The clinical presentation of Type 2 Diabetes in children and adolescents in Western Australia.

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Background: The prevalence of youth Type 2 Diabetes (T2D) is increasing in Australia and worldwide, attributable to the rising rates of childhood obesity[1]. Children with T2D experience faster progression to micro and macrovascular complications compared to adults[2].

Aim: We aim to describe the clinical features at presentation of children with T2D in a tertiary paediatric hospital with a large, ethnically diverse population.

Methods: Retrospective chart review of 229 patients aged <16 years, diagnosed with T2D at Perth Children's Hospital from 1989 to 2019. Results were expressed as descriptive statistics.

Results: At presentation, the mean ±SD (range) age was 13.2 ±1.94 (6.8-16) years. 58% were female, 54% were Indigenous, and 79% had a family history of T2D. 34.5% had classical diabetes symptoms, 3.8% presented emergently, 14.6% had a diabetes related illness, and 47.1% were asymptomatic; diagnosed either via screening (27.6%) or incidentally (13.6%).
62% had acanthosis nigricans, and 11.3% of females had polycystic ovarian syndrome. 78.3% were obese, with a mean ±SD (range) BMI z-score of 2.01 ±0.69 (1.59-3.10). Amongst those screened, a proportion had microalbuminuria (38/178, 21.3%), dyslipidemia (116/163, 71.2%), hypertension (13/229, 5.7%), or deranged LFTs (68/91, 74.7%). 31.9% had psycho-social issues. Pharmacological therapy, initiated in 59.8% of children, included metformin (25.8%), insulin (13.5%) or both (20.5%).

Referral sources included general practitioners (45.8%); community based paediatric services (13.6%), regional hospitals (15.8%), tertiary EDs (3.2%), tertiary paediatric services (19.5%), or others (2.1%).

Conclusion: Children with T2D are more likely to be female, obese, Indigenous and have a strong family history of T2D. Most children lack the classical diabetes symptoms at presentation with many detected via screening. Metabolic, cardiovascular, and mental health comorbidities are common and should be screened for in all children at diagnosis.

References

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