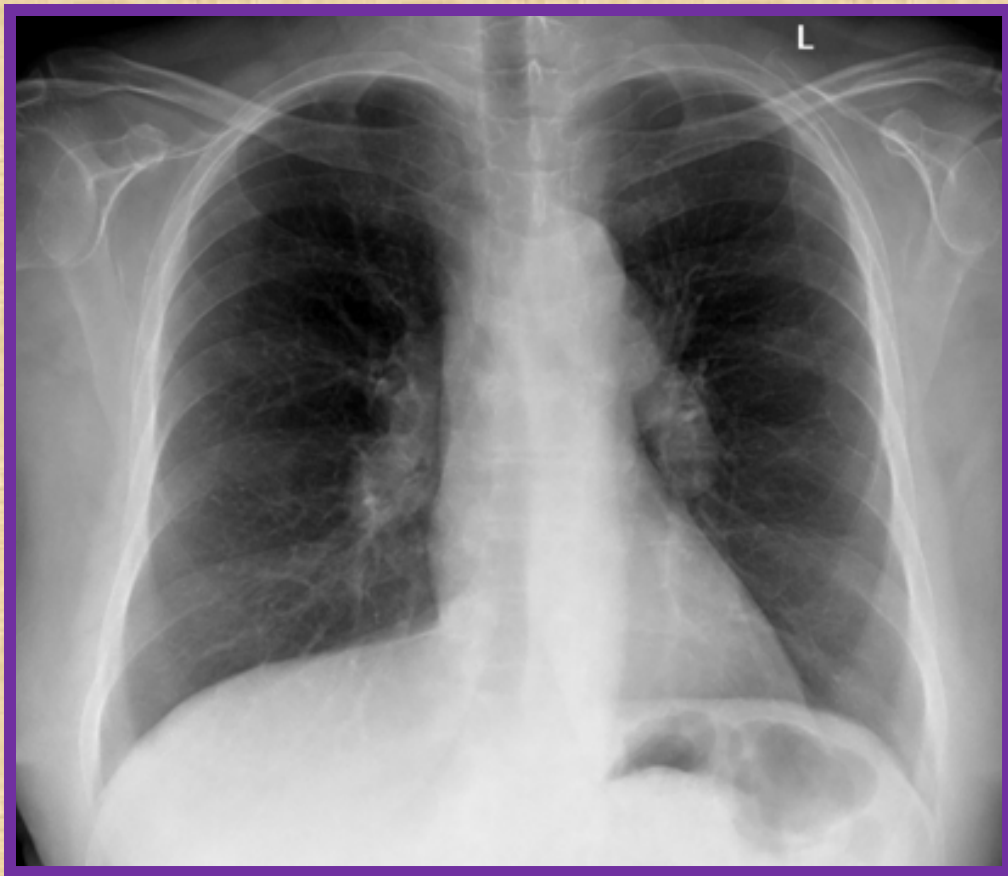


ΣΑΡΚΟΕΙΔΩΣΗ

Ε. Λαζαρίδου, Επιμ. Α'
ΤΜΗΜΑ CT/MRI
ΓΝΑ "Ο ΕΥΑΓΓΕΛΙΣΜΟΣ"

