اثرات بیماریهای مزمن بر تکامل روانشناختی کودکان و نوجوانان

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DIABETES

The human brain undergoes unique dynamic structural and functional changes during childhood and requires continuous delivery of glucose for brain function and growth.

Whether dysglycemia in young children irreversibly compromises key neurodevelopmental processes is unknown.

A growing body of evidence suggests that the *brain is a target for diabetes complications.*

Reduced total, gray, and white matter volumes have been observed in type 1 diabetes particularly in ***temporal and parieto-occipital*** regions.

Regional brain differences have also been linked to:

- severe hypoglycemia,
- higher lifetime HbA1c,
- disease duration,
- severity of microangiopathy

Within a younger type 1 diabetes cohort (mean age 12.6 years), found that greater lifetime hyperglycemia was associated with decreased total gray and white matter volume **in the occipital lobe**.

In older children and adults with type 1 diabetes, they have suggested that effects of **hyper- and hypoglycemic** exposure on brain structure are widely distributed

Frontal and ***parietal-occipital cortical regions*** appear most vulnerable, particularly in individuals with early disease onset

The longest longitudinal study of brain structure and cognition using modern diabetes technology to assess glycemia in a group of young children with type 1 diabetes compared with an age-matched control group without diabetes(4to,10 years of age at study entry) followed at four time points as they grew and progressed into puberty.

They observed significant differences in FSIQ and VIQ and lower total, gray, and white matter volumes in the diabetes group. Differences that persist and, in the case of structural brain measures, increase over time.

These changes are strongly related to short-term and long-term indices of *hyperglycemia* in the diabetes group.

Whether these changes are reversible with scrupulous better control of glucose in these young children requires further study.

Young children with type 1 diabetes (T1D) may be at particularly **high risk of cognitive decline** following diabetic ketoacidosis (DKA).

A single DKA episode is associated with lower IQ scores soon after exposure to DKA in young children.

DKA is associated with *cognitive alterations* in children with type 1 diabetes, including those with newly diagnosed type 1 diabetes.

The neurocognitive effects of DKA in children with preexisting type 1 diabetes should be evaluated in the context of additional variables, including repeated DKA exposure and glycemic control.

These results emphasize the importance of prevention of DKA in children with known type 1 diabetes and of prompt diagnosis during new onset of type 1 diabetes before the development of DKA

In conclusion, the brain is a target of diabetes complications, even in young children.

These data support the lowering of glycemic targets in children; acceptance of higher-than-normal blood sugars as adequate metabolic control in very young children needs to be revisited

THALASSEMIA SYNDROMES

Thalassemia syndromes are the most prevalent monogenic hemoglobinopathy in the world.

In **Iran**, thalassemia is a public health problem because this country has been located on the thalassemia belt.

In recent decades, considering that the life expectancy of patients with thalassemia has dramatically improved, some unrecognized complications have emerged in these individuals.

Patients usually present with severe anemia, jaundice, organomegaly, growth retardation, and skeletal abnormalities.

Therefore, they require **chronic blood transfusion**, which comes with the cost of iron overload and **chronic iron deposition** in different body organs.

Neurological complications and **cognitive dysfunction** have been suggested in βTM patients, either due to *the disease* or *its treatment*.

Many **risk factors** were reported, including:

- chronic hypoxia,
- iron deposition in the nervous system due to frequent blood transfusions,
- silent thromboembolism,
- neurotoxicity of Deferoxamine

Furthermore, frequent school absences, frequent hospitalizations, and physical and social restrictions also lead to cognitive dysfunction

- This neurological involvement in βTM patients is **primarily silent**, with subclinical manifestations that can only be detected by **cognitive assessment tests**

- **MRI** methods can be used to study the potential association of brain iron deposition and cognitive function in patients with β-TM.
- *Quantitative susceptibility mapping* (QSM) provides a novel, noninvasive, and quantitative method to analyze brain iron.
- Going forward, it will be important to determine to what extent and how brain iron overload affects cognitive function in patients with β-TM by combing MRI techniques, neuropsychological tests, and neuro-electrophysiological methods.

IQ correlates with:

- age at diagnosis
- average annual pre-transfusion hemoglobin.

This highlights the importance of early diagnosis and maintenance of satisfactory hemoglobin levels.

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