lowed by oral prednisolone [OP], oral cyclophosphamide [2 mg/kg per day for 2 to 3 months], and dipyridamole). The other 23 patients with <50% crescent formation were given methylprednisolone followed by OP and dipyridamole. The patients with grade 3 or 4 disease were given OP and dipyridamole. Those with grade 1 or 2 disease were not given any immunosuppressive agent. During the follow-up period (mean: 30 ± 3.5 months; range: 12–96 months), 23 patients with grade 1, 38 patients with grade 2, 2 patients with grade 3, 8 patients with grade 4, and 21 patients with grade 5 disease showed complete remission (59%). Of the 5 patients with extensive fibrosis shown by renal biopsy, 2 (1%) had persistent nephropathy and 3 (2%) developed end-stage renal failure. The remaining 59 patients showed near-complete recovery with minimal urinary abnormalities (38%).

CONCLUSIONS: Although initial presentation of renal involvement determines the prognosis for those with HSN, intensive treatment with triple therapy seems to be effective for severe renal disease, especially if started before the development of fibrotic changes in crescents and tubulointerstitial tissue.

Neurology

THYROID FUNCTION IN EPILEPTIC CHILDREN TREATED WITH SODIUM-VALPROATE MONOTHERAPY: A PROSPECTIVE LONG-TERM STUDY

Submitted by Achilleas Attilakos

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INTRODUCTION: Sodium valproate (VPA) is widely used for the treatment of partial and generalized epilepsy in childhood and adolescence. The results of studies that have evaluated the effect of VPA monotherapy on thyroid function in children are controversial.

OBJECTIVE: The aim of this study was to investigate, prospectively, whether treatment with VPA has an effect on serum thyroid hormone concentrations in epileptic children.

METHODS: Serum levels of triiodothyronine, thyroxine, free thyroxine, and thyrotropin were determined in 30 epileptic children (aged 2 to 14 years [mean ± SD: 9.10 ± 3.74 years]) before and after 6, 12, and 24 months of VPA monotherapy.

RESULTS: Serum levels of thyroxine and free thyroxine were significantly decreased after 6 (P = .000 and .000, respectively), 12 (P = .000 and .015, respectively), and 24 (P = .000 and .003, respectively) months of treatment with VPA, whereas serum levels of triiodothyronine were significantly decreased only after 24 months of treatment (P = .043). Serum levels of thyrotropin were significantly increased after 6 (P = .000), 12 (P = .000), and 24 (P = .000) months of treatment with VPA. Thirteen children (43.3%) had thyrotropin values higher than the normal-range maximum after 6, 12, and 24 months of VPA monotherapy. Serum VPA concentrations remained within the therapeutic range during the period of study.

CONCLUSIONS: Our results showed that VPA monotherapy in childhood may cause early and persistent alterations in thyroid function, which suggests a need for early and careful monitoring of serum thyroid hormone concentrations in epileptic children who receive VPA.

CLINICAL MARKERS THAT ENHANCE ETIOLOGIC YIELD IN GLOBAL DEVELOPMENTAL DELAY

Submitted by B. H. Y. Chung

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INTRODUCTION: Etiology remains unknown in 30% to 50% cases of children with global developmental delay (GDD). A selective approach has been recommended to increase etiologic yield.

OBJECTIVE: Our aim was to identify clinical markers that enhance diagnostic yield of GDD at initial assessment.

METHODS: The charts of all patients with GDD (N = 577) followed up at the Duchess of Kent Child Assessment Centre were reviewed. GDD was defined as significant delay (<2 SD) in ≥2 developmental domains. Nine clinical items at initial assessment (gender, severity of delay, parental consanguinity, family history, behavioral disturbance, head size, facial dysmorphism, malformations, and neurologic deficits) were correlated with the likelihood of finding an etiology for GDD.

RESULTS: A significant threshold effect was found between mild and moderate GDD (positive likelihood ratio (LR^+): 1.9; negative likelihood ratio [LR^-]: 0.72). Other items that significantly affected diagnostic yield were (1) female gender (LR^+: 1.62; LR^-: 0.79), (2) behavioral trait (LR^+: 0.24; LR^-: 1.67), (3) microcephaly (LR^+: 2.78; LR^-: 0.79), (4) presence of facial dysmorphism (LR^+: 2.65; LR^-: 0.65), (5) malformation (LR^+: 1.49; LR^-: 0.50), and (6) neurologic deficits (LR^+: 2.86; LR^-: 0.32). A dose-response relationship was found between LR^+ and the number of facial dysmorphisms and malformations.

CONCLUSIONS: Most checklists used for GDD are syndrome specific (eg, fragile X syndrome checklists). These 7 clinical markers are useful in the initial assessment,

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