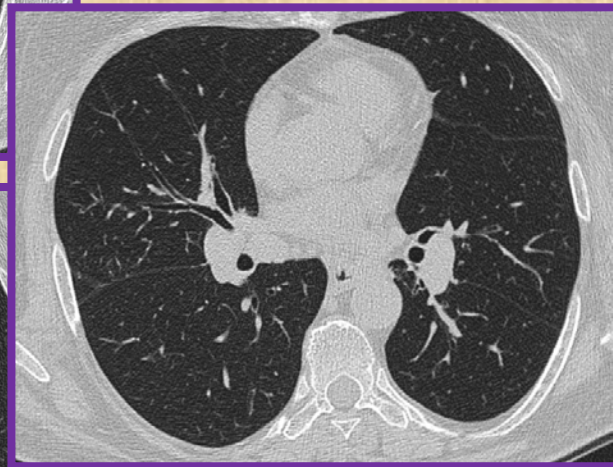
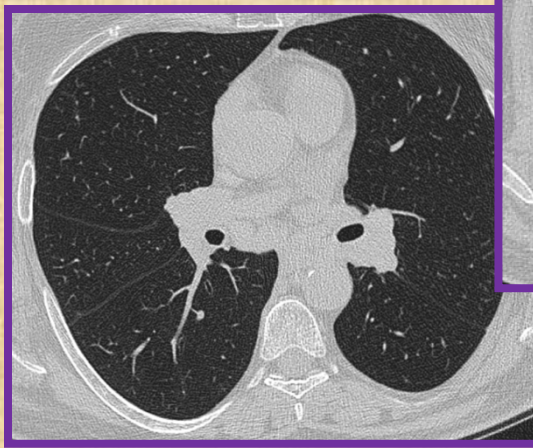
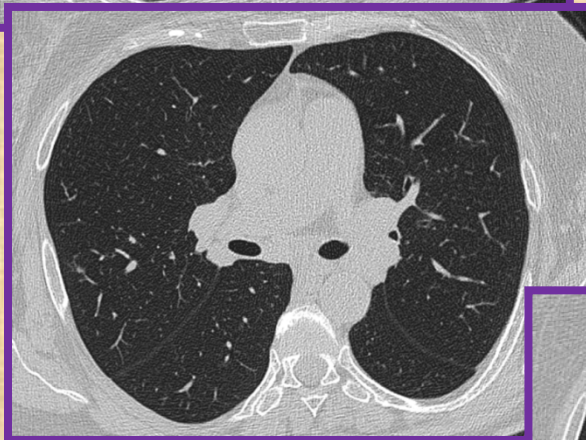
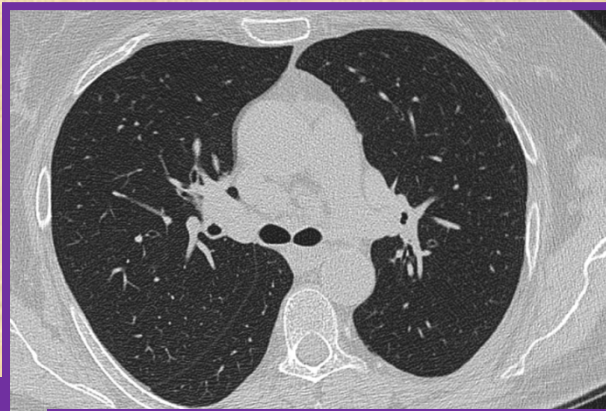


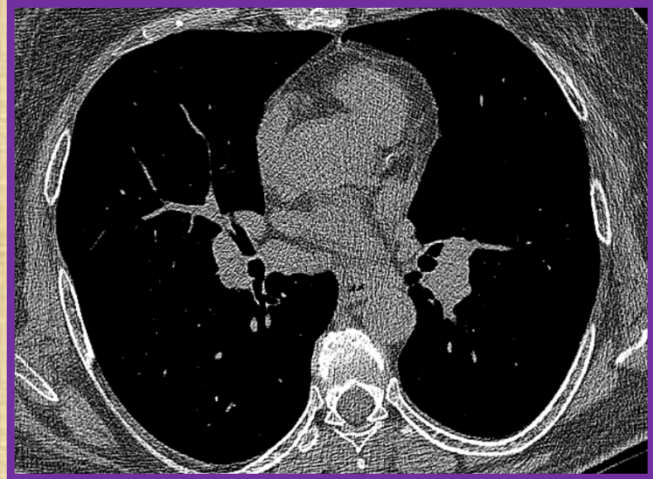
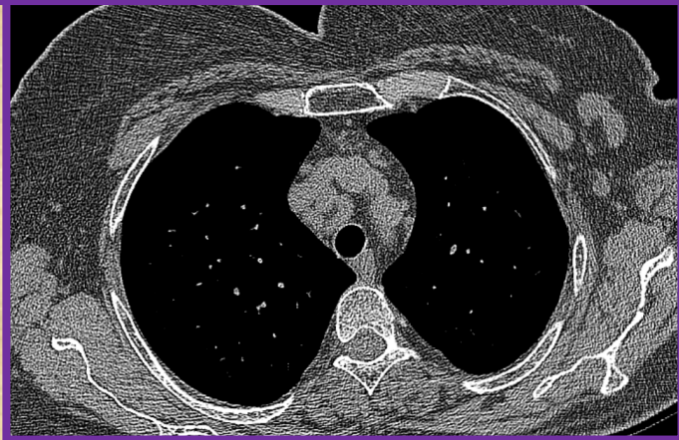
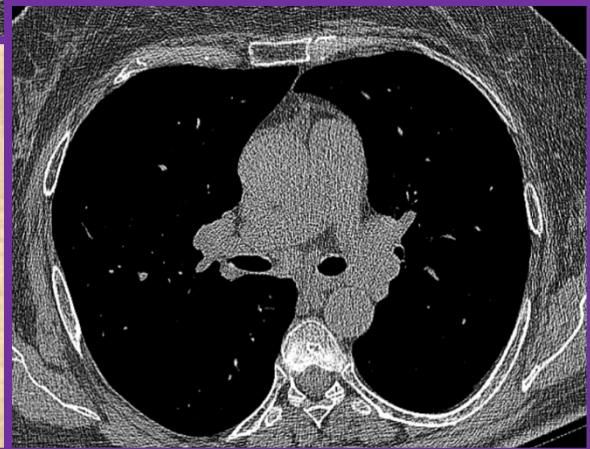
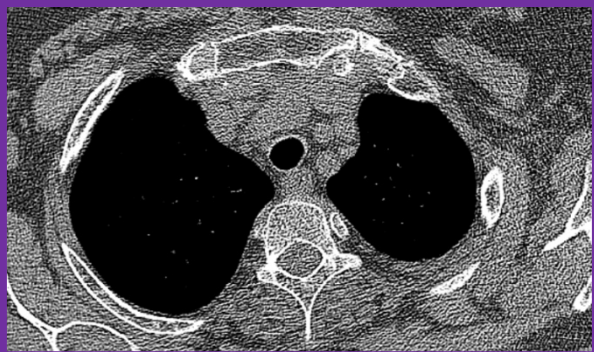
potato nodes

