Introduction
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Malnutrition is defined as “a cellular imbalance between the supply of nutrients and energy, and the body’s demand for them, to ensure growth and maintenance of specific functions” (WHO).

Protein energy malnutrition (PEM) is the most lethal form of malnutrition affecting children and affects every 4th child worldwide (WHO).

The state of world children, 2004 describes the prevalence of children who are underweight, stunted and wasted to be 47%, 46% and 16% respectively. This explains the high morbidity burden in our society.

PEM is the major health and nutritional problem in India with its high incidence in the preschool children. About 6,600 under five children die everyday of malnutrition in India. Protein energy malnutrition (PEM) accounts for death in 7% cases and is underlying cause of death in 46% cases below 5 years of age. As per recent National Family Health Survey, the most common age of PEM is between 6 months and 2 years and around 50-60% of children are malnourished by 2 years, stunting was a major problem and was observed in almost half of children.

Protein energy malnutrition (according to its severity) not only has a profound effect on external appearance of child but also has its impact on various organ systems of the body such as haematopoietic and immune system, thymolymphatic system, gastrointestinal system, musculoskeletal, cardiovascular system etc.
Cardiovascular system bears significant changes in patients with protein energy malnutrition which increases according to the severity of PEM (Singh GR et al, 1989).

Inspite of nutrition therapeutics, severely malnourished children have high mortality rate. About 50% of 10 million children’s death in developing countries is contributed by PEM. The cause of death is not apparent always, even after detailed investigation and most of such death are sudden and unexpected. This instantaneous nature strongly suggests cardiac cause. (Smythe PM et al, 1962).

Earlier concept that heart is spared in malnutrition is shown to be incorrect as stated by Singh GR et al (1989) who observed various cardiovascular changes in children with severe grades of protein energy malnutrition.

Peter B et al (1989) studied the pathophysiology of involvement of heart in protein energy malnutrition and explained that cardiovascular system involvement can be due to various causes such as- (1) Myocardial atrophy secondary to inadequate protein and energy intake (2) Due to alteration of metabolic rate in PEM and thus decreasing the systemic demand for cardiac output and (3) Secondary to reduced blood volume and anaemia in children with PEM.

John G Webb et al (1986) quoted that inadequate intake of protein and energy results in proportional loss of skeletal as well as myocardial muscle. As myocardial mass decreases, it leads to decrease in cardiac contractility and the ability to generate cardiac output. However, various compensatory mechanisms comes into play, which maintains cardiac
index. This cardiac debility in turn results in poor nutrition, which viciously produce clinically significant myocardial atrophy.

Symthe et al (1962) in autopsy study of protein energy malnourished children found that the heart is underweight, looks pale or brown, flabby with slender muscle fibers and the heart walls, especially of atria were extremely thin.

Gopalan et al (1955) and Wharton et al (1962) described the histological changes in myocardial tissue in PEM such as interstitial edema and degenerative changes. Changes are most markedly observed subendocardially, especially in papillary muscles and left ventricle (Whatson et al 1969) and in conducting tissue (Smith BA et al 1962).

Brooke et al (1973) described the state of low cardiac output. He found clinical signs such as cold extremities sometimes cyanosed, decreased heart rate and poor pulse pressure in patient with severe PEM and concluded that there is an overall decrease of peripheral blood flow in severe PEM.

Smythe et al (1962) observed the radiological changes and found that heart is abnormally small which is designated as microcardia and further stated that cardiothoracic ratio which is used as an index of heart size is considerably reduced in PEM. Smythe et al (1962) recorded the electrocardiographic changes in cases of severe malnutrition such as sinus arrhythmia, decrease in amplitude of P wave, QRS complex and T wave in all leads and also prolongation of QT interval, ST segment changes like concave bending and T wave change such as inversion.
Bergman et al (1978) and Hemymssfield et al (1978) studied the echocardiographic changes in protein energy malnourished children and revealed – (1) Decreased chamber size and dimension (2) Decreased left ventricular end systolic and end diastolic volume (3) Decreased contractility and cardiac output and (4) Decreased left ventricular mass.

Large amount of valuable work was done in past decades and is still going on in various parts of world to found out the effect of malnutrition on heart. Important contributions in the above field were also given by Kothari SS et al (1992), Olowanyo MT et al (1993), Phornphatkul C et al (1994), olowanyo MT et al (1995), Ocal B et al (2001), Florea VG et al (2002), Oliovares JL et al (2005) and most recent work by EL Sayed HL et al (2006) in the field of understanding heart involvement in malnutrition by using Clinical, ECG, and Echocardiographic methods. Their work had cleared the previous queries and confirmed that protein energy malnutrition has a profound effect on cardiovascular system.

The present study was undertaken to correlate the clinical radiological, electrocardiographic and echocardiographic changes on heart in cases of severe protein energy malnutrition.