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OSHA: A multidisciplinary scheme for treating autistic children

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OSHA program aims at progressing autistic children by diverting them as nearly as possible to the average normal aspects of social, communication, language and academic skills. This program started successfully in 2002 with 2 autistic children. Today, this program is applied successfully over many autistic children in the Specialized Child Center, Alexandria, Egypt with great success. The criteria for inclusion in the OSHA program is- 1st: The younger the age to start the better is the result (<5 years old); 2nd: full consent of parents for program steps and instructions and; 3rd: the program goes on for 1-3 years. OSHA program starts with a full neuro-psychometric and developmental skills assessment aiming for accurate diagnosis, to assess the severity of the disorder and for progress follow up. The assessment includes: DSM- 5 criteria and CARS scoring for diagnosing autistic children and severity assessment. Criteria of OSHA treatment program include: 1) Severity of Autism: For weekly credit hours of working; 2) Social Defect: For diversity of specialists; 3) Language Defect: Diversity of plans; 4) Learning Defect: Diversity of training places and; 5) Family Role: Recognition and involvement. An example of OSHA program for an autistic girl aged 2 years with severe autism (CARS score is 42.5) will be applied in the presentation. The core of the OSHA program is early intervention with a well-run program and multi-disciplinary team is the key for helping autistic children to reach their potential. The presentation will include video recording of cases with autism before and after OSHA program application.

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The pathogenesis of microcephaly resulting from congenital infections: Why is our baby's head small?

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The main pathogens associated with congenital infection and affliction are Zika virus, Cytomegalovirus, Rubella virus, Herpes simplex virus, Herpes varicella virus, and Toxoplasma gondii, which are often manifested with microcephaly as well as other stigmata, are briefly reviewed. The trimester of maternal infection and the role of non-primary infection with regard to fetal outcome are noted. The epidemiology and manifestations of infections with these organisms in non-pregnant hosts, the pregnant woman, and in the fetus are reviewed. Pathogen specific characteristics are highlighted, including the differences between Zika virus and the other teratogenic pathogens noted above. The pathology of microcephaly is reviewed in detail with a discussion of the similar neuropathogenesis of the infections caused by these agents including some of the other flaviviruses. The immunopathologic findings for these congenital infections are outlined. Hypotheses which might explain why: some infants are not infected in the presence of primary maternal infection, while others acquire subclinical infection, but still others are severely afflicted are discussed. These hypotheses include: pathogen strain differences, trophism to and susceptibility of various pathogens to developing fetal tissues, the role of various subsets of maternal immunity, and aspects of fetal immune responses.

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