

des, and polychlorinated biphenyls (PCBs). Diverse mechanisms explained the role of obesogen chemicals, the majority having disruptive effects on homeostasis of energy balance, glucose and lipid metabolism, and control of adipogenesis. These chemicals disrupt some epigenetic, structural, and functional mechanisms, which control energy homeostasis, lipid metabolism, appetite regulation, and adipogenesis. Exposure to these chemicals has different effects: during fetal and infancy periods may be irreversible and long-lasting for adulthood and exposure to low doses during critical times of differentiation can change the developmental programming and may result in obesity. Prenatal maternal smoking is associated with increased occurrence of overweight among children and early adolescents. Such health consequences may become apparent not only in childhood, but also in adulthood. Other chemicals have impact on intrauterine growth retardation, low birth weight, and prematurity which are documented as predisposing factors for obesity.

Conclusion. Obesity epidemic should be considered a multifactorial complex disorder. The potential effect of chemicals on the developmental programming of obesity is great and there are necessary more studies on chemical exposures during pre- and perinatal development to assess later obesity.



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**COMPLICAȚII PLEURO-PULMONARE LA PACIENȚII CU FIBROZĂ CHISTICĂ
IN RELAȚIE CU ETIOLOGIA BACTERIANĂ A INFECȚIILOR PULMONARE**

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Introducere. Fibroza chistică (FC) este o patologie ereditară cu transmitere autosomal-recesivă, caracterizată prin pneumopatii cronice obstructive, sindroame de malabsorbție și malnutriție, cu evoluție cronică progresivă și cu prevalență variabilă.

Se cunosc peste 2000 de mutații CFTR, cea mai frecventă fiind F508del, identificată la 80% dintre pacienții cu FC. Genotipul homozigot al mutației F508del este responsabil pentru cele mai severe forme clinice cu risc major de complicații pleuropulmonare cu potențial letal major, care în asociere cu germenii multidrogresistenți accelerează procesele distructive ale parenchimului pulmonar și contribuie la dezvoltarea complicațiilor pulmonare.

Scop. Evaluarea complicațiilor pleuropulmonare la pacienții cu FC cu infecții pulmonare în raport cu etiologia lor bacteriană.

Metode. În studiu sunt incluși 80 de pacienți cu FC, spitalizați și evaluați clinic și paraclinic în Centrul de Fibroză Chistică și în Clinica de Chirurgie Pediatrică, Ortopedie și Anesteziologie, pe o perioadă de 20 de ani.

Diagnosticul pozitiv al FC a fost confirmat prin testul sudorii și de cercetarea moleculară a ADN-ului, pentru a determina mutația CFTR. Examinările imagistice includ radiografia pulmonară, tomografia computerizată cu angiografie.

Rezultate. Caracteristicile principale ale grupului: vârsta medie 8,79±0,96 ani (1 lună - 38 ani), distribuție pe sexe 1:1 și vârsta medie a diagnosticului CF – 3,61±0,88 ani, cu variații în perioada neonatală până la vârsta de 34 de ani.

Evoluția progresivă a procesului patologic pulmonar a fost determinată de germeni rezistenți, cum ar fi *Ps.aeruginosa* (62,5%), *S. aureus* (55%), *St.maltophilia*, complexul *B.cepacia*.

Bronșiectaziile (62,5%) la pacienții cu CF au fost predominant localizate în lobi superiori (50%), predominant sacciforme (47,5%), adesea cu niveluri de lichid (18,7%).

Dilatarea hilurilor pulmonari, datorate adenopatiei infecțioase, a fost determinată la ¼ pacienți cu FC și creșterea dimensiunii trunchiului pulmonar s-a evidențiat în 18,7% din cazuri, indicând instalarea hipertensiunii pulmonare și a cordului pulmonar.

Pe perioada studiului, 25% pacienți cu FC cu infecții pulmonare au fost diagnosticați cu pleurezie, ce a necesitat toracocenteza în 8,7% din cazuri. Empiemul pulmonar, confirmat în 7,5% din cazuri, reprezintă o complicație gravă la pacienții cu FC. Distrucții pulmonare (27,5% cazuri) au prezentat pacienții cu FC cu exacerbări ale infecțiilor pulmonare cu *S. aureus*, *Ps.aeruginosa* și *B.cepacia*. 17,5% dintre pacienții cu FC dezvoltă pneumotorace, care apare ca urmare a rupturii țesutului pulmonar, fiind o complicație gravă cu risc pentru viața pacientului.

Concluzii. Sindroamele pulmonare la pacienții cu fibroză chistică evoluează progresiv, cu riscul decesului prin complicații pleuro-pulmonare severe, care sunt cauzate de infecții agresive de *Pseudomonas aeruginosa*, afecțiuni alveolare pulmonare, abcese, pleurezie. Prognosticul FC cu implicare bronhopulmonară este legat de gradul leziunilor respiratorii, natura complicațiilor pleuropulmonare, prezența sindroamelor extrapulmonare.

