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Relations between Clinical Signs of Right and Left Cardiac Decompensation and Radiological Signs Thereof

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ABSTRACT

Ten consequtive patients arriving at the emergency department for severe systemic cardiac decompensation were investigated in respect of 17 clinical and laboratory parameters indicative of right or left heart failure. Investigations were made at arrival to the hospital and after completed in-hospital care.

In respect of left heart failure the prescence of rales and signs of interstitial oedema had a similar sensitivity. Both were of diagnostic value. Right heart failure was best diagnosed with clinical parameters such as pitting oedema, filling of the jugular vein, and liver enlargement. The laboratory parameters were less sensitive and appeared to have not a diagnostic but well a confirmatory value.

INTRODUCTION

Clinical decision-making in cardiac decompensation both in respect of left and right ventricular failure is based on symptoms and signs.Pertinent findings are often verified by chest x-ray. This implies that chest x-ray is as sensitive or a more sensitive method and gives more reliable results than the clinical examination. Ultra-sound scanning of the liver may be used to reveal signs of liver congestion. In fact Henriksson et al reported that the width of the major right hepatic vein was a reliable indication of right heart failure.

With the aim of evaluating the sensitivity of different parameters, an attempt was made to study changes in signs of left and right heart failure at clinical examination, at chest x-ray and at ultrasound investigation of the liver before and after therapy.

MATERIALS AND METHODS

Ten consequtive patients arriving at the emergency department due to severe cardiac decompensation and systemic congestion were studied.The following clinical signs were recorded:

Weight(kg)
Pitting oedema: 0=no oedema, 1=ankle oedema, 2=leg oedema, but not above the knee and 3=oedema above the knee.
Pulmonary rales: 0=no rales, 1=basal rales, 2=rales to the apex of the scapulae, 3=rales to the bases of the scapulae.
Jugular vein(1): Its filling was estimated with the patient recumbent at a 30 degree position.More than 2 cm above the upper central edge of the manubrium sterni was estimated as pathological.The height of the filling (cm) was recorded.
Kussmauls sign(2): The jugular vein was observed at inspiration. 0=decreased filling, 1=no change in filling, 2=increased filling.
Hepatojugular reflux (3): A smooth but firm pressure was applied over the liver during 10 seconds.The jugular filling was observed: 0=no change in filling, 1=increased distension of the jugular vein but no increased filling in height, 2=increased filling in height which was estimated in cm when possible.
Liver: The following criteria was used to estimate whether it was increased: palpable more than one finger below the right arcus. Palpable more than 40% of the distance between the apex of the processus xiphoidues and the umbilicus. In this location the liver is best palpated as no abdominal muscular tissue covers it. Based on these two estimates it was decided whether the liver was enlarged or not.0=liver size within the reference limit, 1=enlarged, 2=painful liver at palpation.

Chest x-ray: Films were taken in upright anterior-posterior and lateral views and when possible, lateral decubitus views for estimation of pleural effusion. The films were then evaluated according to Milne et al for an estimation of vascular pedicle width (VPW) and v azygos width. VPW is formed by the leash of vessels extending from the thoracic inlet to the heart. Milne et al have shown that a change in VPW is closely correlated to the change in total blood volume and that the width of v azygos correlates with mean right atrial pressure. Heart size was estimated according in the upright position by routine methods. The presence of redistribution of pulmonary blood flow, interstitial oedema, and Kerley B-lines were evaluated.

Ultrasound: The major right hepatic vein was measured in accordance to Henriksson et al , at the confluence with v.cava inferior. A Technicare Autosector 1, 3.5 mhz transducer was used.

Statistics: Differences before and after treatment were tested by two-tailed paired Students t-test.

RESULTS

The age of the patients was 74±9 (58-89) years, 3 being female. Table 1 gives the results. In all patients treatment resulted in disappearence of pathological jugular filling, leg oedema and liver enlargement and a decrease in weight and heart size. Pleural effusion decreased in 7/10 and disappeared in 5/10 patients. Hepatojugular reflux and an inspiratory increase of the jugular filling disappeared in about half of the patients.The per cent change was high and in the range of 80 to 85% for the jugular filling, leg oedema and liver score. In respect of the jugular filling changes were in the range of centimeters. Heart size and weight changed in the 10 per cent range. Interestingly, the decrese in absolute heart size was more sensitive (higher P value) than the change in relative heart size.

