Βιβλιογραφική Ενημέρωση

Σταδιοποίηση όγκων του Θύμου
Αντιμετώπιση αυτόματου πνευμοθώρακα

Αθανάσιος Κλέωντας MD, MSc
Χειρουργός Θώρακος

Thymic Epithelial Tumors Staging

Follow-Up Study of Thymomas with Special Reference to Their Clinical Stages

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Follow-up data were obtained for 96 cases of thymoma. The one-year survival rate was 84.3%, the three-year 77.1%, the five-year 74.1%, and the ten-year 57.1%. The five-year survival rate of total resection group was 88.9%; that of non-radically treated group was 44.4%. Clinical stages were defined: Stage I—macroscopically encapsulated and microscopically no capsular invasion; Stage II—1. macroscopic invasion into surrounding fatty tissue or mediastinal pleura, or 2. microscopic invasion into capsule; Stage III—macroscopic invasion into neighboring organ; Stage IVa—pleural or pericardial dissemination; Stage IVb—lymphogenous or hematogenous metastasis. Five-year survival rates of each clinical stage were 92.6% in Stage I, 85.7% in Stage II, 69.6% in Stage III, and 50% in Stage IV. Recurrence after total resection was found in six of 69 cases. Seven of 13 patients treated by subtotal resection survived more than five years with postoperative radiotherapy.

Cancer 48:2485–2492, 1981.

IT HAS BEEN POINTED OUT that there are two categories of thymoma—benign and malignant—, but that histologic identification of malignant thymoma is difficult.[1,2,3] In our experience of 96 cases of thymoma since 1954, we have observed that some thymomas showed invasive growth, pleural dissemination and extrathoracic metastasis in their late clinical courses, even though they had demonstrated a noninvasive character either grossly or histologically in their early periods.

Such biologic behavior of thymoma indicates that in its clinical course this tumor grows at first locally, then infiltratively or disseminatively and finally metastatically. This observation led us to the idea that determining the clinical stage at the start of therapy would aid in the selection of appropriate therapeutic methods, the evaluation of operative results and the establishment of prognosis. In this paper we propose clinical staging criteria for thymoma with special emphasis on the therapy and prognosis.

Materials and Methods

Our study was based on 96 cases of thymoma treated in Osaka University Hospital from January 1954 to April 1979.

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We define thymoma according to Rosai and Levine's definition,[15] namely, that thymoma is an epithelial tumor originating from thymus gland. Germinal tumor, malignant lymphoma, carcinoid tumor and anaplastic carcinoma are excluded from this definition, even if they originated from thymus. Histologically, we classified thymoma in two ways. Based on the shape of neoplastic epithelial cell: thymomas are divided into polygonal cell, spindle cell, mixed cell and clear cell types (Figs. 1A–1D). Based on the ratio of lymphocytes to epithelial cells, thymomas are divided into predominatly epithelial, equal ratio, and predominantly lymphocytic groups. Our clinical staging is shown in Table 1.

General Findings

Age: Patients' ages ranged from 8 months to 67 years. The peak was in the fourth decade (Fig. 2).

Sex: Fifty-six patients were male and 40 were female. Myasthenia gravis (MG) was more frequently associated with thymoma in female patients (Table 2).

TABLE 1. Criteria of Clinical Stages

Stage I:	Macroscopically completely encapsulated and microscopically no capsular invasion
Stage II:	1. Macroscopic invasion into surrounding fatty tissue or mediastinal pleura, *or* 2. Microscopic invasion into capsule
Stage III:	Macroscopic invasion into neighboring organ, i.e., pericardium, great vessels, or lung
Stage IVa:	Pleural or pericardial dissemination
Stage IVb:	Lymphogenous or hematogenous metastasis

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Staging System of Thymoma

Akira Masaoka, MD, PhD

Introduction: Thirty years have gone by since the Masaoka staging system of thymoma was proposed in 1981. Although the Masaoka staging system has been accepted by many surgeons and pathologists, some proposals of revision and improvements have been suggested. At this time, I reinvestigated the Masaoka staging system based on the recent follow-up study of the thymomas resected at Nagoya City University.

Methods: Using the follow-up results of 211 thymomas in Nagoya, I analyzed the following aspects: (1) evaluation of the Masaoka staging system as a prognostic factor in the Nagoya series and (2) critical assessment of the proposals of revision to the Masaoka staging system.

Results: (1) Univariate analysis showed that Masaoka stages were significantly prognostic for overall survival ($p < 0.0001$). (2) The difference of survivals between stage I and II was not significant, but progression-free survival of stage I was 100% for up to 20 years, whereas one tumor death case in stage II was found. (3) Differences of survival between the cases with and without great vessel invasion in stage III were not significant. (4) Prognosis of N^+ tumors was yet better defined.

Conclusion: (1) The Masaoka staging system remains a valuable prognostic factor. (2) Combination of stage I with II and separation of stage III into subgroups are not recommended. (3) At the moment, it is better to include N^+ tumors in stage IVb.

Key Words: Thymoma, Staging system, TNM classification, Follow-up study.

(*J Thorac Oncol.* 2010;5: S304–S312)

In the 1960s, thymomas were classified into two categories: noninvasive and invasive. Bernatz et al.[1] proposed a histologic classification and described four histologic subtypes: predominantly lymphocytic type, predominantly epithelial type, predominantly mixed type, and predominantly spindle cell type. Tumors belonging to the first three categories consisted of round epithelial cells and lymphocytes with differences in the ratio of the two components determining the category that the tumor belonged to. Bernatz et al. found that the predominantly epithelial subtype of thymoma was the most dominant among invasive thymomas.

To quantify the ratio of epithelial cells and lymphocytes, we[2] calculated the numbers of these cells and determined the lymphocytes to epithelial cells ratio (L/E ratio). In our series of 22 cases, L/E ratios ranged from 0.27 to 4.10 with a mean value of 1.93 (Figure 1).

We also found a significant difference in the L/E ratios of invasive and noninvasive thymomas (Figure 2) with invasive thymomas having a lower ratio than noninvasive thymomas. This had also been recognized by Bernatz et al. In our series, we found three cases of recurrent thymoma with L/E ratios that were strikingly lower as compared with tumors at the time of diagnosis (Figure 3). Hence, it seemed that the L/E ratio changed based on the biologic behavior of the tumor: a decrease in the L/E ratio seemed to correlate with a change in the nature of the tumor from encapsulated thymoma to invasive thymoma. This finding led to propose a staging system for thymomas.

MASAOKA STAGING SYSTEM

To establish a new staging system for thymomas, we took into consideration a few basic characteristics of the disease:

1. Thymoma is a very slow growing tumor. In our experience, it could take as long as 30 years for a noninvasive thymoma to become invasive if it was not surgically resected.
2. Some noninvasive thymomas that had been resected completely recurred in the same region.
3. Some patients with completely resected invasive thymomas could survive.
4. Pleural dissemination is a frequent site of progression.
5. Some patients develop lymphogenous or hematogeneous metastases.

Two other staging systems were developed by Bergh et al.[3] and Wilkins and Castleman[4] (Table 1). The difference between the two systems was limited to one point: stage II Wilkins thymoma involved pleura and pericardial invasion.

In our opinion, there were some major inadequacies in the staging systems of Bergh et al. and Wilkins and Castleman: (1) the actual sites of intrathoracic metastasis were not clear, (2) the description of invasion was inadequate, (3) stage III includes too broad spectrum of disease, and (4) hematogenous and lymphogenous metastases were not well represented.

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TABLE 6. The *p* Values of Stage vs. Survival

Author	City	Publish	No. of Case	Survival	*p*	Remarks
Pescarmona et al.[10]	Rome	1990	83	Overall	<0.001	
Regnard et al.[11]	Le Plessis- Robinson	1996	307	Disease free	<0.00001	
Gripp et al.[12]	Düsseldorf	1998	70	Disease free	0.0001	
Wilkins et al.[13]	Baltimore	1999	136	Overall	0.123	
				Thymoma related	0.290	
Lardinois et al.[14]	Lausanne	2000	71	Overall	<0.05	
				Disease free	<0.0001	
Ogawa et al.[15]	Multi institutional	2002	103	Overall	<0.0001	
				Disease free	<0.0001	
Okumura et al.[16]	Osaka	2002	273	Tumor specific	<0.0001	
Nakagawa et al.[17]	Tokyo	2003	130	Overall	0.000	Modified Masaoka
Kondo et al.[18]	Tokushima	2004	100	Disease free	0.002	
Park et al.[19]	Seoul	2004	150	Overall	<0.001	Thymic EP tumor
Rea et al.[20]	Padua	2004	132	Overall	0.003	Thymic EP tumor
Zhu et al.[21]	Shanghai	2004	175	Overall	<0.0001	
				Disease free	<0.0001	
Kim et al.[22]	Seoul	2005	108	Tumor specific	0.000	
Rena et al.[23]	Torino	2005	178	Overall	0.036	
				Disease free	0.012	
Mineo et al.[24]	Rome	2005	88	Overall	0.001	
				Disease free	0.0001	
Wright et al.[25]	Boston	2005	179	Tumor specific	<0.0001	Thymic EP tumor
Bedini et al.[26]	Milan	2005	123	Progression free	0.0001	

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TABLE 1. Staging System by Bergh et al. and Wilkins and Castleman[3,4]

Stage	Description
Staging system by Bergh et al.	
Stage I	Intact capsule or growth within the capsule
Stage II	Pericapsular growth into the mediastinal fat tissue
Stage III	Invasive growth into the surrounding organs, intrathoracic metastases, or both
Staging system by Wilkins and Castleman	
Stage I	Intact capsule or growth within the capsule
Stage II	Pericapsular growth into the mediastinal fat tissue or adjacent pleura or pericardium
Stage III	Invasive growth into the surrounding organs, intrathoracic metastases, or both

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TABLE 10. The Istituto Nazionale Tumori TNM-Based Staging System (Bedini et al.[26])

T1	No capsular invasion			
T2	Microscopic invasion into the capsule, or extracapsular involvement limited to the surrounding fatty tissue or normal thymus			
T3	Direct invasion into the mediastinal pleura and/or anterior pericardium			
T4	Direct invasion into neighboring organs, such as sternum, great vessels, and lungs; implants to the mediastinal pleura or pericardium, only if anterior to phrenic nerves			
N0	No lymph node metastasis			
N1	Metastasis to anterior mediastinal lymph nodes			
N2	Metastasis to intrathoracic lymph nodes other than anterior mediastinal			
N3	Metastasis to prescalene or supraclavicular nodes			
M0	No hematogenous metastasis			
M1a	Implants to the pericardium or mediastinal pleura beyond the sites defined in the T4 category			
M1b	Hematogenous metastasis to other sites, or involvement of lymph nodal stations other than those described in the N categories			
Stage grouping				
I	Locally restricted disease	T1–2	N0	M0
II	Locally advanced disease	T3–4	N0	M0
		Any T	N1–2	M0
III	Systemic disease	Any T	N3	M0
		Any T	Any N	M1
Classification of residual disease				
R0	No residual tumor			
R1	Microscopic residual tumor			
R2a	Local macroscopic residual tumor after reductive resection (>80% of the tumor)			
R2b	Other features of residual tumor			

Adapted from *Ann Thorac Surg* 2005;80:1994–2000.