Pulmonary sarcoidosis may be classified on a chest radiograph into 5 stages (Scadding criteria) ^{1,2}:

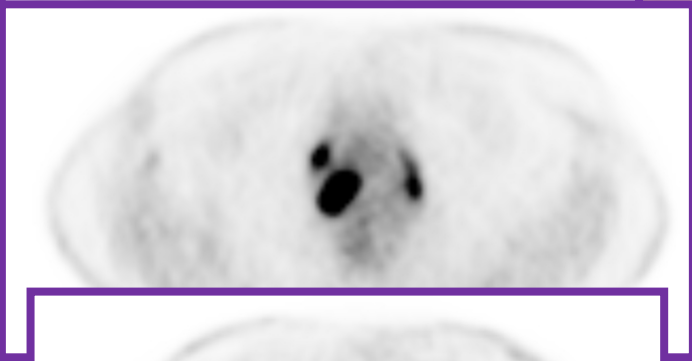
- **stage 0:** normal chest radiograph
 - 5-10% of patients at presentation
- **stage I:** hilar or mediastinal nodal enlargement only
 - 45-65% of patients at presentation
 - 60% go onto a complete resolution
- **stage II:** nodal enlargement and parenchymal disease
 - 25-30% of patients at presentation
- **stage III:** parenchymal disease only
 - 15% of patients at presentation
- **stage IV:** end-stage lung (pulmonary fibrosis)

1n. YT
29/5/17

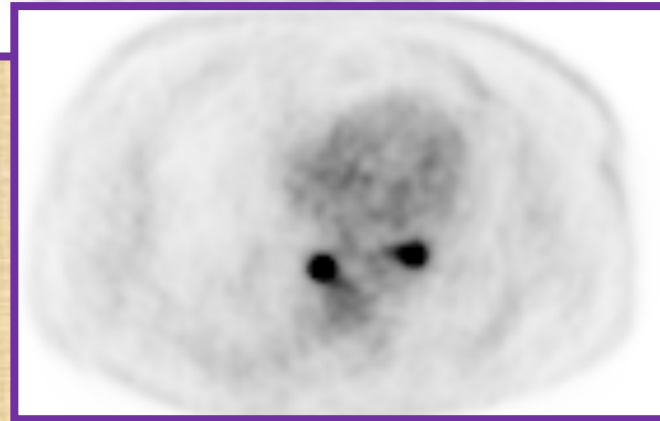
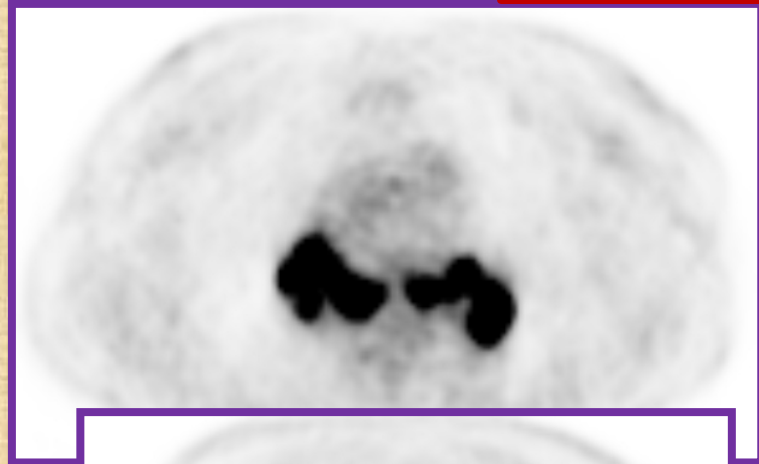




