalizer of filling pressure should be considered

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