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TABLE 9. WHO Staging System: TNM Classification

T-Primary tumor			
TX	Primary tumor cannot be assessed		
T0	No evidence of primary tumor		
T1	Tumor completely encapsulated		
T2	Tumor invades pericapsular connective tissue		
T3	Tumor invades into neighboring structures, such as pericardium, mediastinal pleura, thoracic wall, great vessels, and lung		
T4	Tumor with pleural or pericardial dissemination		
N-Regional lymph nodes			
NX	Regional lymph nodes cannot be assessed		
N0	No regional lymph node metastasis		
N1	Metastasis in anterior mediastinal lymph nodes		
N2	Metastasis in other intrathoracic lymph nodes excluding anterior mediastinal lymph nodes		
N3	Metastasis in scalene and/or supraclavicular lymph nodes		
M-Distant metastasis			
MX	Distant metastasis cannot be assessed		
M0	No distant metastasis		
M1	Distant metastasis		
Stage grouping			
Stage I	T1	N0	M0
Stage II	T2	N0	M0
Stage III	T1	N1	M0
	T2	N1	M0
	T3	N0, 1	M0
Stage IV	T4	Any N	M0
	Any T	N2, 3	M0
	Any T	Any N	M1

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EPILOGUE

The Masaoka staging system still remains a valuable and reproducible prognostic factor of thymoma. However, some proposals of revision of the staging system have been offered, to identify significant differences in survival between each identified stage. In my opinion, the staging system should obey the following principles:

1. It should be logically justified.
2. It should be simple to use.
3. Frequent revisions should be avoided.

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Detterbeck FC, Stratton K, Giroux D, Asamura H, Crowley J, Falkson C, Filosso PL, Frazier AA, Giaccone G, Huang .. Thorac Oncol. 2014 Sep;9(9 Suppl 2):S65-72

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1. The **IASLC/ITMIG Thymic Epithelial Tumors Staging Project**: proposals for the N and M components for the forthcoming (8th) edition of the TNM classification of malignant **tumors**.

   Kondo K, Van Schil P, Detterbeck FC, Okumura M, Stratton K, Giroux D, Asamura H, Crowley J, Falkson C, Filosso PL, Giaccone G, Huang J, Kim J, Lucchi M, Marino M, Marom EM, Nicholson AG, Ruffini E; **Staging** and Prognostic Factors Committee.; Members of the Advisory Boards.; Participating Institutions of the **Thymic** Domain..

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†††See Appendix 1;‡‡‡see Appendices 2, 3, 4;§§§see Appendix 5.

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Thymic Epithelial Tumors Staging

tion throughout the world. At least 15 different stage classification systems have been proposed and used.[1] These have been largely empirically derived, based on data from small numbers of patients. Perhaps the most widely used have been the Masaoka classification (derived from data on 91 patients),[2] and the Koga modification of this (based on 76 patients).[3] Even among centers using one of these classification systems, often the definitions have been interpreted differently because of vague wording, thus hampering the ability to collaborate effectively.[4]

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Stage classification

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Thymic Epithelial Tumors Staging

North and South American

European

Korean

Chinese

Institutions (105)

Japanese Association for Research on the Thymus (JART) database

European Society of Thoracic Surgeons (ESTS) database

10,808 patients

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Overall survival (OS)
Recurrence

as endpoints

Priorities

system that was simple
applicable to clinical staging
able to be used consistently

Thymoma + Thymic Carcinoma

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Of these, 2663 of the patients (25%) were excluded

missing endpoints in 1921 [18%],
date errors in 62,
first treatment before 1990 in 258 [2%],
missing stage or diagnosis data in 422 [4%]

leaving 8145 of patients for analysis

Most of the cases were first treated between 2000 and 2010

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TABLE 3. Stage Grouping

Stage	T	N	M
I	T1	N0	M0
II	T2	N0	M0
IIIa	T3	N0	M0
IIIb	T4	N0	M0
IVa	T any	N1	M0
	T any	N0,1	M1a
IVb	T any	N2	M0,1a
	T any	N any	M1b

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TABLE 1. T Categories and Descriptors

T	Descriptors
T1	A tumor that either is limited to the thymus with or without encapsulation, directly invades into the mediastinum only or directly invades the mediastinal pleura but does not involve any other mediastinal structure For further testing, T1 is subdivided into T1a (no mediastinal pleural involvement) and T1b (direct invasion of the mediastinal pleura) *(Level 1 structures—thymus, anterior mediastinal fat, mediastinal pleura)*
T2	A tumor with direct invasion of the pericardium (either partial or full-thickness) *(Level 2 structures—pericardium)*
T3	A tumor with direct invasion into any of the following: lung, brachiocephalic vein, SVC, phrenic nerve, chest wall, or extrapericardial pulmonary artery or veins *(Level 3 structures—lung, brachiocephalic vein, SVC, phrenic nerve, chest wall, hilar pulmonary vessels)*
T4	A tumor with invasion into any of the following: aorta (ascending, arch, or descending), arch vessels, intrapericardial pulmonary artery, myocardium, trachea, esophagus *(Level 4 structures—aorta [ascending, arch, or descending], arch vessels, intrapericardial pulmonary artery, myocardium, trachea, esophagus)*

T categories are defined by "levels" of invasion; they reflect the highest degree of invasion regardless of how many other (lower level) structures are invaded.
SVC, superior vena cava.

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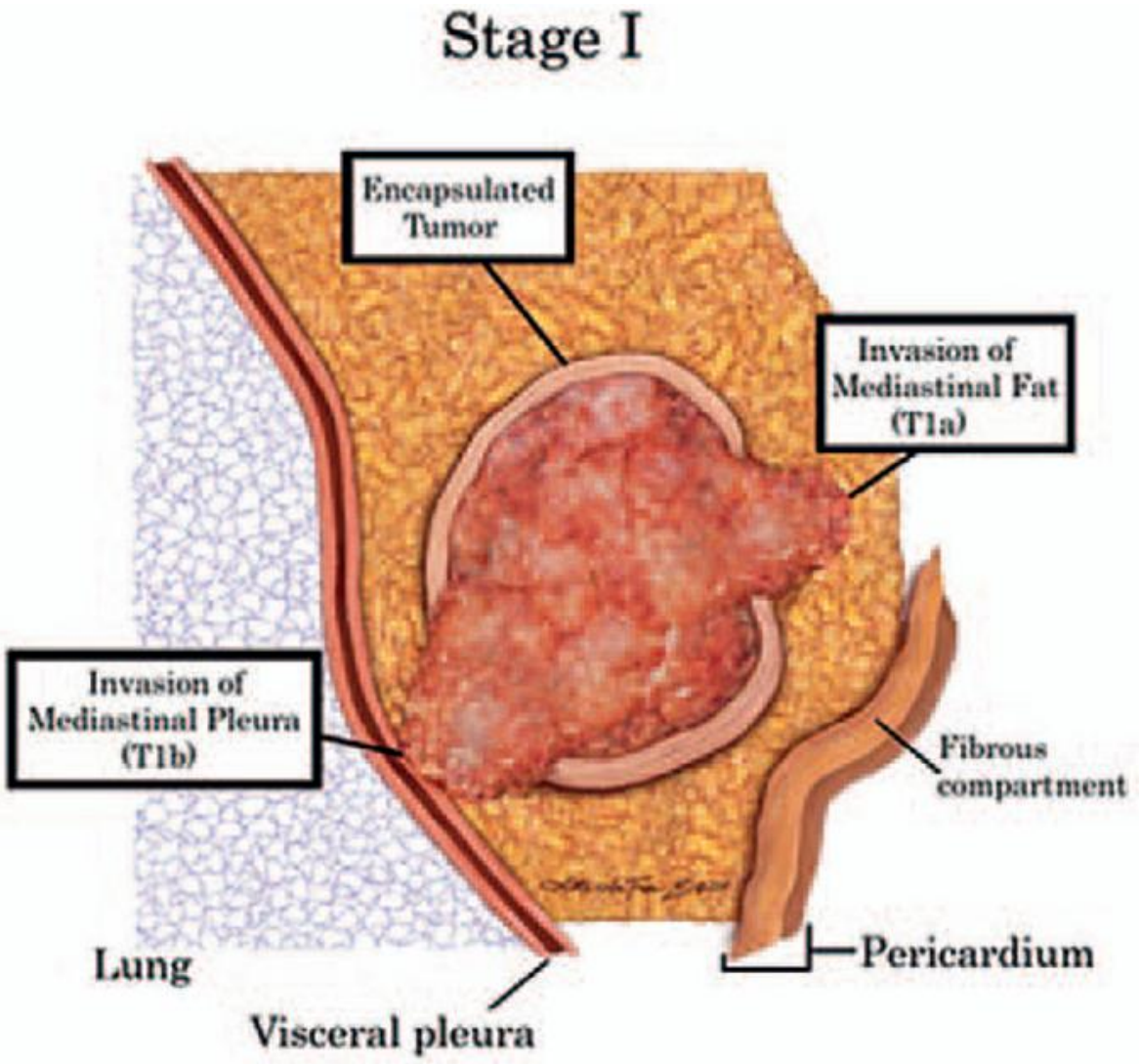

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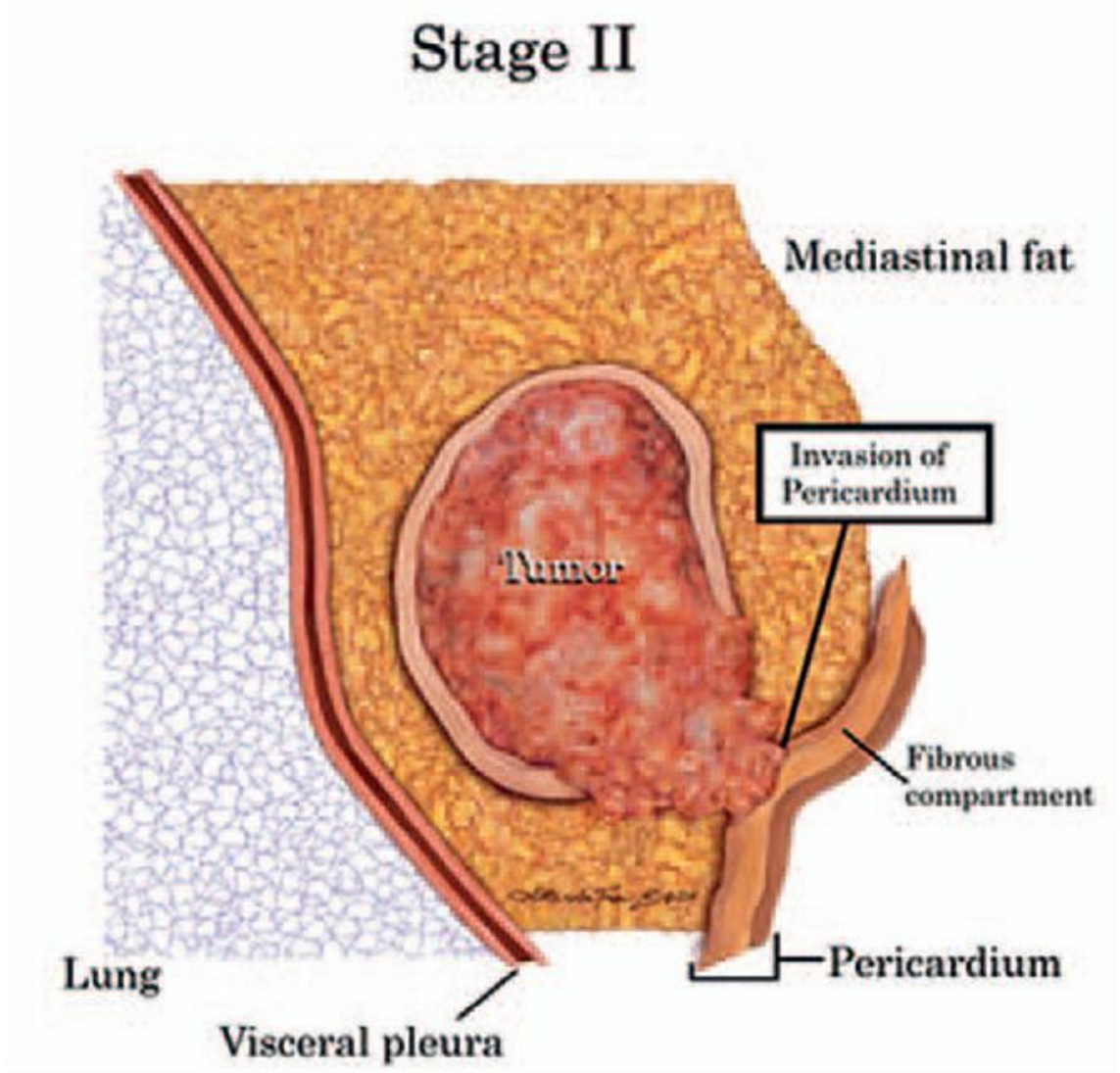