PET/CT
2/6/17

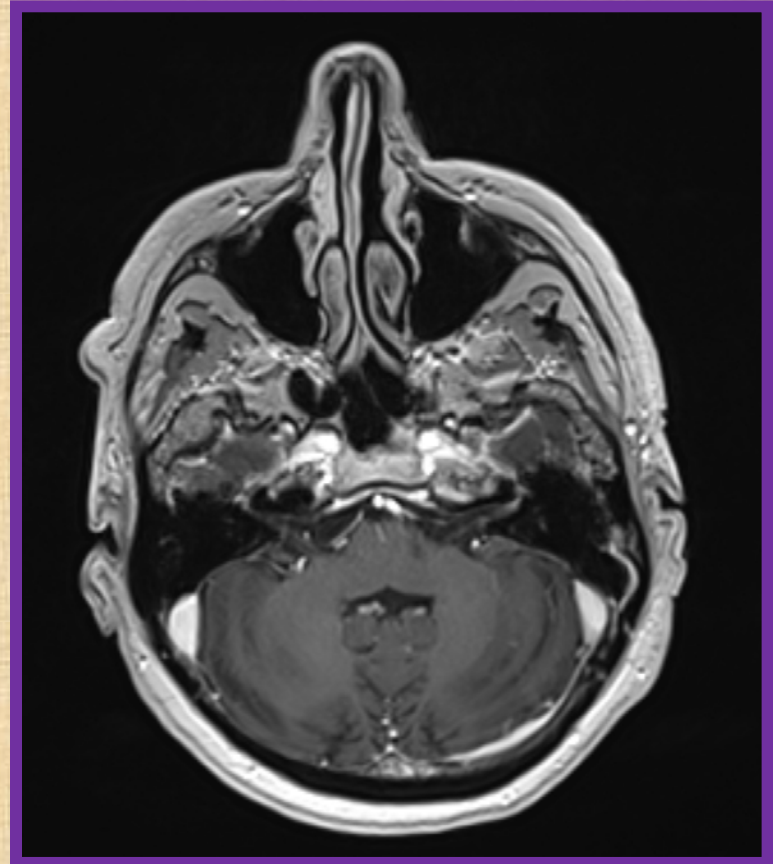
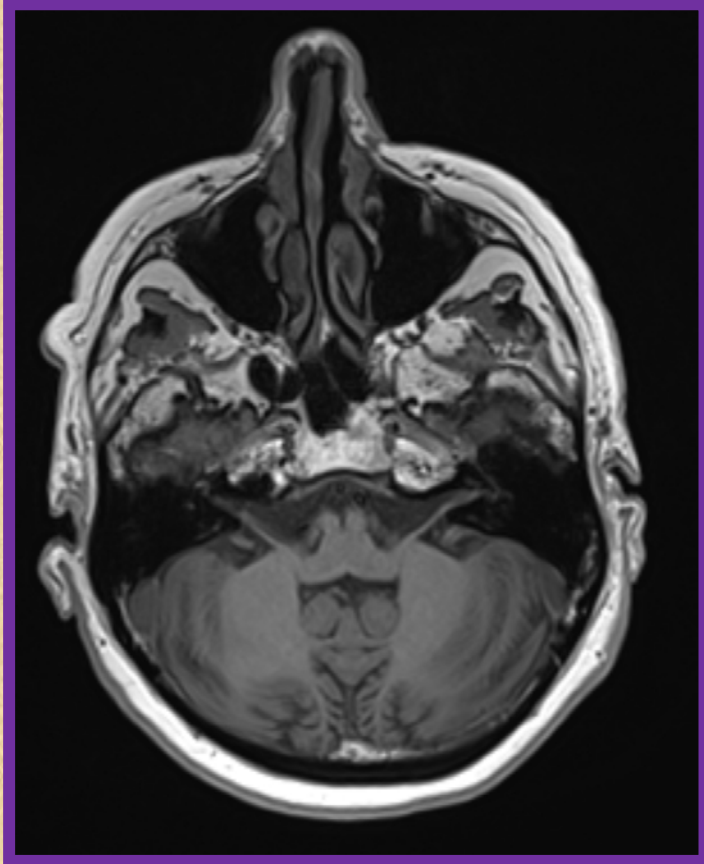


