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