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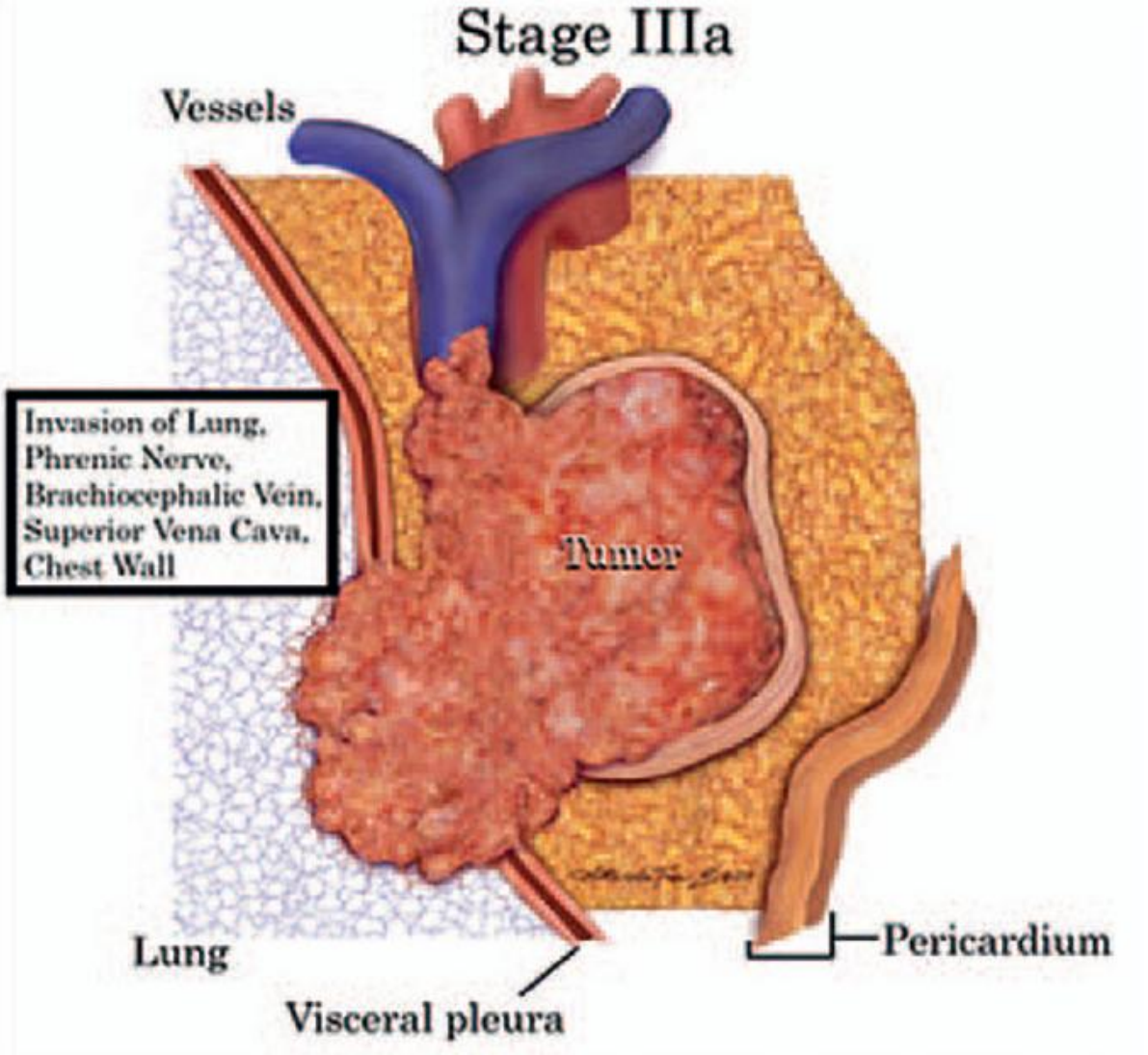

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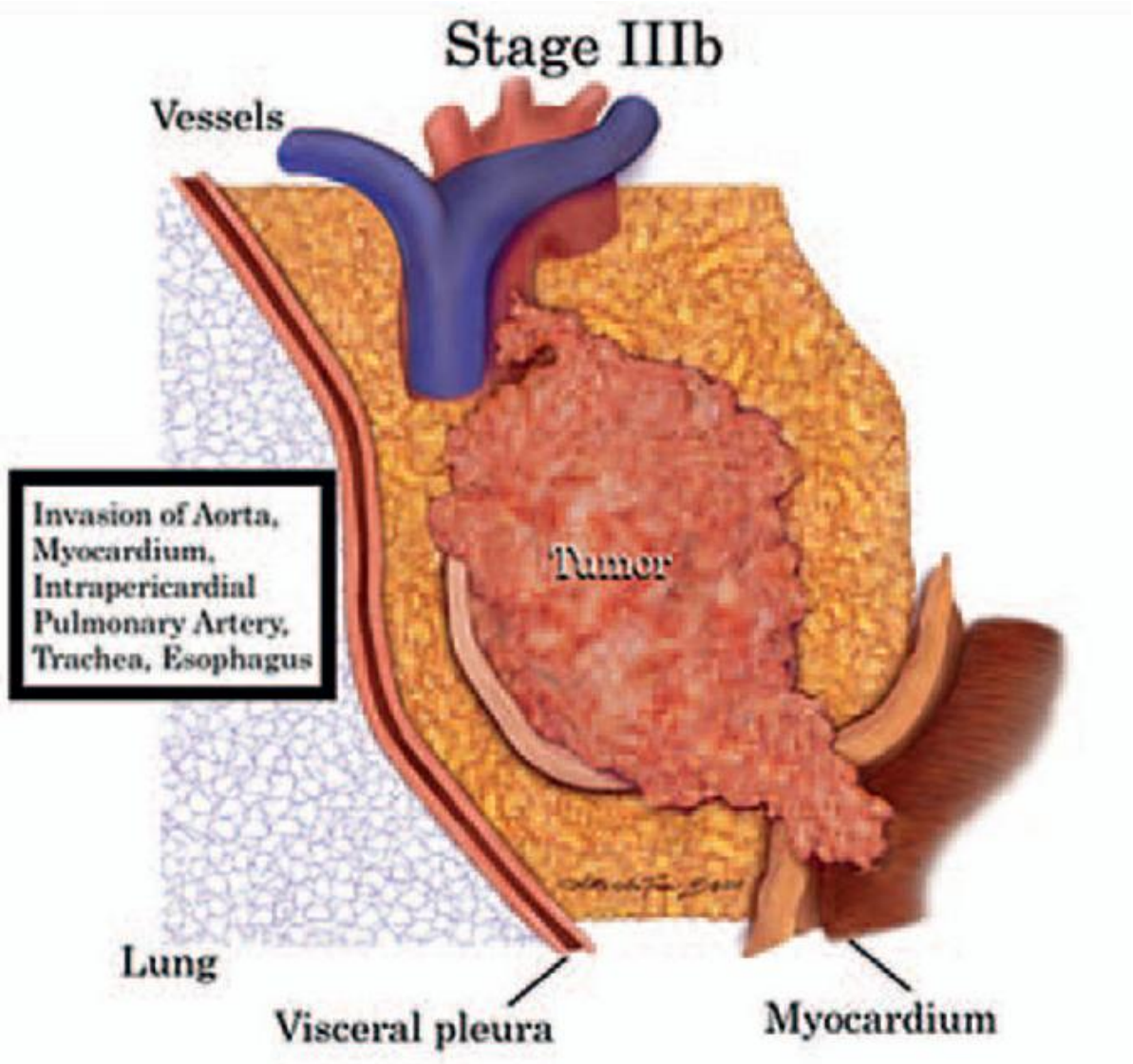

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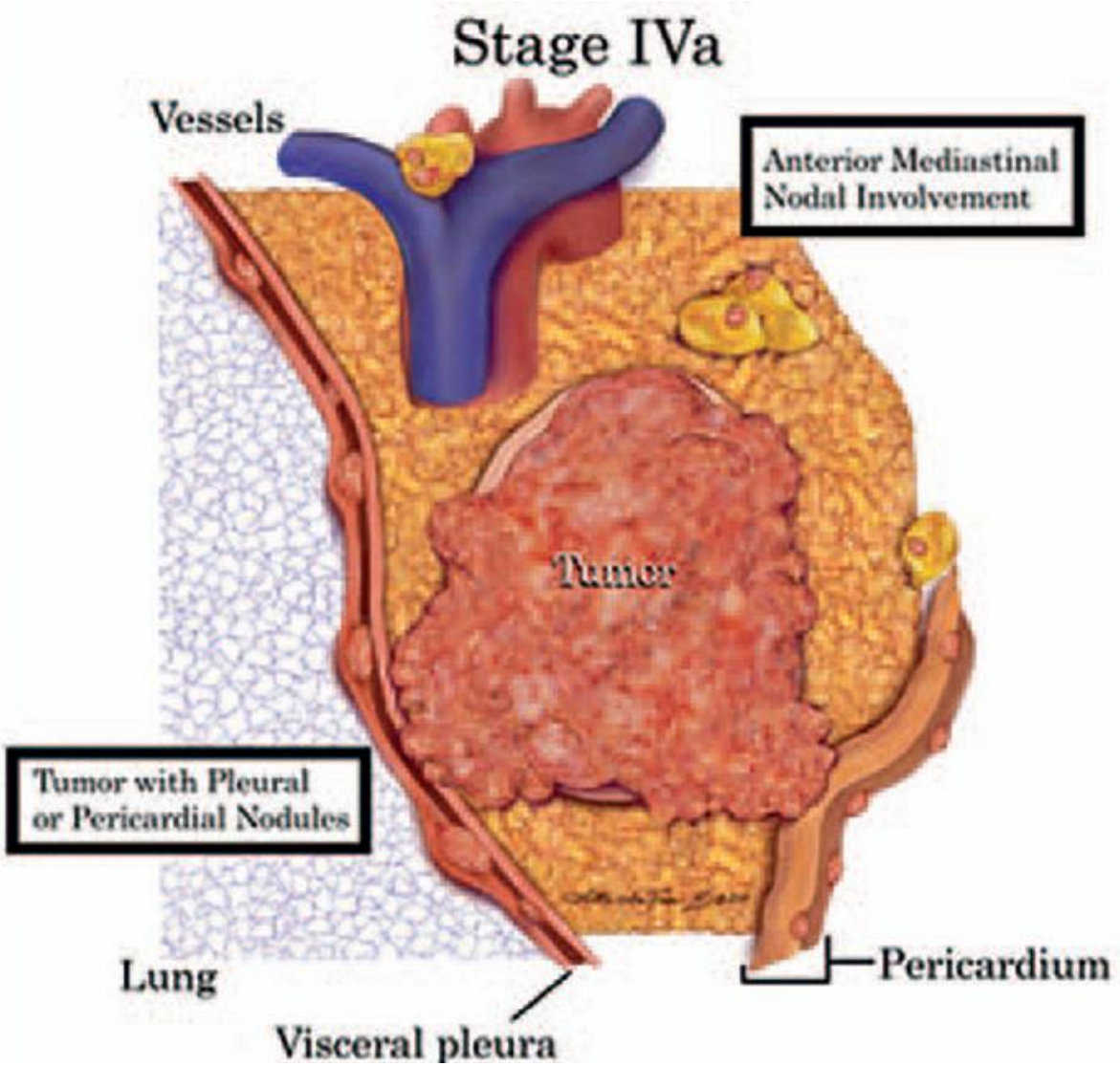

