Development of extra cranial germ cell tumour and their inhibition.

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Abstract

Germ cell tumours (GCTs) arising in infants, children, and adolescents present a set of special challenges. GCTs make up about 3% of malignancies in children aged 0–18 and nearly 15% of cancers in adolescents. Epidemiologic and molecular evidence suggests that GCTs in young children likely represent a distinct biologic group as compared to GCTs of older adolescents and adults. Despite this difference, pediatric GCTs are typically treated with cisplatin-based multiagent regimens similar to those used in adults.

Keywords: Cell tumour, Germ cell tumours, Cancers.

Introduction

Pediatric germ cell tumor is the term used to describe malignant cancers of germ line cells in patients aged 0–18 years. These cancers may arise in the testis, the ovary, or the extra gonadal sites including the sacrococcygeal area and the mediastinum. Germ cell tumors also occur in the brain in children and young adults. Though Intracranial GCTs (iGCTs) are histologically similar to extra cranial GCTs, it is unclear if tumors in the different sites arise by similar or different mechanisms, and the treatment approaches used are somewhat different; for these reasons, iGCTs are not further considered here [1].

Though the biology and clinical presentation of pediatric GCTs share significant overlap with that of adult testicular (T) GCTs, there are important differences that should be kept in mind. First, epidemiologic data reveal two distinct peaks in GCT incidence, one in young children (aged approximately 0–4 years) and a second peak beginning in puberty. While the histologic presentation and molecular biology of GCTs arising in adolescents appear similar to those in adult TGCTs, germ cell tumors in very young children have important differences (reviewed below), suggesting that they may represent a distinct disease [2].

Whether these results apply equally to children with gonadal or extra gonadal GCT remains to be established. In recent years, investigators in the Children's Oncology Group and the Children's Cancer and Leukemia Group (UK) have joined forces to form the Malignant Germ Cell Tumor International Collaborative (MaGIC) Consortium to improve outcomes for patients with germ cell tumors by generating new insights into etiology, prognosis, toxicity reduction, and optimal treatment. MaGIC investigators have produced revised evidence-based risk stratification for pediatric and adolescent GCTs based on amalgamation of 25 years of clinical trial data from the US and the UK [3].

Several lines of evidence suggest that GCTs do not arise from a mature gonadal cell (e.g., a spermatogonial stem cell) but rather from a germ cell in early stages of development. This was discovered through an interesting set of observations that linked etiological phenomena to characteristics of the developing germ line. GCTs can be classified into two major types based on histology, known as seminomatous GCTs and nonseminomatous GCTs. Seminomatous GCTs are tumors which are made of undifferentiated germ cells which can histologically resemble early spermatogonia, oogonia, or even germ cells from developmental lineages [4].

The primordial germ cell, responsible for specification of the germ line, is unique among cells of the body in its requirement to maintain the pluripotent potential necessary for gamete generation. This requirement creates a unique developmental cycle which involves stages of vulnerability to improper differentiation. The PGC must be specified from the rest of the developing embryo through genetic and epigenetic events; it subsequently migrates throughout the body to the site of the gonad, and it must then undergo sex specific differentiation. Each of these stages reflects both a developmental feature and a clue related to the phenotypes and characteristics of germ cell tumors [5].

Conclusio

As the only nonsomatic tumor lineage in the body, GCTs exhibit a unique combination of varied histology, wide range of sites of presentation, and apparent lack of traditional oncogenic drivers, suggesting a prominent role for aberrant developmental pathways in the etiology of these cancers. A perspective that focuses on these mechanisms could be key to the development of differentiation-based therapies using either exogenous signaling ligands or small molecule activators or inhibitors of the relevant pathways, which might one day supplement or substitute for conventional cytotoxic therapies.

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