Sizes of the superior vena cava or the vascular pedicle and the azygos vein on chest x-ray were in the range of 28-43 mm, 63-78 mm and 7-16 mm, respectively. The major right hepatic vein as studied by ultrasound ranged between 6 and 18 mm.These three vessels deacreased their size between 8 and 25% with the highest change observed for the azygos vein. Compared to the upper reference value for these vessels, the change was 40 to 100 per cent. Decreased size of the azygos vein was observed in all patients while the major right hepatic vein decreased only in 75% of the investigated patients.

Rales and x-ray signs of interstitial oedema disappeared in all patients. Signs of redistribution remained in half of the patients.Kerley lines when present disappeared with treatment.

DISCUSSION

Significant decreases were observed in all parameters tested (table 1). The significances were larger for the clinical compared to the x-ray and ultrasound parameters with the possible exception of the width of the vena azygos. In respect of normality

Table 1. Mean±standard deviation of initial value or fraction of patients with an initial prescence of a diagnostic sign, per cent decrease, significance of the decrease (P-value) and the fraction of patients with decrease or normalisation of the 17 parameters measured.

	Initial value	% decrease	P-value	Number of patients
Jugular filling, cm	4.8±2.2	81	<0.0002	10/10
Weight, kg	78±17	12	<0.001	10/10
Leg oedema, score	1.9±0.9	84	<0.001	10/10
Liver, score	1.6±0.5	88	<0.0001	10/10
Hepatojugular reflux, score	1.0±0.5	60	<0.01	7/10
Inspiratory jugular filling, score	1.3±0.7	69	<0.05	5/10
Rales, score	2.2±0.8	95	<0.0002	10/10
Heart size, ml				
absolute	1621±444	14	<0.005	7/8
relative	834±141	10	<0.01	7/8
Pleural effusion, cm	1.3±0.8	54	<0.01	7/10
Vascular pedicle, mm	75±9	9	<0.01	7/8
Superior vena cava, mm	37±8	14	<0.05	7/9
Azygos vein, mm	12±3	25	<0.005	10/10
Right hepatic vein, mm	10±4	20	<0.05	6/8
Interstitial oedema	9/9	100	<0.00001	9/9
Kerley lines	5/9	100	<0.00001	5/9
Redistribution	10/10	40	<0.05	4/10

or not, however, a comparison to reference values must be made. The prescence of clinical signs indicate abnormality and their absence no sign of abnormality. The reference values of the laboratory parameters relates to values found in a group representing the normal population.

Upper reference limits for the vascular pedicle and the azygos vein have been defined by Milne et al (4) and for the major right hepatic vein by Henriksson et al. Values reported are 58, 8 and 9 mm respectively.The observer variation in the estimation of vessels at chest x-ray was reported to be 2±2%. The ultrasound estimate of the major right hepatic vein is probably larger. The vascular pedicle and thus the superior vena cava was pathologically wide in all patients at onset of therapy and remained so after normalisation of clinical parameters except in one patient. The azygos vein was likewise pathological in all patients at onset of study.Normalisation was obtained in three patients. The major right hepatic vein was within normal limits in 4/8 patients at onset of study and in 6/8 at the end of the study. The mean decreases in the width of the 3 vessels studied were 7, 3 and 2 mm respectively.In view of an observer variation (Milne et al) at 2+2% the changes during therapy in this set of patients with severe systemic heart decompensation are minimal and must be considered insensitive.

The tendency observed when comparing values before and after treatment, i.e. that clinical signs of right heart failure are more sensitive parameters than those supplied by the laboratory is thus confirmed when the comparison is made to reference values. In respect of right heart failure the laboratory parameters appear not to have a diagnostic but well a confirmatory value. Such a conclusion is at variance with the report by Henriksson et al. They reported efficient separation between the reference group and patients with severe venous congestion in respect of the major hepatic vein. Maybe different degrees of vascular fibrosis affect the compliance of the veins, including the hepatic vein, where also hepatic fibrosis may be of importance.

The preference of clinical methods for the diagnosis of right heart failure should be compared to the high degree of covariation between the presence of rales and interstitial oedema. Chest x-ray therefore appears to contribute with useful information and confirmation in respect of left heart failure. However, signs of redistribution did not covary with the prescence of rales, possibly due to the fact that this sign indicates slight degrees of left ventricular failure where auscultation may be non-

contributory.

The patient selection in this series was based on undisputable clinical signs of systemic heart decompensation. Thus the prevalence of the disease was 100 %. In groups with lower prevalence it is not excluded that chest x-ray methods may have better predictive values than clinical methods.

In conclusion,left ventricular failure, as diagnosed clinically finds its useful confirmation in traditional findings at chest x-ray.Right ventricular failure on the other hand appears to be best diagnosed and monitored by simple clinical methods.

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