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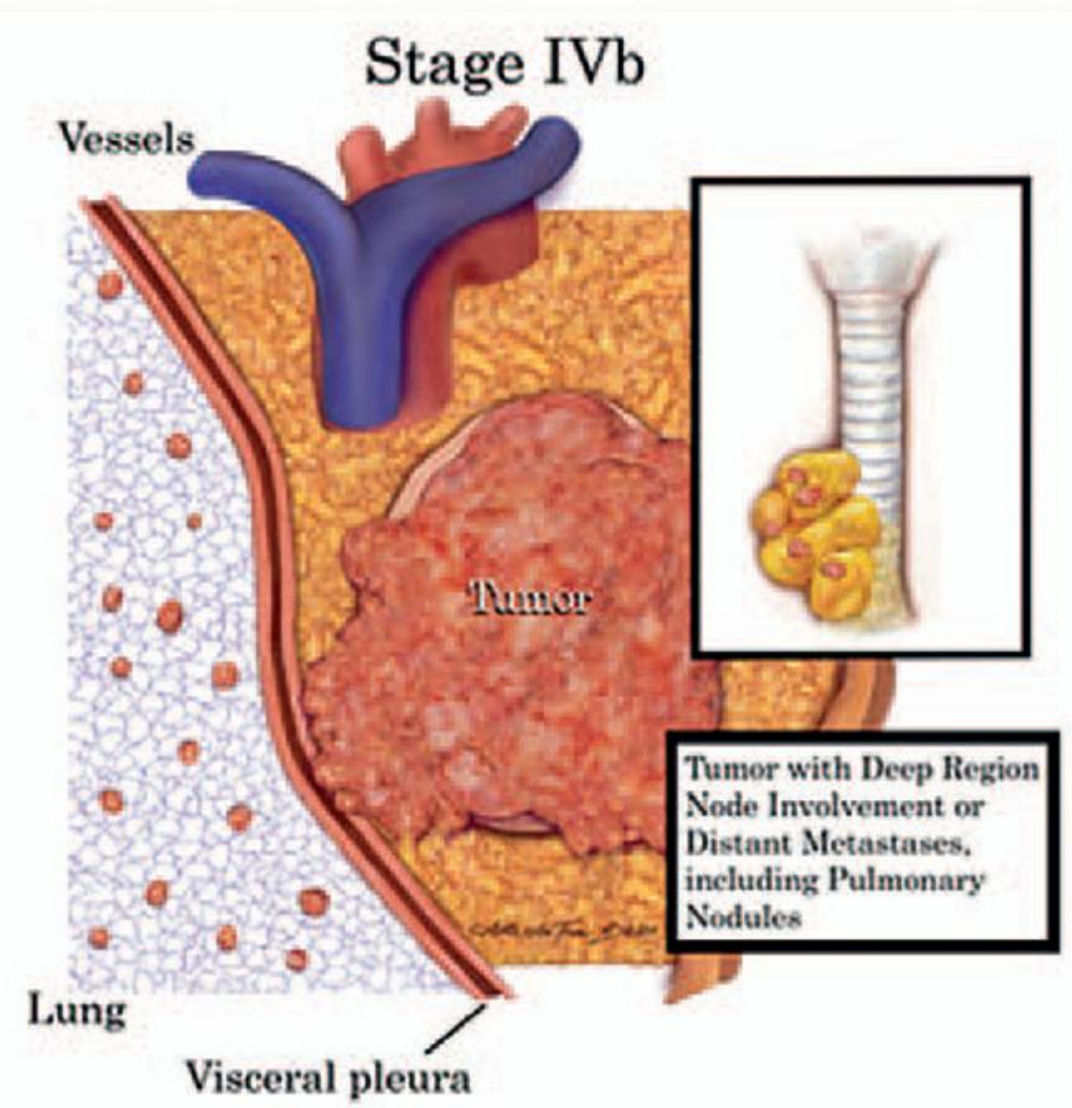

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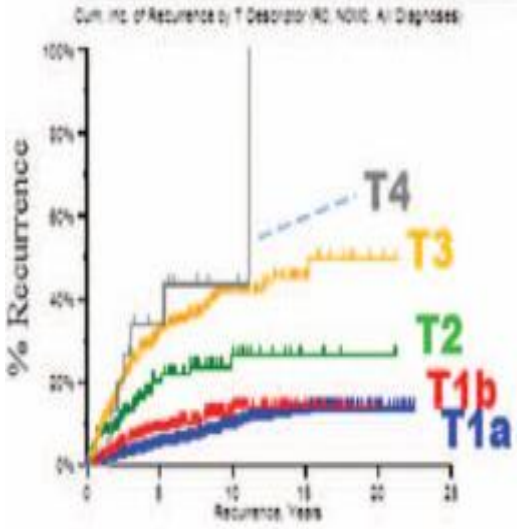
Thymic Epithelial Tumors Staging

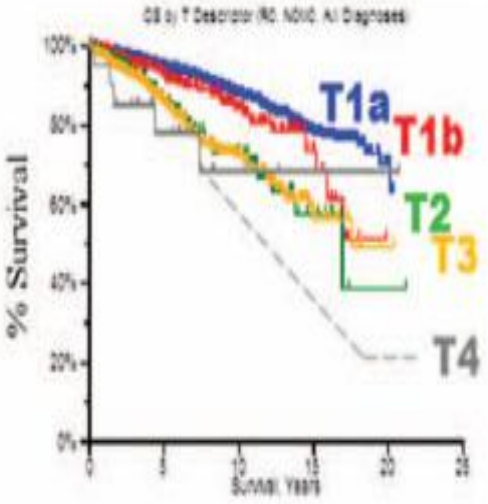
Outcomes of all Patients by T Categories

Recurrence, R0



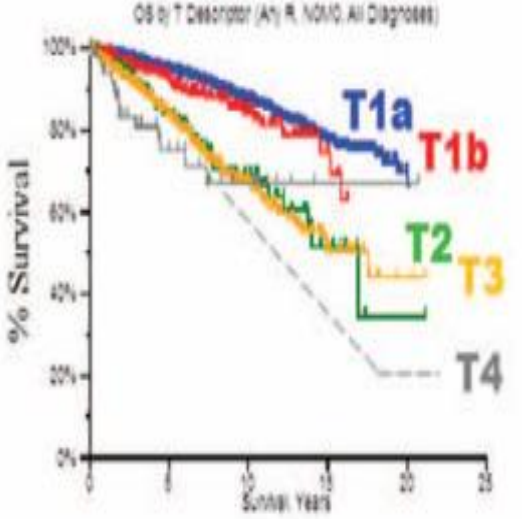
Stage	Events/N	5-Yr Estimate (CI)	10-Yr Estimate (CI)
T1a	168/3383	4.8% (4.8, 4.9)	9.8% (9.2, 9.7)
T1b	34/276	8.7% (6.2, 9)	12.3% (11.5, 13.1)
T2	22/124	20% (17.6, 22.8)	26.6% (22.1, 31.1)
T3	[illegible]	[illegible]	[illegible]
T4	7/18	34% (10.6, 57.4)	43.2% (20, 66.9)

Overall Survival, R0



Stage	Events/N	5-Yr Estimate (CI)	10-Yr Estimate (CI)
T1a	329/4818	95% (93.7, 96.3)	88% (86.4, 89.5)
T1b	34/219	93% (89.6, 96.2)	85% (79.5, 90.5)
T2	30/187	87% (80.3, 93.3)	73% (63.5, 83.1)
T3	[illegible]	[illegible]	[illegible]
T4	8/23	78% (58.7, 97.6)	68% (42.6, 93.1)

Overall Survival, R any



Stage	Events/N	5-Yr Estimate (CI)	10-Yr Estimate (CI)
T1a	367/5136	94% (93.6, 95.1)	87% (85.6, 88.7)
T1b	38/332	93% (89.4, 95.8)	84% (78.6, 90)
T2	43/239	84% (77.6, 90)	69% (59.7, 78.1)
T3	[illegible]	[illegible]	[illegible]
T4	13/37	79% (61.8, 88.1)	67% (50.8, 83.9)

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Thymic Epithelial Tumors Staging

TABLE 2. Total Proportion of Recurrences or Deaths

T Category	Recurrences		Deaths	
	%	n	%	n
T1	5	192/3659	7	363/5134
T1a	5	168/3383	7	329/4815
T1b	9	24/276	11	34/319
T2	18	22/124	16	30/187
T3	31	142/455	19	108/588
T3 single	25	59/240	19	65/335
T3 multiple	39	83/215	17	43/253
T4	39	55/1047/18	22	5/23
Total	10	363/4256	9	506/5932

The total number of recurrences or deaths observed at any time out of the total number of evaluable R0 resected patients in each category.

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Thymic Epithelial Tumors Staging

TABLE 4. Total Proportion of Recurrences or Deaths

Stage	Recurrences		Deaths	
	%	n	%	n
I	5	192/3659	7	363/5134
I (T1a)	5	168/3383	7	329/4815
I (T1b)	9	24/276	11	34/319
II	18	22/124	16	30/187
III	32	149/473	18	113/611
IIIa	31	142/455	18	108/588
IIIb	39	7/18	22	5/23
IVa	59	119/201	30	75/251
N1 M0	54	21/39	28	11/40
N0,1 M1a	60	98/162	30	64/211
IVb	49	17/35	33	14/43
N2 M0,1a, x	45	9/20	36	9/25
N0-2,x M1b	53	8/15	28	5/18
Total	11	499/4492	10	595/6226

The total number of recurrences or deaths observed at any time out of the total number of evaluable R0 resected patients in each category.