**PLEURO-PULMONARY COMPLICATIONS IN PATIENTS WITH CYSTIC FIBROSIS IN RELATION
WITH BACTERIAL ETIOLOGY OF LUNG INFECTIONS**

Introduction. Cystic fibrosis (CF) is a hereditary pathology with autosomal-recessive transmission characterized by obstructive chronic pneumopathy, malabsorption and malnutrition syndromes, with progressive chronic evolution, and with varying prevalence.

Genetic researchers have discovered more than 2000 mutations in the CFTR gene, and the most common mutation is F508del identified in 80% CF patients. The homozygous genotype of the F508del mutation is responsible for the most severe clinical forms with major risks of pleuropulmonary complications, with high fatal potential, which accelerate the destructive processes of the pulmonary parenchyma in association with multidrogeresistant germs, which contribute to the development of pulmonary complications.

Aim. Evaluation of pleural and pulmonary complications in CF patients with lung infections in relation with its bacterial etiology.

Methods. It is a study of 80 patients with CF, hospitalized and assessed clinical and paraclinical in the Cystic Fibrosis Center and in the Clinic of Pediatric Surgery, Orthopedics and Anesthesiology, for 20 years.

The positive diagnosis of CF has been confirmed by the sweat test and molecular DNA research to determine CFTR mutation. The imaging exam included pulmonary radiography, computed tomography with angiography.

Results. The group had characteristics: mean age $8,79 \pm 0,96$ years, with age extremes 1 month - 38 years, distribution by sex 1:1, and average age of CF diagnosis – $3,61 \pm 0,88$ years, with variations in the neonatal period up to the age of 34 years.

The progressive evolution of the pulmonary pathological process was determined by resistant germs such as *Ps.aeruginosa* (62,5%), *S.aureus* (55%), *St.maltophilia*, *B.cepacia complex*.

Bronchiectasis (62,5% patients) in CF patients was predominantly localized in upper lobes (50%) and predominant sacciforms bronchiectasis (47,5%), often with fluid levels (18,7%).

The dilatation of the hills due to infectious adenopathy was determined in $\frac{1}{4}$ patients with CF, and the pulmonary trunk size increase in 18,7% of cases, which indicate the installation of pulmonary hypertension, pulmonary cord.

During surveillance, 25% CF patients with lung infections were diagnosed with pleurisy, which required thoracentesis in 8,7% of cases. Lung empyema was a serious complication in these patients, confirmed in 7,5% of cases. Pulmonary destructions (27,5% cases) developed in CF patients with exacerbations of *S.aureus* lung infections, *Ps.aeruginosa* and *B.cepacia*. 17,5% of CF patients develop pneumothorax, which occurs as a result of pulmonary tissue rupture, which is a serious complication with danger to the patient's life.

Conclusions. Pulmonary syndromes in patients with cystic fibrosis evolve progressively, with the risk of death by severe pleuro-pulmonary complications, which are caused by aggressive *Ps.aeruginosa* infections, by pulmonary alveolar affection, abscesses, pleurisy. The prognosis of CF with bronchopulmonary involvement is related to the degree of respiratory lesions, the character of pleuro-pulmonary complications, the presence of extrapulmonary syndromes.



Ilia Catsap

**PREVALENCE OF ANEMIA AND IRON DEFICIENCY BETWEEN
THE AGES OF 18 AND 36 MONTHS, A STUDY OF 244 INFANTS**

“Clalit” Health Services (Israel)

Background: The prevalence of Microcyte Anemia, which is mostly caused by iron deficiency, has been extensively studied in a mammalian group aged 1 to 2 years.

In a work performed by “Clalit” Health Services in the population of infants aged 9 to 18 months, anemia was found at an average frequency of 11.5%, with no decrease in toward the age of 18 months. The prevalence of the non-Jewish population was twice as high as among Jewish-born infants.

Until today, there is insufficient information about the prevalence of anemia and iron deficiency in reservoirs (low item values) between the ages of 18 and 36 months in Israel and abroad.

Iron deficiency in young ages has been shown to cause cognitive impairment, sometimes with irreversible injury. Therefore, locating a condition of iron deficiency at a young age and providing iron-based preventive treatment is essential for the children's future.

The accepted recommendations in Israel are to provide iron-powered treatment for all infants from 4 months to 1 year of age and to perform a blood count at the age of one year in order to locate babies with anemia and recommend iron treatment. The reduction in the percentage of infants with anemia at one year was accepted as one of the quality indicators of the health services in recognition of the importance of the issue.