

In parallel, recent studies have shown that maternal or infantile atopic diseases are correlated with ASD, and the hypothesis that inflammation of the brain may be involved in the pathogenesis of ASD is under investigation.⁵

On the one hand these findings clarify biological aspects of autism and on the other hand support the concept of autistic spectrum and of a continuum from normal to pathologic behavior. This is reflected in the new *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*, classification in which the term “autism spectrum disorder” comes to replace 4 previously separate disorders, which are now considered as expressions of the same disorder in different degrees of severity.

The spectrum/continuum model, supported by findings that child functional characteristics seem to explain child and family outcomes better than the diagnosis itself,⁶ has paved the way for autistic behavior not being perceived as a nonmodifiable permanent disability. To the contrary, autism is a dynamic process demanding personalized interventions whose implementation should start as early as possible, most appropriately during infancy.

Helen Lazaratou, MD
Marina Economou, MD
Dimitris Dikeos, MD

First Department of Psychiatry
 Medical School
 National and Kapodistrian University of Athens
 Athens, Greece

References

1. Pierce K, Courchesne E, Bacon E. To screen or not to screen universally for autism is not the question: why the task force got it wrong. *J Pediatr* 2016;176:182-94.
2. Kanner L. Autistic disturbances of affective contact. *Nervous Child* 1943;2:217-50.
3. Mevel K, Fransson P. The functional brain connectome of the child and autism spectrum disorders. *Acta Paediatr* 2016;105:1025-35.
4. An JY, Claudianos C. Genetic heterogeneity in autism: from single gene to a pathway perspective. *Neurosci Biobehav Rev* 2016;68:442-53.
5. Theoharides TC, Tsilioni I, Patel AB, Doyle R. Atopic diseases and inflammation of the brain in the pathogenesis of autism spectrum disorders. *Transl Psychiatry* 2016;6:e844.
6. Miller A, Shen J, Mâsse LC. Child functional characteristics explain child and family outcomes better than diagnosis: population-based study of children with autism or other neurodevelopmental disorders/disabilities. *Health Rep* 2016;27:9-18.

Safety and efficacy of ferric carboxymaltose in children and adolescents with iron deficiency anemia



To the Editor:

Powers et al¹ recently reported the use of intravenous ferric carboxymaltose (FCM) in children with iron deficiency anemia

(IDA) refractory to oral iron therapy. We would like to share our own experience that is in agreement with the published report. Over the last 2 years, we have treated with FCM 15 children and adolescents (10 females and 5 males; median age 12 years; range 8-17.9) with IDA. A total of 27 FCM infusions (Ferinject, Vifor SA, France) were administered, that is, 26 infusions of 500 mg and 1 infusion of 1000 mg in an adolescent who was of an adult size. FCM was diluted in normal saline to a final concentration ≥ 2 mg/mL, and administered via an infusion pump over 30 minutes to 2 hours without a test dose or any premedication. Thirteen patients had failed previous oral iron therapy, and all had pretreatment serum ferritin < 12 ng/mL. Five patients had IDA owing to heavy menstrual bleeding, 2 had Crohn's disease, 2 had nutritional IDA (a patient with cerebral palsy and another with severe intellectual disability), 1 each had short gut syndrome, duodenal ulcer, and ulcerative colitis, and 3 patients had IDA of undetermined etiology. The median pretreatment hemoglobin was 73 g/L (range 50-112), and the median post-treatment hemoglobin was 126 g/L (range 80-148) at > 4 weeks after the initial FCM infusion. No case of urticaria, pruritus, or wheezing occurred, and no delayed reactions were observed. The only side effect was a painless extravasation in 1 female that led to mild iron staining of the forearm. We conclude that intravenous FCM seems to be safe and extremely effective, and should be used when oral iron therapy fails, a quick increase in hemoglobin or replacement of the total iron deficit at a single or few settings is desirable in children and adolescents with IDA of diverse etiologies.²

Elpis Mantadakis, MD

Department of Pediatrics
 Pediatric Hematology/Oncology Unit
 University General Hospital of Evros
 Alexandroupolis, Thrace, Greece

Jelena Roganovic, MD

Department of Pediatrics
 Division of Hematology and Oncology
 Clinical Hospital Centre Rijeka
 Rijeka, Croatia

References

1. Powers JM, Shamoun M, McCavit TL, Adix L, Buchanan GR. Intravenous ferric carboxymaltose in children with iron deficiency anemia who respond poorly to oral iron. *J Pediatr* 2016;108:212-6.
2. Mantadakis E. Advances in pediatric intravenous iron therapy. *Pediatr Blood Cancer* 2016;63:11-6.

Neurocognitive function; with or without passive smoke exposure



To the Editor:

Lande et al¹ reported that youth with primary hypertension demonstrated significantly lower performance on neurocognitive testing compared with normotensive controls,