

PERSPECTIVES IN PEDIATRIC PATHOLOGY

# Perspectives in Pediatric Pathology, Chapter 5. Gonadal Dysgenesis

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Received April 23, 2014; accepted May 20, 2014; published online August 13, 2014

## ABSTRACT

One of the most challenging areas in pediatric testicular pathology is the appropriate understanding and pathological diagnosis of disorders of sexual development (DSD), and in particular, the issue of gonadal dysgenesis. Here we present the main concepts necessary for their understanding and appropriate classification, with extensive genetic correlations.

## INTRODUCTION

We define gonadal dysgenesis (GD) as an incomplete or defective gonadal differentiation resulting from disturbances in germ cell migration and/or adequate germ cell organization in the gonadal ridge. Morphologically, GD expresses a variable degree of differentiation toward ovary, testis, or both. The term gonadal dysgenesis refers to a broad spectrum of lesions, with gonadal agenesis at one end, followed by persistence of undifferentiated gonadal tissue, continuing with the presence of a classical streak gonad (*vide infra*), and ending with a testis surrounded by ovarian cortex-like stroma. Therefore, GD includes all kinds of gradually advancing gonadal phenotypes before reaching the full architecture of an ovary or testis. It is possible that all phenotypes of GD represent a spectrum of the same disorder [1].

In the classical sense, GD comprises several disorders characterized by amenorrhea and so-called "streak gonads" in phenotypically female patients with hypoplastic uteri and fallopian tubes. The pathologic realm of a streak gonad is shared by different phenotypes, such as those of Turner, Swyer, and Sohlval syndromes, all of which are included under the umbrella of GD.

Clinical entities that should also be considered syndromes of GD are grouped under the terms "male

pseudohermaphroditism with müllerian rests" (including Sohlval syndrome), male with uterus, and even dysgenetic male pseudohermaphroditism with preferential testicular differentiation of one or both gonads.

Normal gonadal development requires both sex chromosomes but also the participation of genes located in autosomes. Deficiencies of genetic material from the sex chromosomes also lead to characteristic phenotypical extragonadal alterations.

With regard to nomenclature, the 2005 International Consensus Conference on Intersex [2] considered terms such as pseudohermaphroditism, hermaphroditism, sex reversal, and intersex to be obsolete. The conferees introduced the designation disorders of sexual development (DSD) to include all congenital conditions in which chromosomal, gonadal, or anatomic sex is atypical (Table 1). However, this classification has received critical comments [3] because it was based on the somatic chromosomal constitution (peripheral karyotype of leukocytes). For example, in cases of GD, entities with a completely different histopathology, such as 45,X0 Turner syndrome and its variants, and mixed GD (MGD) (45,X/45,XY), have been included together with Klinefelter syndrome and variants under the term "disorders of sex development." Two facts contribute to the problem of the classification of these disorders: (1) the same karyotype can give rise to different phenotypes; and (2) it has been progressively understood that GD, instead of showing one cellular line, carries mosaicisms or more complex chromosomal constitutions. This problem is even more complicated if results of chromosomal studies from gonadal tissues are taken into account. Therefore, the classification of these disorders, considering genetic, endocrinological, clinical, and histopathological issues, should still be regarded a work in progress.

The pathological diagnosis, the histologic nature of the gonad, is based on a fact just as real as the karyotype.

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