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Thymic Epithelial Tumors Staging

TABLE 3. Differences between T Categories

	CIR, R0 (363/4256)[a]		OS, R0 (506/5932)[a]		OS, any R (624/6561)[a]	
Variable	HR	*p*	HR	*p*	HR	*p*
HR vs. adjacent T category						
T2 vs. T1	3.10	<0.0001	2.05	0.0002	2.30	<0.0001
T3 vs. T2	1.67	0.025	1.03	NS	1.00	NS
T4 vs. T3	1.30	NS	1.00	NS	0.94	NS

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Thymic Epithelial Tumors Staging

TABLE 5. Differences between Stage Groups (all Diagnoses)

	CIR, R0 (499/4492)[a]		OS, R0 (595/6226)[a]		OS, any R (876/7314)[a]	
Variable	HR	*p*	HR	*p*	HR	*p*
HR vs. adjacent stage						
II vs. I	3.21	<0.0001	2.05	0.0002	2.28	<0.0001
IIIa vs. II	1.72	0.02	1.03	NS	1.00	NS
IIIb vs. IIIa	1.30	NS	1.01	NS	0.94	NS
IVa vs. IIIb	1.67	NS	1.72	NS	2.00	0.02
IVb vs. IVa	0.77	NS	1.29	NS	1.26	NS

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Thymic Epithelial Tumors Staging

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*Pathology, Royal Brompton Hospital, London, United Kingdom; †Thoracic Surgery, Yale University, New Haven, Connecticut; ‡Pathology, Regina Elena National Cancer Institute, Rome, Italy; §Thoracic Surgery, Samsung Medical Center, Seoul, South Korea; ‖Biostatistics, Cancer Research And Biostatistics, Seattle, Washington; ¶Thoracic Surgery, National Cancer Center Hospital, Tokyo, Japan; # Radiation Oncology, Queen's University, Ontario, Canada; **Thoracic Surgery, University of Torino, Torino, Italy; ††Medical Oncology, Georgetown University, Washington, District of Columbia; ‡‡Thoracic Surgery, Sloan Kettering Cancer Center, New York, New York; §§Thoracic Surgery, University of Tokushima, Tokushima, Japan; ‖‖Thoracic Surgery, University of Pisa, Pisa, Italy; ¶¶Radiology, MD Anderson Cancer Center, Houston, Texas; ##Thoracic Surgery, Osaka University, Osaka, Japan; and ***Thoracic Surgery, Antwerp University Hospital, Antwerp, Belgium.

†††See Appendix 1; ‡‡‡see Appendices 2, 3, and 4; and §§§see Appendix 5.

Disclosure: The authors declare no conflict of interest.

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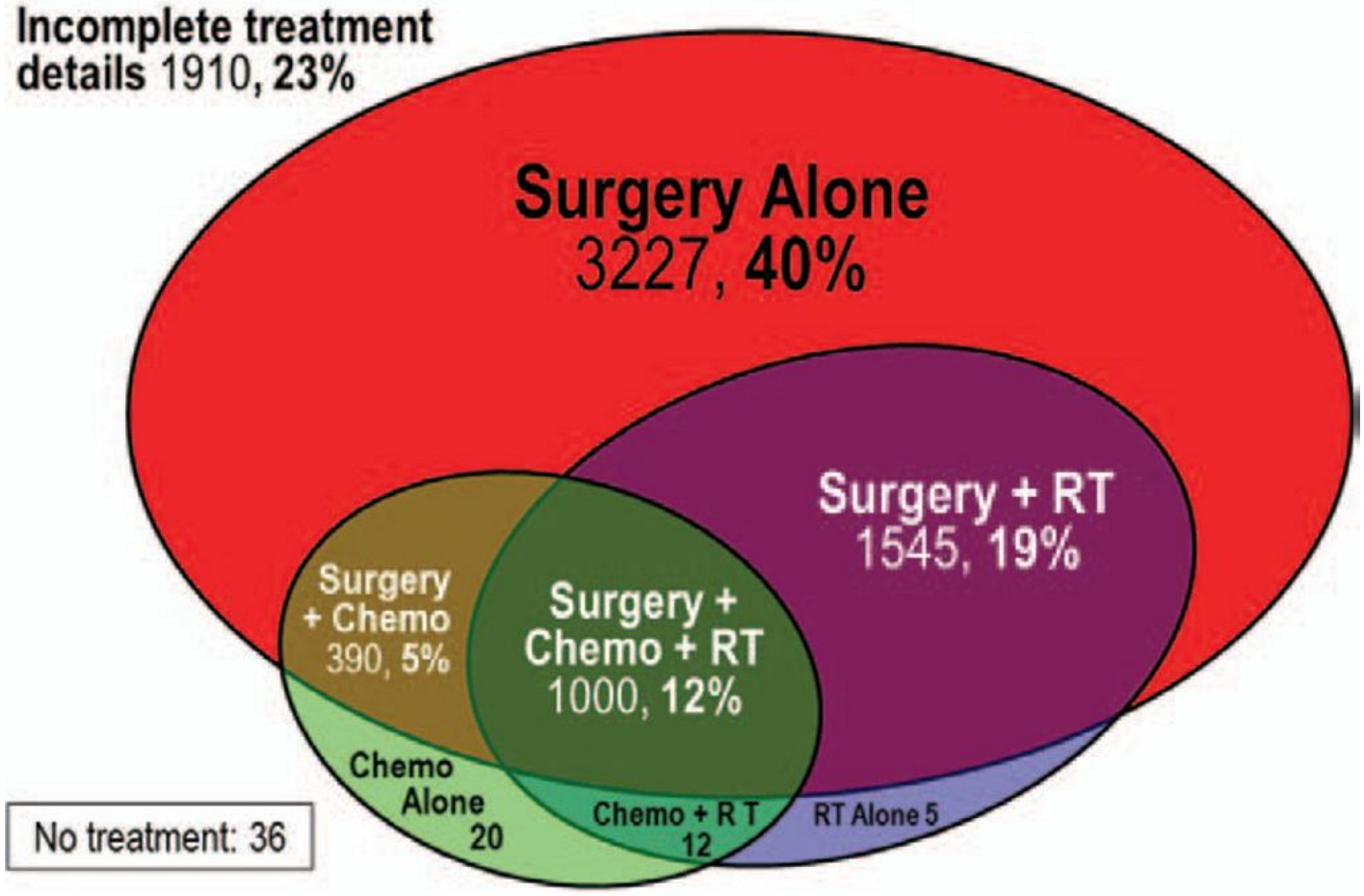
Thymic Epithelial Tumors Staging

ITMIG/IASLC Retrospective Database
Treatment Modalities, 8,145 screened cases



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Thymic Epithelial Tumors Staging

TABLE 1. N, M Descriptors

Category	Definition (Involvement of)[a]
N0	No nodal involvement
N1	Anterior (perithymic) nodes
N2	Deep intrathoracic or cervical nodes
M0	No metastatic pleural, pericardial, or distant sites
M1	
a	Separate pleural or pericardial nodule(s)
b	Pulmonary intraparenchymal nodule or distant organ metastasis

[a]Involvement must be pathologically proven in pathologic staging.

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Thymic Epithelial Tumors Staging

nodal involvement
an anterior (perithymic, N1)
a deep (N2) category

The anterior region extends
from the hyoid bone to the diaphragm,
bounded anteriorly by the sternum,
posteriorly by the trachea (neck) and pericardium (chest),
And laterally
by the medial border of the carotid sheaths (neck)
and the mediastinal pleura (chest).

The distal boundaries of the deep region are defined
by the medial edge of the trapezius muscle (neck)
and the pulmonary hila (chest) laterally
and the esophagus and vertebral column posteriorly.
The deep region includes paratracheal, subcarinal, aortopulmonary window, hilar, jugular, and supraclavicular nodes.

Involved nodes outside these regions (e.g., axillary, subdiaphragmatic) are outside the N category and considered a distant metastasis.

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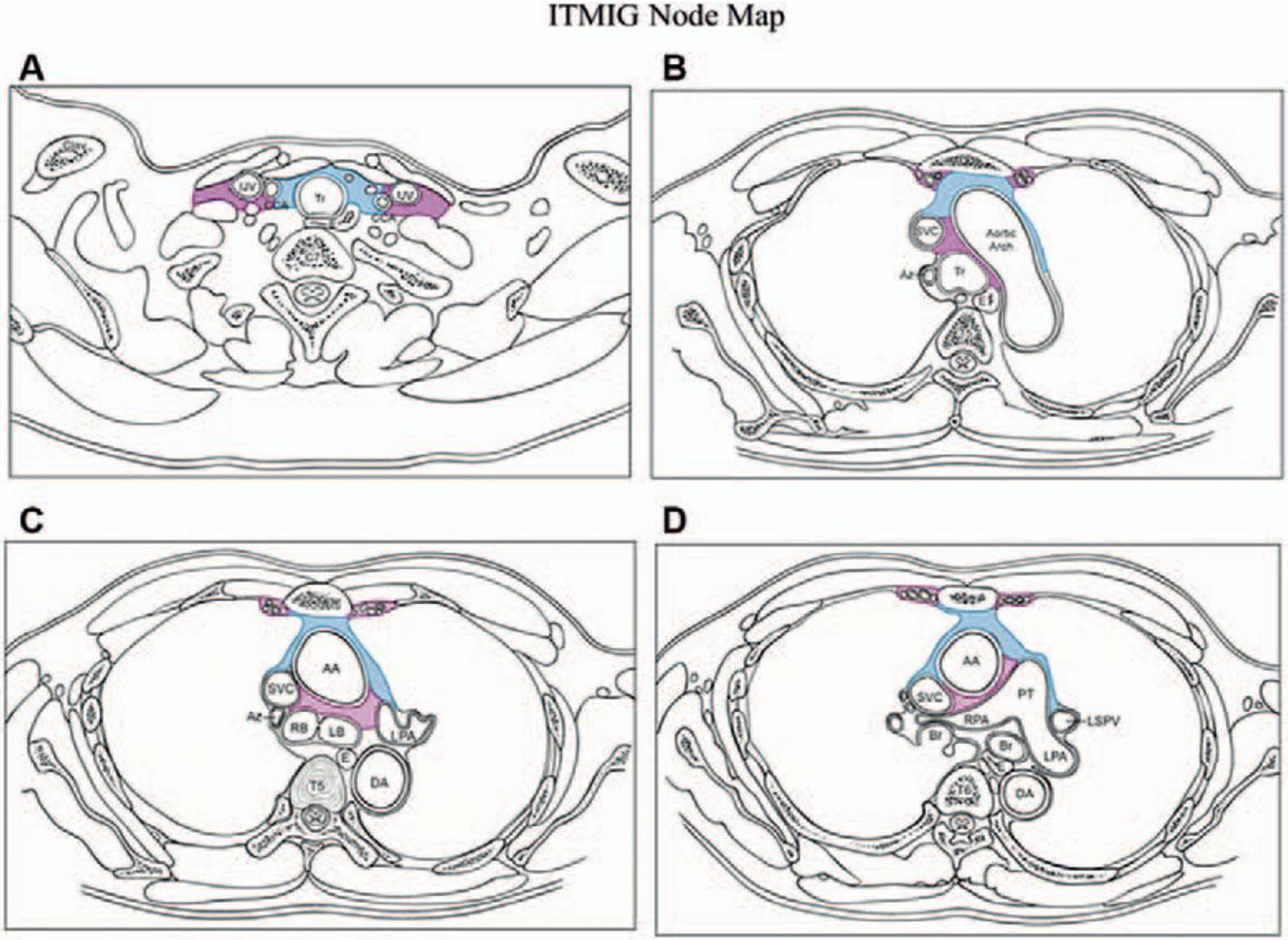

