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High Risk Assessment Involved in Germ Cell Tumours

Alexander Abasheli*

Department of Oncology, University of Stony Brook, USA

DESCRIPTION

Germ cell tumours arise from primordial germ cells. Most occur along the gonads or midline structures of the body. Genetic abnormalities that lead to disruption of molecular signaling involved in migration of original germ cells early in development may provide an explanation for why germ cell tumours arise at extragonadal sites. Establishing best practices for the management of childhood germ cell tumours remains an area of active investigation. Recent advances have been made in limiting treatment toxicity, identifying new therapies for recurrent and refractory tumours, defining best practices for surgical staging and resection, and developing new methods to monitor disease recurrence. Testicular germ cell tumour is one of the most common malignancies in children and young adults. Genomic studies have identified characteristic molecular profiles of testicular cancer associated with histologic subtypes that can predict clinical behaviour, including treatment response. Emerging molecular techniques for analyzing tumour genomics, transcriptomics, and proteomics can now guide the precise treatment of testicular cancer. Laser-assisted micro dissection methods, such as laser capture micro dissection, efficiently separate selected tumour cells from normal pathology specimens, avoiding contamination from non-target cell populations. Combining laser capture micro dissection with next-generation sequencing enables accurate, high-throughput genetic characterization effectively and efficiently. The use of Laser Capture Micro Dissection (LCM) for molecular testing may offer significant advantages in the clinical management of patients with testicular cancer. This review describes an application protocol for laser-assisted micro dissection to study testicular germ cell tumours.

Mediastinal Germ Cell Tumour (MGCT) is the most common extragonadal Germ Cell Tumour (GCT), most commonly occurring in the anterior mediastinum and favouring men. MGCT is also preferred for patients with Klinefelter syndrome and other genetic disorders. MGCTs, like GCTs, are thought to arise at other extragonadal sites from improperly retained germ cells during midline migration during embryogenesis. Like their testicular counterparts, MGCTs are classified into seminoma and nonseminoma His GCTs. Seminoma MGCT refers to pure seminoma, nonseminoma MGCT includes mixed GCTs including any combination of GCT types including pure yolk sac tumour, embryonal carcinoma, choriocarcinoma, mature or immature teratoma, and seminoma. It is included. Somatic or hematologic malignancies may also occur in association with primary MGCT. MGCT shares molecular findings with GCTs at other sites, most commonly the presence of chromosome 12p gain and homo chromosomal. Treatment includes neo adjuvant chemotherapy followed by surgical resection of residual tumour, whereas benign teratomas require only surgical resection without chemotherapy. This review highlights and updates the pathological, clinical, and molecular features of MGCT.

CONCLUSION

Somatic analysis of the molecular signature of human tumours by many efforts, including the Cancer Genome Atlas consortium, has yielded unprecedented insight into the biology underpinning cancer behaviour. These studies employ a number of genome-wide sequencing techniques to understand the differences in DNA mutations, epigenomic alterations, and ultimately protein expression profiles in different cancer types. The falling cost of next-generation sequencing and the increased power and ease of use of computational resources will allow researchers to apply these techniques to more specific cancer situations and to rarer types of tumours.

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CONFLICTS OF INTERESTS

The authors declare that they have no conflict of interest.

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Corresponding author Alexander Abasheli, Department of Oncology, University of Stony Brook, USA, E-mail: abasheli972@gmail.com

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