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Challenges in the Clinical Management of Fetal Alcohol Spectrum Disorders (FASD): An Updated Review

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Abstract

Background: Fetal Alcohol Spectrum Disorder (FASD) remains one of the leading preventable causes of developmental disabilities and intellectual impairment worldwide. Despite its high prevalence, especially in the European region, it is frequently underdiagnosed or misidentified due to its complex clinical presentation and phenotypic overlap with other neurodevelopmental conditions.

Aim: The aim of this paper is to provide a comprehensive review of the current literature on fetal alcohol spectrum disorders (FASD), with particular emphasis on neurobiological mechanisms, diagnostic challenges and contemporary therapeutic strategies. The paper aims to systematise knowledge about clinical differences within the spectrum and to identify key factors influencing patient prognosis.

Material and Methods: The PubMed, Google Scholar and Scopus databases were analysed using the keywords: 'FASD', 'fetal alcohol syndrome', 'pathophysiology', "diagnosis" and

‘intervention’. The literature search was updated in January 2026 to include the most recent clinical guidelines and neurodevelopmental research.
Twenty-five English-language bibliographic items were included in the review, including meta-analyses, systematic reviews and current clinical guidelines.
Results: The analysis showed that prenatal alcohol exposure leads to permanent structural changes in the brain through oxidative stress and epigenetic disorders, resulting in profound deficits in executive functions. It was found that standardised coding systems significantly increase the accuracy of differential diagnosis in relation to ADHD and other neurodevelopmental disorders.
Conclusions: Early identification of disorders and the implementation of multidisciplinary support before the age of 6 are crucial for minimising the secondary effects of FASD. Total abstinence by pregnant women remains the only effective method of prevention, requiring ongoing public education.
Keywords: Fetal Alcohol Spectrum Disorders, Prenatal Alcohol Exposure, Neurodevelopmental Disorders, Executive Functions, Early Intervention

I. Introduction

Fetal Alcohol Spectrum Disorders (FASD) are currently one of the main non-genetic causes of intellectual disability and neurodevelopmental disorders in children worldwide. The term is not a diagnostic entity in itself, but rather an umbrella term covering a range of effects resulting from prenatal alcohol exposure (PAE). This spectrum includes, among others, Fetal Alcohol Syndrome (FAS), partial FAS (pFAS) and Alcohol-Related Neurodevelopmental Disorders (ARND).

According to a meta-analysis, the global prevalence of FASD in the general population is estimated at approximately 7.7 cases per 1,000 live births.[1] However, it should be noted that these statistics vary greatly from region to region. The highest rates are reported in the WHO European Region, which is associated with high alcohol consumption among women of childbearing age. It is estimated that approximately 1 in 67 women who drink alcohol during

pregnancy will give birth to a child with full-blown FAS, illustrating the scale of risk to which the developing fetus is exposed.

The evolution of diagnostic criteria over the last few decades has led to a better understanding that cognitive and behavioral deficits can occur even in the absence of characteristic facial dimorphisms, which are key to the diagnosis of FAS.

Early identification of these disorders is critical because it allows for the implementation of appropriate interventions that can prevent secondary disorders such as mental health problems, learning difficulties, or conflicts with the law in adulthood. [2]

II. Pathophysiology: Mechanism of alcohol's effect on the developing brain

The teratogenic effect of alcohol on the central nervous system (CNS) is a multidirectional and complex process. Ethyl alcohol and its metabolite, acetaldehyde, easily cross the placental barrier, reaching concentrations in the amniotic fluid similar to those in the mother's blood. Since the fetus does not yet have fully developed liver enzymes capable of metabolising ethanol, its toxic effects persist much longer than in adults.