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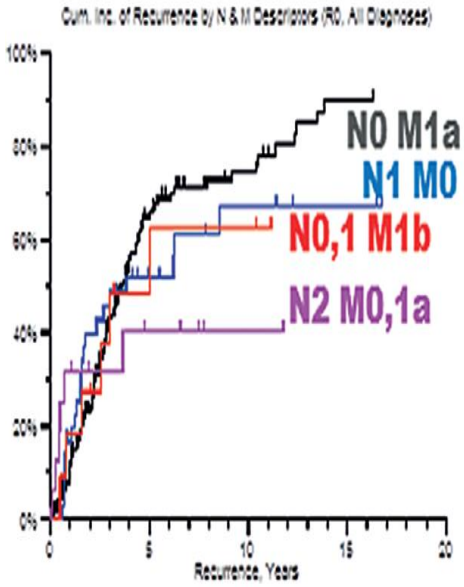
Thymic Epithelial Tumors Staging

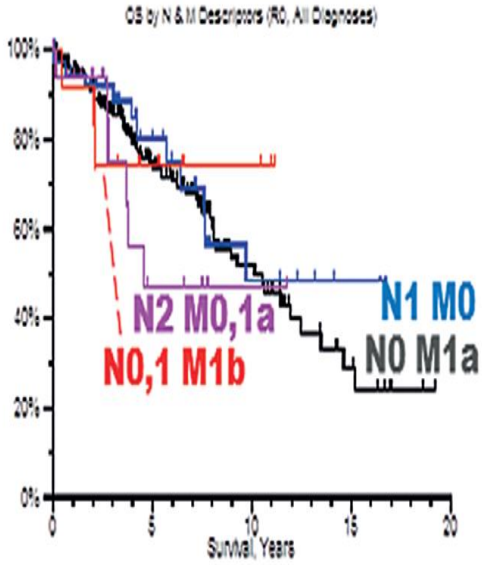
Outcomes of All Patients by Proposed N and M Categories

Recurrence, R0



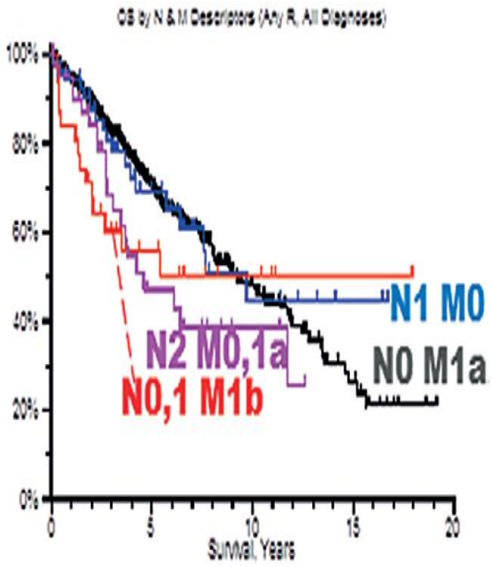
Stage	Events/N	5-Yr Estimate (CI)	10-Yr Estimate (CI)
T any N0 M1a	94/154	65% (45.3, 84.3)	75% (36.3, 100)
T any N1 M0	21/39	52% (31.8, 72)	67% (13.8, 100)
T any N0,1 M1b	6/11	49% (0, 100)	63% (0, 100)
T any N2 M0,1a	6/17	40% (19.6, 61.1)	40% (19.6, 61.1)

Overall Survival, R0



Stage	Events/N	5-Yr Estimate (CI)	10-Yr Estimate (CI)
T any N0 M1a	61/203	74% (66.2, 81.4)	52% (40.5, 63.2)
T any N1 M0	11/40	80% (65.7, 94.9)	49% (24.5, 72.5)
T any N0,1 M1b	3/12	74% (48.7, 99.4)	74% (48.7, 99.4)
T any N2 M0,1a	6/17	47% (17.2, 76.5)	47% (17.2, 76.5)

Overall Survival, any R



Stage	Events/N	5-Yr Estimate (CI)	10-Yr Estimate (CI)
T any N0 M1a	179/579	71% (65.9, 75.2)	48% (41.2, 54.6)
T any N1 M0	18/54	69% (54.5, 83.4)	45% (24.4, 64.7)
T any N0,1 M1b	14/31	56% (37.7, 74.2)	50% (30.9, 69.8)
T any N2 M0,1a	20/42	47% (29, 65.2)	39% (20.4, 56.7)

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Thymic Epithelial Tumors Staging

TABLE 2. Total Proportion of Recurrences or Deaths

	Recurrence, R0		Deaths, R0		Deaths, any R	
	%	Events/*n*	%	Events/*n*	%	Events/*n*
Stage IVa	59	119/201	30	75/251	32	209/654
N1 M0	54	21/39	28	11/40	33	18/54
N0 M1a	61	94/154	30	61/203	31	179/579
N1 M1a	50	4/8	38	3/8	57	12/21
Stage IVb	49	17/35	33	14/43	43	43/99
N2 M0,1a	35	6/17	35	6/17	48	20/42
N0,1 M1b	55	6/11	25	3/12	45	14/31
N2 M1b/X + NX M1b	71	5/7	36	5/14	35	9/26

The total number of recurrences or deaths observed at any time out of the total number of evaluable patients in each category.

R, resection status; R0, complete resection.

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Thymic Epithelial Tumors Staging

Weaknesses and limitations

The available data were heavily weighted toward surgical cases likely representing the greater ability of surgeons and pathologists to have collected data to contribute.

More advanced tumors are probably underrepresented, beyond their simple lower incidence compared to earlier stage tumors.

Furthermore, the limited availability of details despite the unprecedentedly large database means that some aspects had to be decided upon primarily by consensus after consideration of practical, anatomic and logical factors.

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ITMIG Standards and Definitions

A prerequisite for collaboration is the ability to speak the same language. Differences in the interpretation of terms were surprisingly wide in this field and largely unrecognised. The ITMIG community organized a broad process to clarify critical terms. Multiple workgroups were assembled,and core members drafted initial proposals, which were vetted with workgroup members.

At a 2-day workshop at Yale University with broad international representation, these definitions were discussed and revised so that they would be aligned with one another. These were then further refined by the workgroups with input from the entire ITMIG membership. The final documents were approved by the ITMIG membership for use in all ongoing research and publications. The level of engagement and broad consensus in this process was in itself a major accomplishment of ITMIG. These definitions were published in a supplement to the Journal of Thoracic Oncology (JTO) in July 2011 and are openly available for download from the ITMIG or JTO (www.jto.org) websites.

- ITMIG Standards-English.pdf
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Thymic Epithelial Tumors Staging

Introduction to Institutional Summary Kits of ITMIG Standard Definitions and Policies

Οι κακοήθειες του θύμου είναι σχετικά σπάνιες, για αυτο ειναι συμαντικη η συλλογη δεδομενων απο διαφορες πηγες (εργαστηρια, ινστιτουτα). Αυτό απαιτεί σταθεροτητα στη χρηση των όρων/ορισμων, τυποποίηση των βασικών πρακτικών και μια σταθερή βάση για την αναφορά των αποτελεσμάτων. Η Διεθνής Ομάδα μελετης κακοήθειων του θυμου (ITMIG) έχει αναπτύξει μια σειρά από ορισμούς που εχουν γινει ευρέως αποδεκτές από εμπειρογνώμονες του θυμου σε όλο τον κόσμο. Η πλήρης σειρά των άρθρων είναι διαθέσιμα σε μια σειρά από αρθρα στο Journal of Thoracic Oncology, 2011, Volume 7, Supplement 3 (http://www.itmig.org/?page_id=315).
Για να ειναι τα κυριότερα σημεία διαθέσιμα οταν χρειάζονται/ανα πασα στιγμη, η ITMIG έχει συγκεντρώσει μια σειρά από συνοπτικά δελτία που αφοτουν τις διαφορές ειδικότητες. Το παρον αρθρο ειναι φτιαγμενο για εκτύπωση διπλής όψης. Ιδανικα, επιμερους κομματια μπορουν να διανεμηθουν στους αντίστοιχους ειδικούς σε ένα ίδρυμα για να διευκολύνει την επικοινωνία τοσο μεσα στο ίδρυμα οσο και σε διεθνές επίπεδο. Αντιστοιχα, επιμερους κομματια μπορουν να γινουν download.

Thymic Epithelial Tumors Staging

IV **a** **Pleural or pericardial metastases**
μεταστάσεις στον υπεζωκότα ή το περικαρδο

Microscopically confirmed nodules, separate from the primary tumor, involving the visceral or parietal pleural surfaces, or the pericardial or epicardial surfaces,
Μικροσκοπικά επιβεβαιώθηκαν οζίδια, χωριστά από τον πρωτογενή όγκο, με τη συμμετοχή των επιφανιων του σπλαχνικού ή του περικαρδίου ή των επικαρδιακων επιφάνειων,

b **Lymphogenous or hematogenous metastasis**
Λυμφογενεις ή αιματογενεις μεταστασεις

Any nodal involvement (e.g. anterior mediastinal, intrathoracic, low/anterior cervical nodes, any other extrathoracic nodes)

Distant metastases (i.e. extrathoracic and outside the cervical perithymic region) or pulmonary parenchymal nodules (not a pleural implant)

Κάθε συμμετοχή γαγγλιων (π.χ. πρόσθιου μεσοθωρακίου, ενδοθωρακικο, χαμηλο / πρόσθιο γαγγλιο του τραχήλου της μήτρας, κάθε άλλο εξωθωρακικο γαγγλιο)

Απομακρυσμένες μεταστάσεις (εξωθωρακικές και έξω από την περιοχή του τραχήλου της μήτρας τον γυρω) ή

πνευμονική παρεγχυματική οζίδια (όχι εμφύτευμα του υπεζωκότα)

Thymic Epithelial Tumors Staging

Reference: Detterbeck F, Moran C, Huang J et al. Which Way is Up? Policies and Procedures for Surgeons and Pathologists Regarding Resection Specimens of Thymic Malignancy. J Thorac Oncol 2011;6(7,Suppl 3):S1730-8

Available for download at: http://www.itmig.org/?page_id=315
Recommended Routine Policies for Microscopic Findings:

ΜΕΧΡΙ ΕΔΩ ΕΧΩ ΠΡΟΛΑΒΕΙ

Thymic Epithelial Tumors Staging

clinical practice guidelines

Annals of Oncology 26 (Supplement 5): v40–v55, 2015
doi:10.1093/annonc/mdv277

Thymic epithelial tumours: ESMO Clinical Practice Guidelines for diagnosis, treatment and follow-up†

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clinical practice guidelines

incidence and epidemiology

Thymic epithelial tumours represent a heterogeneous group of rare thoracic cancers, with reported annual incidence ranging from 1.3 to 3.2 per million [1]. Thymic epithelial tumours are classified according to the World Health Organization (WHO) histopathological classification, which distinguishes thymomas from thymic carcinomas.

thymomas

Thymomas are further subdivided into different types (called A, AB, B1, B2, B3 and rare others) based upon the morphology of epithelial tumour cells, the relative proportion of the non-tumoural lymphocytic component (decreasing from type B1 to B3) and resemblance to normal thymic architecture (Table 1) [2, 3]. The term 'benign thymoma' should be avoided. Thymomas are far more frequent than thymic carcinomas, which have an incidence of 0.2 to 0.5 per million [3].

thymic carcinomas

Thymic carcinomas are similar to their extrathymic counterpart, the most frequent subtype being squamous cell carcinoma. Neuroendocrine tumours may occur in the thymus, and will not be discussed in these guidelines; while localised primary thymic neuroendocrine tumours may benefit from surgical resection, similar to other thymic carcinomas, the prognosis is poor given frequent recurrences; for recurrent, advanced and metastatic tumours, the management actually follows that of extra-thoracic neuroendocrine tumours.

epidemiology

Mean age at diagnosis is 50–60 years of age, but thymic tumours may actually be diagnosed in children as well as in elderly patients. There is no consistent gender predilection in thymomas overall, even if a slight female preponderance has been reported for type A, AB and B1 subtypes in most studies, and a male predominance in carcinomas [2–7].

