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Anthracycline induced early cardiotoxicity in a very young Omani patient with Acute Myeloid Leukemia

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Background: Anthracycline-cardiomyopathy is of concern in children treated for acute myeloid leukemia (AML) as it may be progressive and fatal. It can present as early cardiac dysfunction with onset during or after chemotherapy.

Objective: We aim to highlight the risk of early-onset cardiotoxicity with a low cumulative anthracycline dose in a child with AML with concurrent sepsis.

Material and Methods: Two-year old Omani boy with AML-M7 with complex karyotype, baseline echocardiography anatomically normal heart with left ventricular ejection fraction (LVEF) 55-60% was planned for chemotherapy. He was initiated on antibiotics (febrile neutropenia guidelines) for high grade fever. Septic work-up was unremarkable, chemotherapy was initiated in second week. Repeat echocardiography revealed no vegetation and an LVEF of 55%. By end of second week, chemotherapy was continued. After second dose of daunorubicin, patient developed tachycardia, tachypnea, respiratory distress, desaturation with a brief seizure terminating in bradycardia and hypotension requiring resuscitation and ventilation. He had LVEF of 30%, was hypertensive, milrinone, dobutamine were initiated, later shifted to frusemide, spironolactone and captopril; digoxin was added as cardiac function remained depressed. Remainder of daunorubicin was skipped. Post-induction BMA revealed remission. Patient received three more courses of anthracycline-free chemotherapy. Prior to course three chemotherapy, patient had another cardiac arrest. Echocardiography one month later revealed global dyskinesia and LVEF of 40-45%. Patient is on regular cardiac monitoring, currently on frusemide, spironolactone, captopril and digoxin at six months follow-up.

Results: Refer Table 1

Conclusion: We conclude that in presence of other known risk factors for cardiac dysfunction like severe sepsis, there is probably no risk-free dose of anthracycline; decline in cardiac function may occur early in therapy even after a small cumulative dose requiring close monitoring of cardiac status during chemotherapy. Association of other risk factors need to be explored by evaluation of larger

cohort of such patients.

| | Baseline | For vegetation | 1st arrest | Pre 2nd arrest | 2nd arrest | Follow-up at 3 months | Follow- up at 6 months |
|-------|----------|----------------|------------|----------------|------------|-----------------------|------------------------|
| LVIDd | 42 | 42 | 50 | 50 | 48 | 45 | 45 |
| LVIDs | 30 | 31 | 42 | 39 | 38 | 36 | 35 |
| FS | 29 | 28 | 14 | 23 | 19 | 21 | 22 |
| EF | 56 | 55 | 30 | 45 | 37 | 43 | 45 |
| MR | Trivial | Trivial | Mild+ | Mild | Mild+ | Trivial | Trivial |
| AR | Nil | Nil | Mild | Mild | Mild | Trivial | Trivial |

Biography

Surekha Tony graduated in medicine in 1995 and specialized in pediatrics with training in hematology in Bangalore, India. She is staff pediatric haemato-oncologist in the Hematology/Oncology Unit at the Department of Child Health, Sultan Qaboos University Hospital Muscat Oman with an active clinical practice for patients with benign and malignant hematological disorders including bone marrow transplantation. She has particular interest in thalassemia and has worked as principal and co-investigator in clinical trials. She has authored and co-authored numerous abstracts and manuscripts and has been active as invited speaker at national and international conferences. She is actively involved as trainer and examiner for junior-senior clerks and pediatric residents. She is a member of the Oman Medical Association, Oman Society of Hematology and the Oman Medical Specialty Board.

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