According to research, alcohol disrupts key neurodevelopmental processes such as proliferation, migration and differentiation of nerve cells. The main mechanisms of damage include:

-Oxidative stress: Excessive production of free radicals leads to apoptosis (programmed death) of neurons and glial cells.[3]

-Neurogenesis disorders: Alcohol inhibits the formation of new cells in areas such as the hippocampus (responsible for memory) and the cerebellum (coordination and executive functions). [4]

-Impact on neurotransmitters: Ethanol interferes with glutamate (NMDA) and GABA systems, leading to abnormal brain ‘wiring’ and subsequent problems with excitability and inhibition. [5]

-Epigenetic damage: Alcohol can modify the expression of genes responsible for brain development, which explains why the effects of exposure are permanent and irreversible.[6][7]

The degree of damage depends on the dose, drinking pattern (binge drinking is particularly dangerous) and stage of embryogenesis. [8]For example, exposure in the first trimester has the strongest effect on facial dimorphisms and organ defects, while exposure in the third trimester is associated with massive neuron loss and synaptic connection disorders.

An important aspect of the FASD pathomechanism is also the effect of ethanol on micronutrient metabolism; observations indicate that alcohol disrupts the transport of key nutrients through the placenta, which exacerbates the negative neurodevelopmental effects in the fetus and may determine the severity of symptoms within the spectrum.[9]

III. Diagnosis: Challenges and differential diagnosis

The diagnostic process for FASD is complicated due to the high heterogeneity of symptoms and the overlap of symptoms with other neurodevelopmental disorders. Differential diagnosis remains a key challenge, especially in relation to ADHD. Although both disorders are characterised by attention deficits and hyperactivity [10], the underlying mechanisms are different – in FASD, these problems result directly from structural brain damage caused by alcohol exposure, which is often associated with more profound executive function and working memory impairments than in isolated ADHD. [11]

Contemporary diagnostics aim to objectify assessment through the use of standardised tools such as the 4-Digit Diagnostic Code. This allows for a precise assessment of four key areas: growth retardation, characteristic facial dimorphisms, central nervous system damage, and confirmed prenatal exposure to alcohol. [12] As recent analyses indicate, an interdisciplinary approach to diagnosis is essential to avoid misdiagnosis and ensure that patients receive support appropriate to their neurodevelopmental profile, rather than just their behavioral symptoms.

The differential diagnosis of FASD is a complex process due to the wide range of symptoms, which often overlap with other neurodevelopmental disorders. The greatest challenge remains distinguishing attention deficits resulting from alcohol-related damage from the classic form of ADHD, which requires a precise neurobehavioral assessment. There is also often an overlap between FASD symptoms and autism spectrum disorders and attachment disorders, which can lead to misdiagnosis and inadequate treatment. Rare genetic syndromes, such as Dubowitz syndrome or Cornelia de Lange syndrome, which may present similar facial dysmorphic features, should also be ruled out.[13] An important part of the diagnostic process is neuroimaging, which allows for the detection of specific anomalies in the structure of the

cerebellum or hippocampus, which are less common in other disorders. The final diagnosis should always be based on a multidisciplinary assessment, taking into account both physical characteristics and the patient's detailed cognitive profile.

IV. Cognitive profile and executive function disorders

Cognitive deficits in the FASD spectrum extend significantly beyond general intelligence, which in many patients may be within the normal range.[14] The most characteristic element of the neuropsychological profile of individuals with FASD is executive function disorders, including difficulties with planning, organisation, flexible thinking and response inhibition.[15] These ‘invisible’ impairments make it extremely difficult for children to understand cause-and-effect relationships and learn from their mistakes. [16]

Another key area is working memory impairments and deficits in social information processing. Research shows that children with FASD often show significant discrepancies between their chronological age and their developmental age in the adaptive sphere. [17] This means that they may have relatively well-developed language skills that mask a profound misunderstanding of instructions and an inability to function independently in a peer group.