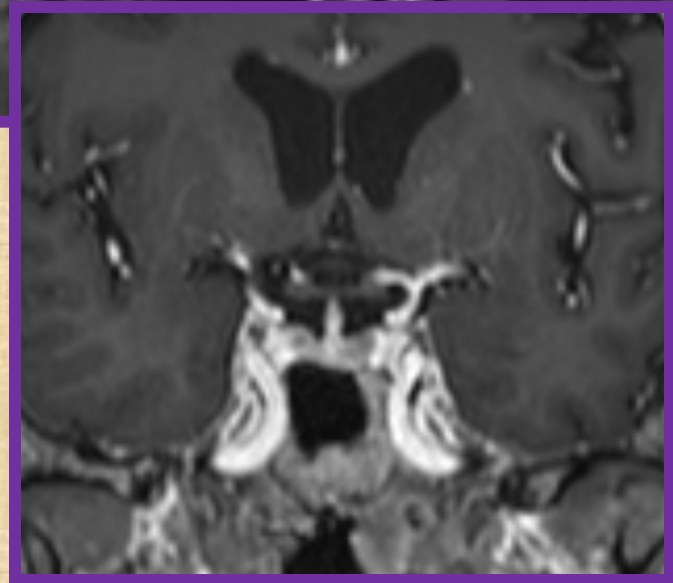
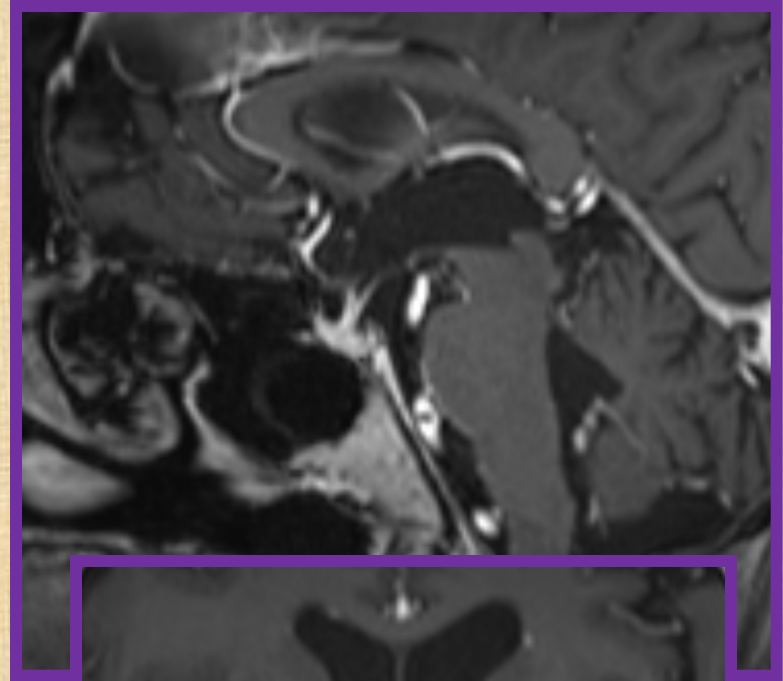
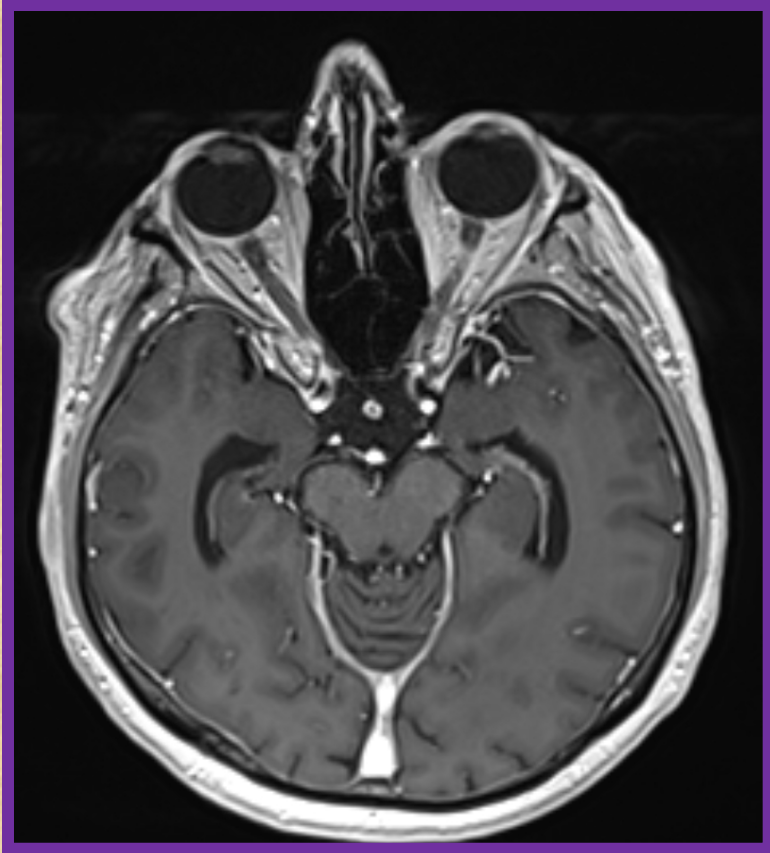
1,2,3 sign



