chemotherapy. In Lugano III, 12 events occured: 6 DOD during initial treatment, one progression, 5 relapses. All of these patients had received platinum-based chemotherapy. Of the 5 relapses, 4 could by salvaged by high-dose platinum chemotherapy or other regimes. One patient died after high-dose platinum chemotherapy.

GCT-13 Treatment outcome of extracranial germ cell tumours in Chinese children in Hong Kong

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Background: We reviewed the treatment outcome for children with extracranial germ cell tumour (GCT) in Hong Kong.
Methods: Prospective territory-wide cohort study of children with GCT treated in five paediatric oncology centres in Hong Kong from January 1995 to December 2017. All patients were treated with a unified treatment protocol (HK-GCT protocol, adopted from CCLG GC protocol). Surgery was the only treatment for non-malignant GCT or early-stage malignant GCT. Carboplatin/Etoposide/Bleomycin (JEB) chemotherapy was directed to advanced stage disease.
Results: 205 patients enrolled (5% of childhood cancers in Hong Kong compared with 3% in Western populations). Age-range is day one of age to 18.6 years. The majority groups of histology were teratoma (51.7%), yolk-sac tumour (25.9%), mixed GCT (13.2%) and germinoma (5.4%). The primary sites were gonad (53.2%), mediastinum (15.6%), sacrococcygeal region (14.6%), abdomen (8.8%), pelvic (2.4%) and non-CNS head & neck (1%). The stages of the GCT patients were I (63.9%), II (6.8%), III (16.6%) and IV (12.7%) respectively. 58% patients were treated with surgery alone and 38.5% patients received JEB chemotherapy. The overall 5-year overall-survival was 91.3% (±2%) and 5-year event-free-survival was 87.1% (±2%). The median follow-up time was 8.3 years with 163 patients (79.5%) alive and disease-free. Eighteen cases relapsed (8.8%), 16 patients died (7.8%) and 26 patients lost to follow-up (12.7%). Seven patients (3.4%) developed second cancer. Current treatment of extracranial GCT in Chinese children is effective and comparable to Western studies; risk-stratified treatment is effective/safe. The second cancers deserve further investigation for underlying risk factors.

GCT-14 Rare localization in 44 paediatric germ cell tumours

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Background: There are few studies describing germ cell tumours (GCT) arising from rare primary sites such as head and neck, vagina among others.
Methods: We reviewed three National Brazilian Protocols (GCT 91, 99 and 2008) and identified 44 cases of GCTs arising from rare regions.
Results: Childhood GCT arising from kidney, dorsal region, pericardial, midline between stomach and esophagus, uterus, abdomen, vagina and head neck were identified. Vaginal GCT accounted for 27.9% of the cases, head and neck 41.8% and others 30.3%. From 18 cases of head & neck, histology were for the majority pure or immature teratoma, followed by yolk-sac tumour (YST) less often, then embryonal carcinoma and mixed GCTs. The most common histological subtypes of vaginal disease were YST. Most vaginal tumours were high risk, in contrast with head & neck tumours, where the majority were low-risk. Overall survival (5y OS) was 83.3%, 93.8% and 84.6% for vagina, head & neck, and rare sites, respectively. GCTs arising in rare locations with malignant histology could be treated with excellent rates of survival including the ones who presented vaginal primary. Usually tumours in rare sites lead to worse rates of survival in relation to other rare sites. This may occur due to resection and/or staging, since 83% of these tumours were classified as high-risk. Regarding benign tumours, the survival rate was related exclusively to resection.

GCT-15 Overcoming patient factors in the care of under-served testicular cancer patients

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Background: To determine whether patient factors at a safety net hospital are overcome through the standardized treatment of testicular cancer (TC) at a university tertiary care center.
Methods: The electronic medical records of patients who underwent orchiectomy at our university and safety net hospitals from 2006 to 2018 were reviewed. Variables were compared based on treatment setting. Comparison of continuous variables were reported as medians, and categorical variables were reported as percentages.
Results: 95 patients (47%) at the university hospital and 106 patients (53%) at the safety net hospital were included. Safety net patients had delayed presentation after symptom onset (median 65 vs 31 days, $p = 0.001$), were more likely to initially present to the emergency department (76% vs 8%, $p < 0.001$), and had shorter median time from diagnosis to orchiectomy (1 vs 4 days, $p < 0.001$). These patients had larger median tumour size (50 vs 30 mm, $p < 0.001$), were more likely to have higher T-stage ($p = 0.018$), were less likely to be Stage I (58% vs 73%, $p = 0.028$) and more likely to be Stage III (23% vs 9%, $p = 0.013$). However, there was no significant difference in median numbers of surveillance imaging (3 vs 3 CT scans, $p = 0.77$), urology clinic visits (4 vs 4 visits, $p = 0.73$), rate of cancer recurrence (13% vs 9%, $p = 0.51$), or mortality (4% vs 0%, $p = 0.12$) between safety net and university patients (Table 1). The integrated care of safety net patients at our academic centre appears to overcome socioeconomic barriers that exist in the treatment of testicular cancer.