No environmental or infectious factors have been demonstrated to play a role in the pathogenesis of thymic epithelial tumours. Reports on development of thymoma after radiation, solid-organ transplantation and immunosuppression, including the context of human immunodeficiency virus infection, are rare; differential diagnosis with thymic rebound hyperplasia may be discussed in this setting (see below).

Genetic risk factors, such as multiple endocrine neoplasia 1 (MEN1), may influence the development of thymomas, as well as thymic carcinoids, given their reported familial occurrence as well as their association with cancer susceptibility syndromes [8].

Moreover, extrathymic haematopoietic cancers (mostly diffuse large B-cell lymphoma and leukaemia) and a broad spectrum of solid cancers (stomach, pancreas, colon and thyroid) have been reported to occur more frequently in thymoma patients, particularly subsequently [9]. This might be related to a shared unknown oncogenic trigger, a thymoma-associated immune deficiency or (less likely) to adverse effects of treatments.

diagnosis

imaging and laboratory tests

Standard imaging for thymic tumours is i.v. contrast-enhanced computed tomography (CT) scan of the thorax, allowing a complete exploration of the mediastinum and the pleura from the apex to the costodiaphragmatic recesses [IV, A]. CT is equal or superior to magnetic resonance imaging (MRI) for the diagnosis of mediastinal anterior masses, except in the setting of cystic lesions [IV, B] [10].

One-third of patients with thymoma present with autoimmune disorders (Table 2), mainly myasthenia gravis which is particularly common in type AB, B1 and B2 thymomas and almost always associated with anti-acetylcholine receptor antibodies (Table 1) [11]. Other frequent disorders include pure red cell aplasia (5% of cases) and hypogammaglobulinaemia (Good syndrome: 5% of cases) [12].

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Thymic Epithelial Tumors Staging

distant metastatic invasion. The IASLC/ITMIG TNM system of thymic tumours will be incorporated as the official thymic tumour staging system into the 8th edition of the TNM staging system of thoracic malignancies expected in 2016–2017. From our standpoint, the Masaoka-Koga staging should remain the standard for the routine management of patients, pending the approval of the American Joint Committee on Cancer (AJCC) and Union for International Cancer Control (UICC) [III, A]. Moreover, given the major switch that the TNM system represents and the limited amount of fair level of evidence data to support our current treatment strategies (especially postoperative radiotherapy), the value of the TNM system to drive the therapeutic strategy has to be assessed. Correlative clinical data based on this system may be encouraged in a research setting.

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Thymic Epithelial Tumors Staging

Table 3. Staging of thymic epithelial tumours: Masaoka-Koga-based staging system [25, 26], International Thymic Malignancy Interest Group refinements [27] and overall survival and recurrence-free survival (range)[a] [28]

	Masaoka-Koga, 1994	International Thymic Malignancy Interest Group, 2011	10-year overall survival	10-year cumulative incidence of recurrence	
				Thymoma	Thymic carcinoma
Stage I	Grossly and microscopically completely encapsulated tumour	– Invasion into but not through the capsule – In the absence of capsule, absence of invasion into surrounding tissues	84% (81%–86%)		
Stage IIA	Microscopic transcapsular invasion	– Microscopic transcapsular invasion (<3 mm)	83% (79%–87%)	8% (7%–8%)	25% (22%–29%)
Stage IIB	Macroscopic invasion into thymic or surrounding fatty tissue, or grossly adherent to but not breaking through the mediastinal pleura or pericardium	– Gross extension into normal thymus or perithymic fat surrounding the tumour (microscopically confirmed) – Adherence to pleura or pericardium, with microscopic confirmation of perithymic invasion			
Stage III	Macroscopic invasion into neighbouring organ (i.e. pericardium, great vessel or lung)	– Microscopic invasion of the mediastinal pleura (either partial or penetrating the elastin layer) – Microscopic invasion of the pericardium (either partial in the fibrous layer or penetrating through to the serosal layer) – Microscopically confirmed direct penetration into the outer elastin layer of the visceral pleura or into the lung parenchyma – Invasion into the phrenic or vagus nerves (microscopically confirmed) – Invasion into or penetration through major vascular structures (microscopically confirmed) – Adherence (i.e. fibrous attachment) of lung or adjacent organs only if there is mediastinal pleural or pericardial invasion (microscopically confirmed)	70% (64%–75%)	29% (27%–31%)	59% (44%–76%)
Stage IVA	Pleural or pericardial metastasis	– Microscopically confirmed separate nodules in the visceral or parietal pleural, pericardial or epicardial surfaces	42% (26%–58%)	71% (34%–100%)	76% (58%–100%)
Stage IVB	Lymphogenous or haematogenous metastasis	– Lymphogenous or haematogenous metastasis	53% (32%–73%)	57% (24%–90%)	54% (37%–67%)

[a]Information reprinted from [27] with permission of John Wiley & Sons, Inc.

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Primary Spontaneous Pneumothorax

Open Access Protocol

BMJ Open

Study protocol for a randomised controlled trial of invasive versus conservative management of primary spontaneous pneumothorax

Simon G A Brown,[1,2] Emma L Ball,[1,3] Kyle Perrin,[4,5] Catherine A Read,[1] Stephen E Asha,[6,7] Richard Beasley,[4,5] Diana Egerton-Warburton,[8,9] Peter G Jones,[10] Gerben Keijzers,[11,12,13] Frances B Kinnear,[14,15] Ben C H Kwan,[16,17] Y C Gary Lee,[18,19] Julian A Smith,[20,21] Quentin A Summers,[22] Graham Simpson,[23] the PSP Study Group

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ABSTRACT

Introduction: Current management of primary spontaneous pneumothorax (PSP) is variable, with little evidence from randomised controlled trials to guide treatment. Guidelines emphasise intervention in many patients, which involves chest drain insertion, hospital admission and occasionally surgery. However, there is evidence that conservative management may be effective and safe, and it may also reduce the risk of recurrence. Significant questions remain regarding the optimal initial approach to the management of PSP.

Methods and analysis: This multicentre, prospective, randomised, open label, parallel group, non-inferiority study will randomise 342 participants with a first large PSP to conservative or interventional management. To maintain allocation concealment, randomisation will be performed in real time by computer and stratified by study site. Conservative management will involve a period of observation prior to discharge, with intervention for worsening symptoms or physiological instability. Interventional treatment will involve insertion of a small bore drain. If drainage continues after 1 hour, the patient will be admitted. If drainage stops, the drain will be clamped for 4 hours. The patient will be discharged if the lung remains inflated. Otherwise, the patient will be admitted. The primary end point is the proportion of participants with complete lung re-expansion by 8 weeks. Secondary end points are as follows: days in hospital, persistent air leak, predefined complications and adverse events, time to resolution of symptoms, and pneumothorax recurrence during a follow-up period of at least 1 year. The study has 95% power to detect an absolute non-inferiority margin of 9%, assuming 99% successful expansion at 8 weeks in the invasive treatment arm. The primary analysis will be by intention to treat.

Ethics and dissemination: Local ethics approval has been obtained for all sites. Study findings will be disseminated by publication in a high-impact international journal and presentation at major international Emergency Medicine and Respiratory meetings.

Trial registration number: ACTRN12611000184976; Pre-results.

Strengths and limitations of this study

- This will be the largest randomised trial of the management of large primary spontaneous pneumothorax (PSP) that includes long-term follow-up.
- The study includes an assessment of efficacy, safety, healthcare usage and patient-centred outcomes such as pain and return to work.
- The findings will provide high-level evidence for the optimum initial management of PSP and will be generalisable to clinical practice internationally.
- The nature of the treatments being compared means that blinding of participants to treatment allocation is not possible; however, the ability for patients to decline involvement combined with unbreakable allocation concealment means that participant and clinician bias towards one treatment modality or another is less likely to confound the study results.

INTRODUCTION

Primary spontaneous pneumothorax (PSP) is a significant global health problem affecting adolescents and young adults. The incidence of PSP is around 18–28/100 000 per year for men and 1.2–6/100 000 per year for women.[1 2] It usually occurs in the absence of underlying lung disease or trauma; however, anatomical abnormalities such as subpleural blebs are present in up to 90% of cases.[3 4] Tobacco smoking is a major risk factor, and otherwise healthy male smokers have a

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Respiratory medicine

Study protocol for a randomised controlled trial of invasive versus conservative management of primary spontaneous pneumothorax

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Primary Spontaneous Pneumothorax

Conservative treatment

Repeat assessment 90 min after randomisation

↓ Improved with analgesia & SpO_2 > 90% breathing room air

Final assessment 4 hours after randomisation with repeat CXR

↓ Observations stable (off O_2) regardless of pneumothorax size

Check can walk comfortably

↓ **Discharge**

Any of; RR>30, SpO_2 <90% on room air, SBP<90, HR≥ SBP, or intolerable symptoms such that patient is unwilling to continue

Trend in obs suggests developing tension (e.g. ↑HR, ↑RR) or intolerable pain or breathlessness so that patient is unwilling to continue

Unable to walk (i.e. not capable of activities of daily living)

→ **Consider switch to interventional treatment** (or longer period of observation)