Lambda sign

PET/CT
23/6/17





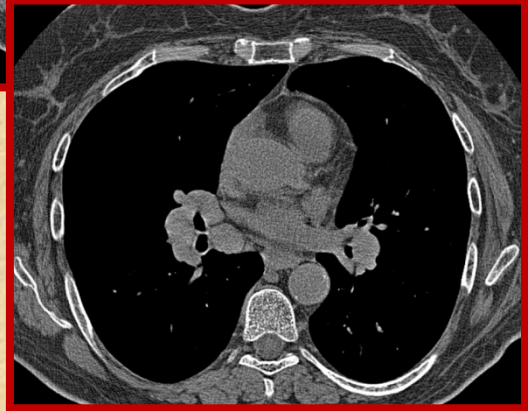
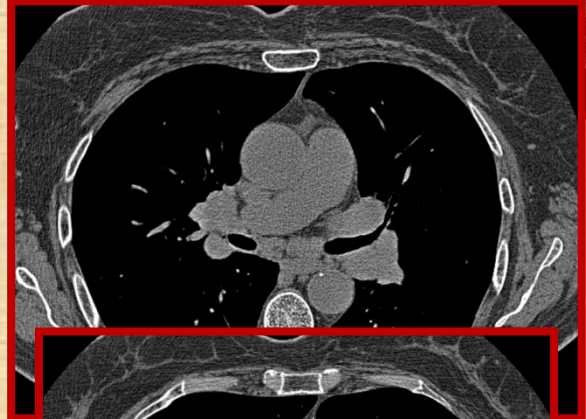
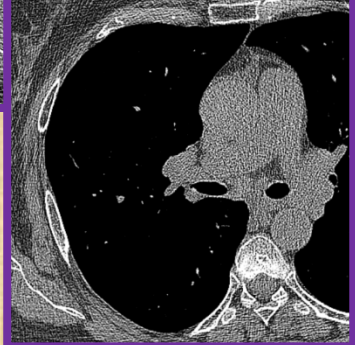
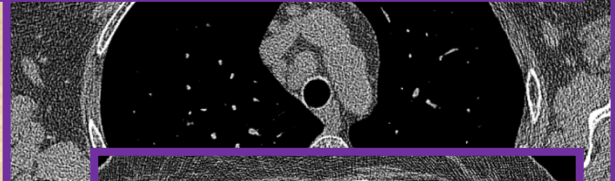
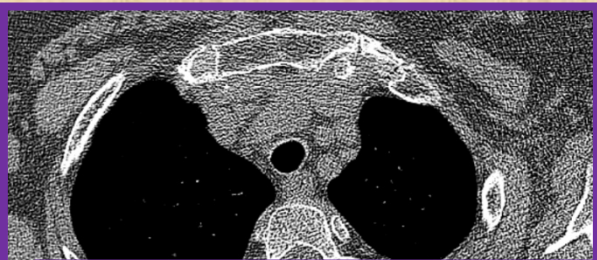
Neurosarcoidosis manifestation ^a	Prevalence	Comments
Cranial nerve palsy	31–55%	Facial and optic nerves are the most commonly affected; uni or bilateral involvement
Chronic aseptic meningitis	16–37%	Subacute or chronic lymphocytic meningitis; dural involvement including pachymeningitis, dural mass mimicking meningioma
Spinal cord disease/myelitis	18–23%	Subpial intramedullary lesions, typically longitudinally extensive; myelitis predilection cervicothoracic
Cerebral parenchymal disease	21%	Small cortical or periventricular white matter lesions; mimicking multiple sclerosis or micro-ischemic lesions, larger solitary aggregates of granulomas can masquerade as neoplasms
Neuroendocrine (hypothalamo-pituitary) involvement	6–9%	Hormonal disturbances including hypothyroidism, hypogonadism, panhypopituitarism, SIADH
Hydrocephalus	9–10%	Communicating and noncommunicating hydrocephalus; combination with leptomeningeal enhancement along the skull base
Cerebral infarction	6%	Stroke can be because of in situ thrombosis, compression of a large vessel by a granulomatous mass, sinovenous thrombosis, and intracerebral hemorrhage
Peripheral nervous system	17%	Large fiber involvement: most commonly axonal distal sensorimotor polyneuropathy or asymmetric polyradiculoneuropathy (nonlength dependent distribution)