V. Support and Therapy: Interventions and the importance of the environment

Therapeutic interventions in FASD aim not only to minimise the effects of primary brain damage, but above all to prevent so-called secondary disorders, such as interruption of education, problems with the law or substance abuse. As researchers emphasise, the most effective therapeutic approaches are based on a multisensory model and a highly structured environment.[18] Children with FASD function best in a predictable environment where instructions are short, specific and visually supported.

A key element of support is also the education of parents and teachers.[19] Programmes such as GoFAR and Families on Track focus on training executive functions and self-regulation in children, while supporting the parenting skills of carers. Research shows that a stable home environment and diagnosis before the age of 6 are the strongest protective factors that allow people with FASD to better adapt socially in adulthood.

The contemporary approach to caring for patients with fetal alcohol spectrum disorders is evolving towards integrated and multi-level support systems. A key element of modern clinical management is the so-called ‘pediatric medical home’ model. It involves building lasting

therapeutic cooperation and providing care in a trauma-informed manner. This approach allows for continuous supervision of the patient and flexible adaptation of interventions to the changing needs of the child at different stages of their development [20].

Current scientific evidence strongly emphasises that effective support must include not only the child, but also their immediate environment. Family-centred interventions, including caregiver education and techniques for reducing intra-family stress, have been shown to be crucial for stabilising patient behaviour. A stable and predictable home environment plays a protective role, being able to partially mitigate neurodevelopmental deficits resulting from prenatal alcohol exposure through epigenetic mechanisms. [21] [22]

In the field of neurobiology, the latest reports indicate specific neurotransmitter function disorders, which directly translate into cognitive function problems. This suggests that modern therapy should increasingly combine behavioral techniques with targeted pharmacotherapy to improve patients' social functioning and educational outcomes [23]. In addition, data from long-term observations confirm that early implementation of integrated community support is the most effective method of preventing secondary disorders and significantly improves the long-term quality of life of people with severe alcohol-related brain damage.[24]

Table 1. Comparative analysis of the major FASD diagnostic systems based on clinical and functional criteria.

Comparative Criterion	**4-Digit Diagnostic Code (University of Washington)**	**Hoyme et al. / IOM Criteria (Revised Guidelines)**	**Canadian Guidelines**
Primary Approach	Objective, based on a quantitative Likert scale.	Clinical, based on predefined phenotypic parameters.	Functional – emphasis on the neuropsychological profile.
Facial Dysmorphia	Computer-aided objective photographic analysis.	Clinical examination.	Clinical assessment with a wider margin for subjective interpretation.

Growth (Height/Weight)	Mandatory diagnostic parameter.	Key clinical element.	Frequently omitted or considered secondary.
CNS Assessment (OUN)	Scalar assessment: from normal to severe structural changes or microcephaly.	Requires evidence of structural abnormalities or neurological impairment.	Most rigorous**:** Requires impairment in at least 3 neurodevelopmental domains.
Confirmation of Maternal Alcohol Consumption	Not required for a diagnosis of full FAS.	Required for ARND, but not essential for full FAS.	Key requirement in most diagnostic categories.
Advantages	High objectivity and diagnostic reproducibility.	Most intuitive and widely utilized in clinical medical practice.	Best reflects the child's real-life functional challenges (learning, emotions).

FAS – Fetal Alcohol Syndrome; ARND – Alcohol-Related Neurodevelopmental Disorder; CNS – Central Nervous System; IOM – Institute of Medicine. [12] [15] [22] [25]

Discussion

Analysis of the collected evidence confirms that FASD is a much more complex problem than suggested by early diagnostic models focused mainly on full-blown FAS. The results of the meta-analysis indicate an alarmingly high incidence of disorders in Europe, which calls into question the effectiveness of previous prevention campaigns.[1] In light of the pathophysiological data presented, particularly the role of oxidative stress and nutritional deficiencies, it becomes clear that the degree of CNS damage is the result of many variables, which explains why children with similar prenatal exposure may present extremely different cognitive profiles.[3]

Differential diagnosis remains a key point of contention in the literature. As demonstrated in the section on diagnosis, the overlap between FASD and ADHD symptoms leads to frequent clinical errors.[11] This problem is exacerbated by the fact that many countries (including

Poland) still lack a unified coding system, which makes guidelines a necessary document, although still insufficiently implemented in routine pediatric practice. [12] The lack of characteristic dimorphisms in patients with ARND means that this group remains ‘invisible’ to the system, only coming to the attention of specialists when serious secondary disorders occur.

