SUMMARY AND CONCLUSION

The etiology of growth failure in children with CHD appears to be multifactorial and not entirely accounted for by haemodynamic disturbances or nutrition alone.

The aim of the present study was to evaluate the role of IGF-1 and IGFBP3 in congenital heart diseases.

This study was conducted on 30 children diagnosed as congenital heart disease, attending cardiology department clinic in Abo El-Rish Hospitals.

The study were divided into 3 groups:

Group I : Control group: 15 patients with an age (3.1 ± 1.9 years), 7 males and 8 females.

Group II : Acyanotic group : 15 patients with an age (2.16 ±1.1 years), 9 males and 6 females.

Group III : Cyanotic group : 15 patients with an age (2.87± 1.7 years), 10 males and 5 females.

The patients were compared to a control group which included 15 well nourished apparently healthy children.

All the studied groups were subjected to :

- Full history taking with special emphasis on the presence or absence of cyanosis, feeding habits, caloric intake and presence or absence of complications.
- Thorough physical examination with special emphasis on physical appearance and complete cardiac examination including inspection, palpation, pericardial percussion and auscultation.

- Anthropometric measures (weight, height, BMI).

Serum IGF-1 and IGFBP3 were assessed in all patients and controls.

In the present work, both cyanotic and acyanotic groups were stunted and underweight, with more severe affection of cyanotic patients compared to acyanotic patients and the controls.

The results of this study revealed that serum IGF-1 level was unrelated to age or sex in the cyanotic group as well as the control group.

There was non significant correlation between serum IGF-1 level and age in acyanotic children.

The present work showed that serum level of IGF-1 was statistically significantly lower in the cyanotic and acyanotic groups than serum IGF-1 level of the controls ($P < 0.001$). There was statistically significant difference between cyanotic and acyanotic groups regarding the serum level of IGF-1 ($P < 0.05$).

Cyanotic patients were several malnourished with statistically significantly lower serum IGF-I level in the malnourished cyanotic group ($P < 0.05$).

We observed that there was statistically significant difference in serum IGFBP3 between patients with cyanotic and acyanotic CHD ($P < 0.001$). In both groups, serum IGFBP3 was lower than the controls.

There was a statistically significant positive correlation between IGFBP3 and BMI in cyanotic patients.

In summation, the present study refers growth failure associated with the small size of CHD patients to several factors including endocrinal factors, chronic hypoxia, decreased energy intake and increased energy expenditure.

CONCLUSIONS

1- Cyanotic congenital heart diseases in children caused more pronounced growth retardation in comparison with acyanotic congenital heart diseases.

2- Children with CHD had lower IGF-1 levels than controls and the malnourished cyanotic children had the lowest IGF-I levels.

3- The lower IGF-1 levels in children with CHD might be related to compromised nutrition from hypermetabolism even without overt evidence of heart failure.

4- Chronic hypoxia had a direct effect to reduce serum IGF-1 concentration and this might be a cause of the increased growth failure in patients with cyanotic CHD.

5- Chronic hypoxia might contribute to the feeding problem in cardiac patients.

RECOMMENDATIONS

1- Further research into the mechanisms of growth failure in CHD from a biochemical level is needed to guide the possible management options.

2- Further studies are required to determine the role of increased energy expenditure and its correlation to serum leptin level in infants with CHD.

3- Factors other than those studied maybe important in determining circulating IGF-1 levels in infants with CHD.

4- Factors that lead to intrauterine growth retardation for patients with CHD requires further elucidation and represent an important area of research.

5- A systematic approach must be used to identify specific reasons for this failure to thrive to develop a strategy for a structured feeding program and to educate parents as to the best ways to feed their children.

6- Further research into the role of recombinant growth hormone to treat those children who continue to exhibit growth delay after surgical correction.