Of any pituitary dysfunction following brain injury, growth hormone (GH) deficiency (GHD) is the most prevalent. The cut-off point for defining GHD has been placed at 7 ng/mL, representing optimum test performance. We hypothesised this cut-off may be set too low for genetically taller children with an acquired brain injury, notably brain tumours, who demonstrate severe growth failure but repeatedly fail to meet diagnostic thresholds for GH replacement until several centiles have been crossed downward over time; this treatment delay may ultimately compromise metabolic status and post-injury wellbeing.

We reviewed the possibility that this cut-off, and its undifferentiated applicability to a broad variety of taller children with clear longitudinal growth failure, requires re-consideration for this cohort, according to their midparental height (MPH) and body mass index (BMI).

We reviewed retrospectively the parental heights, longitudinal growth records and charts of 30 children diagnosed with a brain tumour (47) or traumatic brain injury (3) and noted, at intervals, height, weight, Tanner stage and peak GH (pkGH) to dynamic provocation at first onset of growth failure and at any subsequent re-test for persistent growth failure. BMI, BMI SDS, height SDS and midparental target height SDS (MPHSDS) were calculated.

Patients were categorised into three respective groups according to how many times (once, twice or thrice) they required testing for persistent growth failure before meeting diagnostic criteria for GH deficiency (GHD) for the first time. Data are shown as Mean and 95% CI and one-way ANOVA was used for between cohort comparisons.

At the time of this patient's brain tumour diagnosis, his height SDS was 0.39 and his midparental target height SDS was -0.41. At his first GH test, 3.52 years later, his growth had deteriorated to a height SDS of -4.76 and he remained prepubertal.

However, with a pkGH of 9.2 ng/mL, he did not yet meet diagnostic criteria for GHD. Neither did he at his second test (pkGH 7.7 ng/mL) 0.92 years later, when height SDS was still -0.77. Even at his third GH test (0.98 years later), he did not meet criteria for GHD (pkGH 12.7ng/mL). His height SDS was still -0.77.

CONCLUSIONS

Our data before GH treatment do not suggest that a midparental target height above average or an increment of BMI-SDS impairs diagnostic validity of current pkGH. Instead this may represent a physiological 'catch-down growth' towards MPHSDS, not requiring immediate GH treatment.

Severe GHD may ensue, which requires continuous monitoring after recanalization into the mid-parental centile and at the onset of puberty. A post-treatment review is still required to ensure that these children achieve their innate growth potential without compromise.