It is also worth noting the optimistic trend in the latest research on neuroplasticity. Although brain damage in FASD is irreversible, reports show that early and multi-level intervention can significantly modify a child's development. [13] However, the availability of these methods remains debatable – most effective therapeutic programes require a high level of financial and human resources, which in many healthcare systems is an insurmountable barrier. [18]

The contemporary approach to FASD is evolving from a purely medical model towards holistic bio-social support, which poses new organisational challenges for clinicians. The main problem remains the lack of globally standardised diagnostic criteria, which leads to a significant underestimation of the number of cases in many regions of the world. [20] The results of the latest cohort studies suggest that early identification of alcohol exposure is crucial to reducing future healthcare costs. In addition, the growing role of epigenetic markers opens up new perspectives in understanding individual variability in symptoms in children with similar degrees of prenatal exposure. These findings point to the need for closer cooperation between gynecologists, pediatricians and addiction treatment specialists in order to prevent the effects of FASD at an early stage.

In summary, the main challenge remains the transition from a reactive model (diagnosing effects) to a proactive model (precise diagnosis of cognitive profile and immediate support). Future research should focus on the search for biomarkers of prenatal exposure that would allow the identification of children at risk even before the first educational deficits appear.[24]

Conclusions

Analysis of the evidence clearly indicates that fetal alcohol spectrum disorders remain one of the most serious challenges in modern medicine and developmental psychology. At the same time, it is a condition that can be completely prevented. Given the lack of a scientifically defined safe dose of alcohol and the variable individual sensitivity of the fetus to teratogens, the only effective preventive strategy remains the promotion of total abstinence among women of childbearing age. A key conclusion from the latest research is the urgent need to standardise diagnostic processes. The implementation of precise tools, such as multidimensional coding

systems, is essential for identifying patients with pFAS and ARND, who, due to the lack of clear dysmorphic features, often remain outside the support system, which drastically increases their risk of developing secondary disorders.

An equally important change seems to be the shift in approach to therapy – from reactive to proactive. The priority should be to minimise the time between exposure and diagnosis, as early intervention during the period of greatest brain plasticity offers a real chance to optimise the child's cognitive and social development. However, the effectiveness of these measures is directly dependent on the creation of a multi-specialist care model that integrates the activities of pediatricians, psychiatrists and educators, while offering systemic support for carers. In terms of future research, it is crucial to search for objective markers and further explore neurobiological mechanisms, which will allow for even better adaptation of rehabilitation methods to the individual neuropsychological profile of patients with FASD. It is also necessary to intensify educational activities aimed not only at patients, but above all at primary care medical staff. Raising awareness among gynecologists and midwives about routine screening for alcohol consumption can become the foundation for effective primary prevention. Only through a systemic change in social attitudes and the elimination of stigmatisation of mothers of children with FASD will it be possible to create an environment conducive to early detection of risk.

In the coming years, a key challenge will be to fully exploit the potential of advanced neuroimaging techniques to map functional deficits in the brain, allowing a shift away from universal treatment protocols towards personalised medicine. In addition, the development of telemedicine and digital support tools can significantly increase access to specialist care for families in excluded areas, which is an important step towards democratising support for people with FASD.

Disclosure

The authors declare no financial or non-financial conflicts of interest related to this manuscript.

Supplementary Materials

No supplementary materials are associated with this article.

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All authors have read and approved the final version of the manuscript.

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