

SUMMARY AND CONCLUSION

Cardiovascular complications are the most frequent cause of death in children with end-stage renal disease.

Measuring the serum CTnI levels in uremic patients can give an idea about the diagnosis of even a small area of myocardial cell injury as serum CTnI is now one of the most accurate available biochemical marker of myocardial damage .

This study was conducted on 40 patients with CRF (20 on regular HD and 20 on conservative treatment), their age ranged from 5-15 years (27 male and 13 female) of those attending The Pediatric Nephrology and Dialysis Unit, Children Hospital, Ain- Shams University.

The kidney statue was evaluated by the routine workup including complete renal profile (bl.urea, s.creatinine, serum Na, serum Ca, serum K, serum phosphorus) and complete blood count.

Cardiovascular status was evaluated by the use of chest X-ray, ECG and echocardiographic examination.

Myocardial damage was evaluated by measuring the serum cardiac troponin-I by Immulyte as an indicator of myocardial cell injury in patients with ESRD.

Serum CTnI level was increased in 17 patients (24.5%) out of 40 patients with ESRD on both heamodialysis and conservative treatment .

Chest X-ray showed cardiomegaly in 4 patients (10%) as cardiothoracic ratio more than 50% .

ECG showed 8patients (20%) with left ventricular hypertrophy , only one patient (2.5%) with left atrial dilatation , no criteria for right artial or ventricular dilatation,no arrhythmia no change in QRS complex and there was 7 patients (17.5%) with ischemic criteria (inverted T-wave)

Echocardiographic examination showed left ventricular hypertrophy and cardiac dilation.

Left atrial diameter was increased in 6 patients (15%), right ventricular diameter was increased in one patient (2.5%), interventricular

septum thickness was found in 21 patients (50.2%), left ventricular posterior wall thickness was found in 34 patients (85%), left ventricular end diastolic diameter was increased in 3 patients (7.5%) and left ventricular mass index was increased in 22 patients (55%).

Left ventricular systolic function was impaired in 14 patients (35%) with low EF and FS.

Mitral regurgitation was observed in 6 cases (15%), aortic regurgitation in 4 cases (10%) and tricuspid regurgitation in 7 cases (17.5%) this is related to cardiac dilatation and septal hypertrophy.

Pericardial effusion was found in 3 cases (7.5%) due to volume overload and metabolic changes in uremic patients.

From all the above, we can conclude that:

Children with CRF had systemic hypertension, cardiac chamber dilatation and ventricular hypertrophy, particularly affecting the interventricular septum. Left ventricular hypertrophy acts as a compensatory mechanism to maintain left ventricular systolic function as represented by EF and FS.

These findings are more marked in children with CRF on conservative treatment in comparison to those on regular hemodialysis.

Echocardiography is superior to chest X-ray and ECG in evaluating CRF children with heart failure and myocardial damage, but it discovers these manifestations late after development of uremic cardiomyopathy.

ECG adds an important diagnostic point in diagnosis of myocardial ischemia.

Children with CRF showed manifestations of cardiac affection with increased serum CTnI levels.

There is a positive correlation between serum CTnI and myocardial damage as detected clinically and by investigations (Echo, ECG and chest X-ray).

Cardiac troponins, specially CTnI, has a sensitive diagnostic and predictive value for myocardial***