Table 1.
Clinicopathological Characteristics of Patients Receiving Orchiectomy at Safety Net and University Hospital Patients with Testicular Cancer

Characteristic	No. of Patients (%)				p-Value
	Safety Net		University		
All patients	106	(100)	95	(100)	
Age: Median [IQR], years	29	[24–35]	33	[26–41]	0.005
Tobacco Use	52	(49)	28	28(29)	0.006
Marijuana Use	7	(18)	7	(7)	0.048
Presented to ED	81	(76)	8	(8)	<0.001
Insured	21	(20)	84	(88)	<0.001
Symptom onset to Diagnosis: Median [IQR], days	65	[21–183]	31	[12–89]	0.001
Diagnosis to Orchiectomy: Median [IQR], days	1	[1–3]	4	[1–9]	<0.001
Median tumour size, [IQR], mm	50	[27–87]	30	[16.5–50.5]	<0.001
Tumour Stage					0.018
T0	2	(2)	3	(3)	
T1	53	(50)	53	(56)	
T2	32	(30)	34	(36)	
T3	9	(8)	0	(0)	
T4	3	(3)	0	(0)	
TX	7	(7)	5	(5)	
AJCC Stage					0.026
Stage I	61	(58)	69	(72)	
Stage II	21	(20)	17	(18)	
Stage III	24	(23)	9	(9)	
Post-orchiectomy initial management					0.001
Chemotherapy	50	(47)	22	(23)	
RPLND	10	(9)	14	(15)	
XRT	3	(3)	7	(7)	
Surveillance	32	(30)	48	(51)	
Lost to Follow-Up (%)	11	(10)	4	(4)	
Post-Chemotherapy RPLND	19	(18)	10	(11)	0.16
CT imaging: Median [IQR]	3	[2–7]	3	[2–7]	0.77
Urology clinic visits: Median [IQR]	4	[2–8]	4	[2–6]	0.73
Oncology clinic visits: Median [IQR]	5	[1–9]	1	[0–8]	0.007
Cancer recurrence	14	(13)	9	(9)	0.51
Orchiectomy to Recurrence: Median [IQR], months	10	[5–15]	8	[2–11]	0.23
Overall mortality	4	(4)	0	(0)	0.12

Abbreviations: No., number; IQR, interquartile range; ED, emergency department; CT, computed tomography; AJCC, American Joint Committee on Cancer; RPLND, retroperitoneal lymph node dissection; XRT, radiation therapy.

Biology – Genomics and Developmental

GCT-16 Germline genomic architecture of testicular germ cell tumours: Lessons from a decade of experiments in genotyping and sequencing

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Background: As well as being a fascinating model of tumorigenesis, testicular germ cell tumours (TGCTs) have one of the highest familial relative risks of common adult-onset cancers and the advent of large-scale genotyping and sequencing technologies heralded great promise in revealing the genomic determinants thereof.
Methods and Results: I shall present how the experiments of the last decade have enlightened our understanding as to the germline genomic architecture of TGCT, how that has informed our understanding of the biology of disease and the implications for identification of individuals at elevated risk, screening and cancer prevention.

GCT-17 Developmental origins of testicular germ cell tumours (TGCT)

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Background: This review summarises evidence for the developmental origin of TGCT that are derived from germ cell neoplasia in situ (GCNIS) and discusses the main pathways implicated in the pathogenesis of this cancer.
Methods: Literature review.
Results: The GCNIS-derived TGCTs occur predominantly in young men, starting from adolescence. Epidemiological evidence (e.g. birth cohort effects) and a high risk of TGCT in patients with differences in sex development and testicular dysgenesis syndrome (TDS) are consistent with origin in the developmental period. GCNIS cells are transformed primordial germ cells/gonocytes and retain a high expression of pluripotency factors, hence maturation arrest during early development is considered the first hit in the TGCT pathogenesis. The arrest can be caused by a disruption of the cross-talk between developing germ cells and their somatic niche, which is regulated by a multitude of factors. Among the disrupted pathways, the sex differentiation signalling cascade, the androgen signalling, the TGF-beta pathway, the gonadotrophin signalling and the sex-dimorphic mitosis-meiosis switch have been identified, but other pathways likely will be discovered. The rapidly changing incidence of TGCT and other TDS components implicate mainly environmental/lifestyle factors, while ethnic differences in the incidence support a role of genetic background. Susceptibility loci identified in genome-wide and targeted genomic studies have also implicated pathways involved in sex differentiation and germ cell development (e.g. *KITLG, DMRT1, BAK1, SPRY4*). TGCT is a developmental disease, with multifactorial aetiology. The pathogenesis of TGCT is complex, and likely involves disruption of normal gonadal development, which is modulated by genetic variation.

GCT-18 Germ cell commitment occurs after PGC colonization of the gonad in mammals

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Background: In mammalian development, primordial-germ-cells (PGCs) are induced in epiblast and later migrate to nascent gonads, undertaking gametogenesis. Despite dramatic differences in early steps of germ-line development, evidence suggests that, once formed, migratory PGCs remain developmentally uncommitted, not yet