Flow diagram: Conservative treatment

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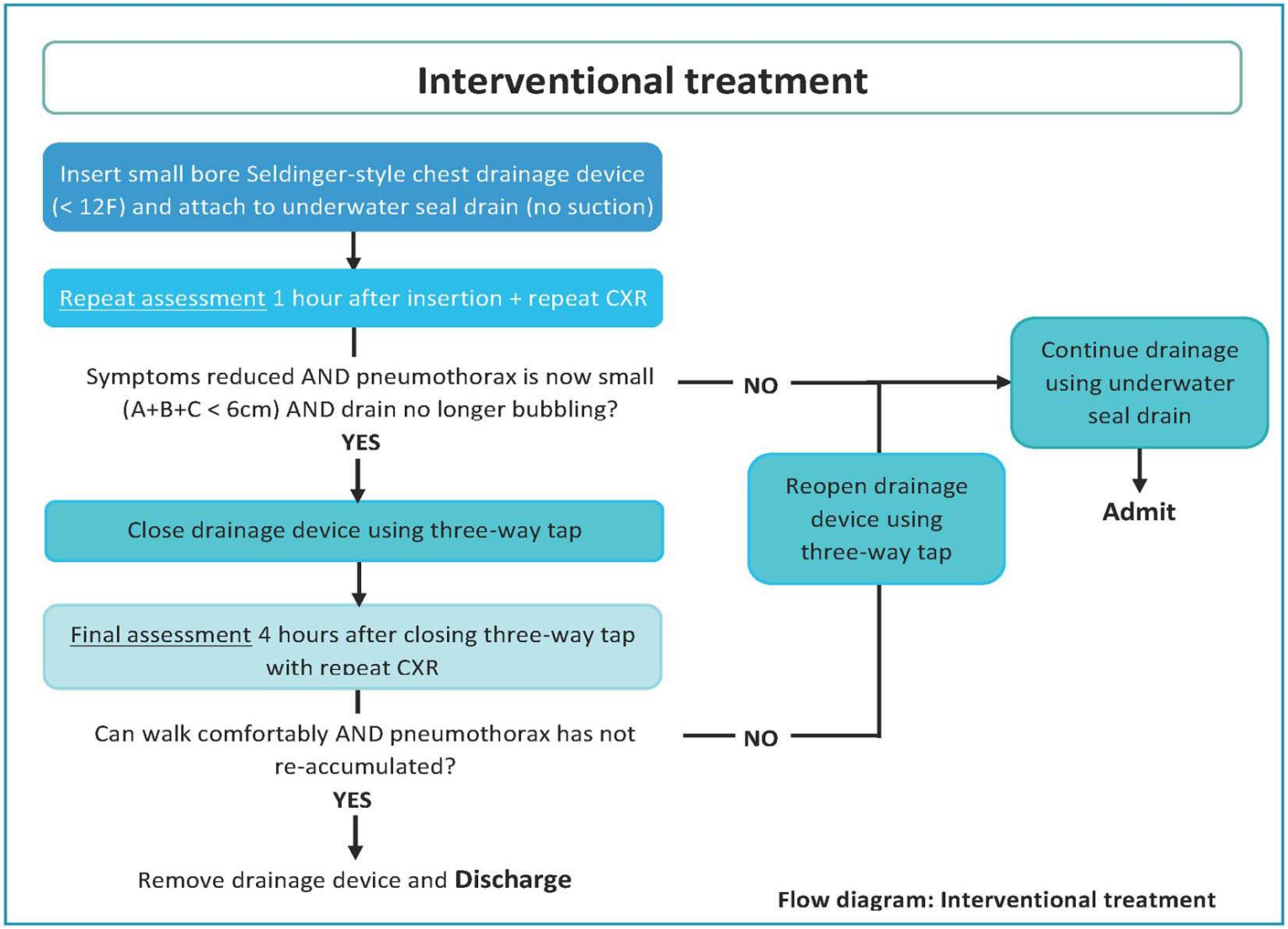
Primary Spontaneous Pneumothorax



Flow diagram: Interventional treatment

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Primary Spontaneous Pneumothorax

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ORIGINAL ARTICLE

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Surgical management of spontaneous pneumothorax: are there any prognostic factors influencing postoperative complications?

Jean-Philippe Delpy[a], Pierre-Benoit Pagès[a,*], Pierre Mordant[b], Pierre-Emmanuel Falcoz[c], Pascal Thomas[d], Francoise Le Pimpec-Barthes[e], Marcel Dahan[f] and Alain Bernard[a] on behalf of the EPITHOR project (French Society of Thoracic and Cardiovascular Surgery)

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Abstract

OBJECTIVES: There are no guidelines regarding the surgical approach for spontaneous pneumothorax. It has been reported, however, that the risk of recurrence following video-assisted thoracic surgery is higher than that following open thoracotomy (OT). The objective of this study was to determine whether this higher risk of recurrence following video-assisted thoracic surgery could be attributable to differences in intraoperative parenchymal resection and the pleurodesis technique.

METHODS: Data for 7647 patients operated on for primary or secondary spontaneous pneumothorax between 1 January 2005 and 31 December 2012 were extracted from Epithor®, the French national database. The type of pleurodesis and parenchymal resection was collected. Outcomes were (i) bleeding, defined as postoperative pleural bleeding; (ii) pulmonary and pleural complications, defined as atelectasis, pneumonia, empyema, prolonged ventilation, acute respiratory distress syndrome and prolonged air leaks; (iii) in-hospital length of stay and (iv) recurrence, defined as chest drainage or surgery for a second pneumothorax.

RESULTS: Of note, 6648 patients underwent videothoracoscopy and 1004 patients underwent OT. When compared with the thoracotomy group, the videothoracoscopy group was associated with more parenchymal resections (62.4 vs 80%, $P = 0.01$), fewer mechanical pleurodesis procedures (58 vs 77.5%, $P < 10^{-3}$), fewer postoperative respiratory complications (12 vs 8.2%, $P = 0.01$), fewer cases of postoperative pleural bleeding (2.3 vs 1.4%, $P = 0.04$) and shorter hospital lengths of stay (16 vs 9 days, $P = 0.01$). The recurrence rate was 1.8% ($n = 18$) in the thoracotomy group versus 3.8% ($n = 254$) in the videothoracoscopy group ($P = 0.01$). The median time between surgery and recurrence was 3 months (range: 1–76 months).

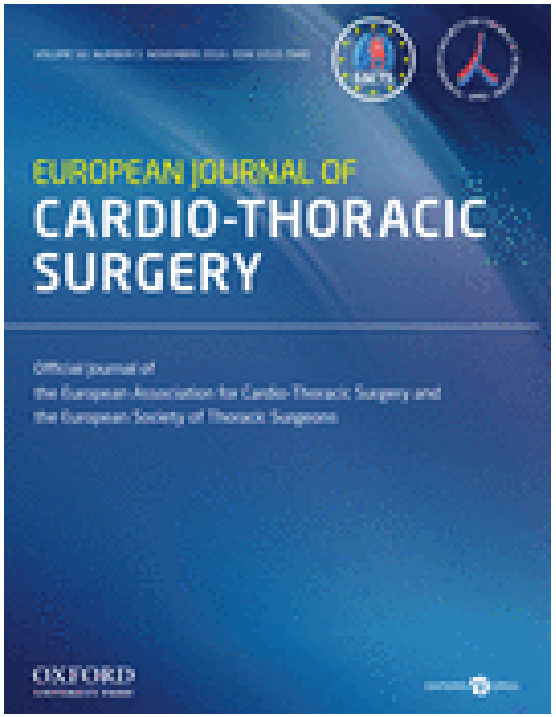
CONCLUSIONS: In the surgical management of spontaneous pneumothorax, videothoracoscopy is associated with a higher rate of recurrence than OT. This difference might be attributable to differences in the pleurodesis technique rather than differences in the parenchymal resection.

Keywords: Videothoracoscopy • Thoracotomy • Pneumothorax • Recurrence • Pleurodesis

INTRODUCTION

Spontaneous pneumothoraces (SPs) occur without preceding trauma or an obvious underlying precipitating cause and are subclassified into primary and secondary SP [1]. Primary SP affects patients without clinically apparent disorders, whereas secondary SP affects patients with underlying pulmonary disease. Primary SP is a common disease, with incidences of 18–28/100 000 in men and 1–6/100 000 in women [2, 3]. Guidelines from the American College of Chest Physicians and the British Thoracic Society indicate that surgical management should be reserved for ipsilateral or contralateral recurrence of primary SP or persistent air leaks after pleural drainage. Two surgical approaches are possible: open thoracotomy (OT) and video-assisted thoracoscopic surgery (VATS). The surgical technique then includes the resection of parenchymal bullae and pleurodesis [4, 5].

Both surgical approaches are feasible, but the guidelines do not specify which one should be used as the first choice. To date, no large randomized clinical trials (RCTs) have been conducted to



Primary Spontaneous Pneumothorax

METHODS Data for 7647 patients operated on for primary or secondary spontaneous pneumothorax between 1 January 2005 and 31 December 2012 were extracted from Epithor®, the French national database. The type of pleurodesis and parenchymal resection was collected. Outcomes were (i) bleeding, defined as postoperative pleural bleeding; (ii) pulmonary and pleural complications, defined as atelectasis, pneumonia, empyema, prolonged ventilation, acute respiratory distress syndrome and prolonged air leaks; (iii) in-hospital length of stay and (iv) recurrence, defined as chest drainage or surgery for a second pneumothorax.

RESULTS: Of note 6643 patients underwent videothoracoscopy and 1004 patients underwent OT. When compared with the thoracotomy group, the videothoracoscopy group was associated with more parenchymal resections (62.4 vs 80%, $P = 0.01$), fewer mechanical pleurodesis procedures (93 vs 77.5%, $P < 10^{-3}$), fewer postoperative respiratory complications (12 vs 8.2%, $P = 0.01$), fewer cases of postoperative pleural bleeding (2.3 vs 1.4%, $P = 0.04$) and shorter hospital lengths of stay (16 vs 9 days, $P = 0.01$). The recurrence rate was 1.8% ($n = 18$) in the thoracotomy group versus 3.8% ($n = 254$) in the videothoracoscopy group ($P = 0.01$). The median time between surgery and recurrence was 3 months (range: 1–76 months).

Surgical management of spontaneous pneumothorax: are there any prognostic factors influencing postoperative complications?
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Primary Spontaneous Pneumothorax

Table 1: Demographic data according to the surgical approach (study group, *n* = 7647)

	VATS group (*n* = 6643)	OT group (*n* = 1004)	*P*-values
Demographics			
Gender			
Male	5145 (77%)	781 (78%)	0.81
Female	1498 (23%)	223 (22%)	
Age (years)	32 ± 0	40 ± 1	0.01
Smoker	2399 (36%)	260 (26%)	0.01
ASA			
I	539 (54%)	4956 (75%)	0.01
II	1300 (20%)	310 (31%)	
III	387 (6%)	155 (15%)	
BMI (kg/m^2)	21 ± 0	22 ± 0	0.01
Respiratory past history			
Asthma	181 (3%)	12 (1%)	0.01
Chronic bronchitis	1070 (16%)	253 (25%)	0.01
Emphysema	842 (13%)	202 (20%)	0.01
Chronic respiratory failure	94 (1%)	49 (5%)	0.01
Cardiovascular past history			
Coronary artery disease	41 (1%)	23 (2%)	0.01
Cardiac arrhythmia	46 (1%)	11 (1%)	0.17
Congestive heart failure	46 (1%)	8 (1%)	0.7
Peripheral artery disease	48 (1%)	21 (2%)	0.01
Stroke	18 (0%)	5 (0%)	0.22
Other past medical history			
Diabetes	45 (1%)	11 (0%)	0.14
Steroid therapy	11 (0%)	2 (0%)	0.8
Neurological disease	32 (0%)	5 (0%)	0.9
Psychiatric disease	260 (4%)	30 (3%)	0.15
Malignant past history			
Malignant disease	153 (2%)	38 (4%)	0.01
History of chemotherapy	66 (1%)	21 (2%)	0.01
History of radiotherapy	40 (1%)	13 (1%)	0.01

ASA: anesthesiologist; BMI: body mass index.

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