Lymphomas	Features	Behavior	Presentation	Therapy
Small	Naïve B-cells (prior to ag-driven	Indolent	Increase in normal-	Early stages: radiation
lymphocytic	differentiation w/in germinal center)	Incurable	appearing lymphocytes in	Later stages: observation
lymphoma (SLL)	IgM or IgD		peripheral blood and/or	to aggressive (chemo,
Chronic	CD5 (T-cell)		infiltration into nodes,	auto HSCT, local
lymphocytic			spleen, solid organs	radiation, retuxan vs
lymphoma (CLL)				CD20, interferon)
Follicular	Germinal center lymphocytes	Indolent	Follicles proliferate w/o	Early stages: radiation
lymphoma	T(14;18) involving <i>bcl-2</i> gene	Incurable	respect for architecture	Later stages: observation
	important in blocking apoptosis		Fewer mitotic figures (less	to aggressive
			turnover than hyperplasia)	
Large cell	Diffuse large B-cell lymphoma	Aggressive	No follicles	Chemo, CNS
lymphoma	(DLBCL)	Curable		prophylaxis
Burkitt's	Origin in germinal center	Extremely	Abdominal pain/discomfort	Chemo, CNS
lymphoma	U.S.: distal small intestine	Aggressive	No follicles	prophylaxis
	Africa: head & neck (EBV)	Curable	Starry sky pattern	
	T(8;14) involving <i>c-myc</i> oncogene			
Mantle cell	Diffuse lymphoma	Aggressive		
lymphoma	IgM or IgD	Incurable		
	CD5 (T-cell)			
	T(11;14) involving <i>cyclin-D1</i> gene			
	important for entry into cell cycle			
Marginal zone	Small lymphocytes	Indolent	bean-shaped nuclei w/	Abx for h pylori infec
lymphoma		Incurable	abundant cytoplasm	Early stages: radiation
MALT lymphoma			MALT: found in stomach,	Later stages: observation
			salivary & lacrimal glands,	to aggressive
			linked w/ h pylori	
Hodgkin's disease	<i>Reed-sternberg cells</i> derived from B-	Aggressive	Anterior mediastinel mass	Early stages: radiation,
(HD)	cells of germinal center	Curable	on routine chest x-ray	chemo, or both
	Nodular sclerosis type		Inflammatory infiltrate w/	Later stages: combo
	Mixed cellularity type (no sclerosis)		minority of cytologically	chemo (ABVD)
	Lymphocyte depletion type (little		distinct neoplastic cells	Salvage: chemo w/ auto
	inflammation)		(lacunar & hodgkin cells)	HSCT
	Nodular lymphocyte predominance		Contiguous spread	
	type (distinct, like low-grade NHL)			

Dysproteinemia & plasma cell	Diagnostic	Presentation	Therapy
proliferation disorders	criteria		
Multiple myeloma	10% plasma cells	HyperCa (OAF), fractures, renal failure (lambda	No cure
	or plasmacytoma	light chain deposits), hypervolemia & CHF (M	Palliative: chemo,
	M protein in	prot causing hi osmotic pres in vasc), hypervisc	auto/allo HSCT,
	serum & urine	(bulky M prot causing bleeding, retinopathy,	radiation, interferon,
	(bence-jones	neuro), cryoglobulinemia (M prot pcpt at low T),	pamidronate (prevent
	proteins)	hemostatic (M prot interfere w/ platelet function &	fractures), thalidomide,
	Multiple lytic	coag factors), anion gap (falsely low serum Na),	management of hyperCa,
	bone lesions	amyloidosis (light chain deposits), infec (pneum)	renal fail, pain etc.
Smoldering multiple myeloma		All criteria for multiple myeloma except no	May be left untreated for
		skeletal lesions, renal insufficiency or anemia	yrs before it evolves
Solitary plasmacytoma of bone		Solitary lytic lesion, no marrow involvement	Radiation
		Will eventually progress to MM	
Extramedullary plasmacytoma		Upper air passages (tonsil, palate, sinuses, orbit)	Radiation
		No evidence of plasma cell neoplasm	
		Broader age group	
		Less likely to progress to MM than plasmacytoma	
Waldenstrom's		Low-grade lymphoma w/ secretes high amounts of	Plasmapheresis if high
macroglobulinemia		IgM	viscosity
			Low-grade lymphoma
			chemo
Monoclonal gammopathy of			Observation
uncertain significance (MGUS)			No treatment