SIADH, syndrome of inappropriate antidiuretic hormone.

^aone individual patient can have one or more neurosarcoidosis manifestation(s).

2η. ΥΤ
10/5/18

Χωρίς διαφοροποίηση ως προς τα ευρήματα του
πνευμονικού παρεγχύματος.



Radiologic Studies to Diagnose Sarcoidosis

STUDY	FINDINGS
Chest computed tomography	Useful particularly for the differential diagnosis of diffuse interstitial changes in lung parenchyma and pulmonary fibrosis
Chest radiography	Bilateral hilar lymphadenopathy and interstitial changes, necessary for staging
¹⁸ F-fluorodeoxyglucose positron emission tomography	Useful for finding areas to biopsy
	May aid in the diagnosis of cardiac sarcoidosis
	May correlate with active inflammation and disease activity
Gallium scan	Lambda sign: increased uptake in the bilateral hilar and right paratracheal lymph nodes
	Panda sign: increased uptake in the parotid and lacrimal glands
	Combined lambda and panda signs may be specific for sarcoidosis
Magnetic resonance imaging	Central nervous system: useful for identification of lesions
	Cardiac magnetic resonance imaging:
	Findings include focal intramyocardial zones of increased signal intensity due to edema and inflammation
	Delayed gadolinium enhancement is a predictor of ventricular arrhythmias and poor outcomes
Thallium scan	In cardiac sarcoidosis:
	Nonspecific areas of decreased myocardial uptake, not delimited by coronary artery distribution
	Improvement or reverse distribution following dipyridamole (Persantine) administration allows differentiation from coronary artery disease

Radiologic Staging of Sarcoidosis

STAGE	CHEST RADIOGRAPHY RESULTS	RATES OF SPONTANEOUS RESOLUTION	SUGGESTED FOLLOW-UP TIMELINE*
0	Normal	—	—
I	Bilateral hilar lymphadenopathy	55% to 90%	Initially every six months, then annually if stable
II	Bilateral hilar lymphadenopathy and pulmonary infiltrates	40% to 70%	Every three to six months indefinitely for stages II, III, and IV
III	Pulmonary infiltrates without bilateral hilar lymphadenopathy	10% to 20%	
IV	Pulmonary fibrosis	0% to 5%	

Nunes H, et al. *Semin Respir Crit Care Med.* 2007;28(1):102-120.
 Sobic-Saranovic D, et al. *Semin Nucl Med.* 2013;43(6):404-411.
 Am J Respir Crit Care Med. 1999;160(2):736-755.
 Soto-Gomey N., et al. *Am Fam Physician.* 2016 May 15;93(10):840-850.