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Research Article

Clinical, Neurophysiological and Radiological Descriptive Analysis: A Case Series of Diaphragmatic Palsy

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Abstract

Background: When diaphragmatic palsy is suspected due to its symptoms, the implementation of neurophysiological studies, imaging tests, and pulmonary function tests are essential for confirming the diagnosis. We present a series of 38 cases between 2009 and 2022 of unilateral or bilateral diaphragmatic palsy secondary to iatrogenic or idiopathic neuromuscular disease or other diseases.

Method: In all cases, pulmonary function tests, imaging tests, and neurophysiological studies (motor nerve conduction studies of the phrenic nerve and electromyography of the right and left hemidiaphragms, without ultrasound guidance) were performed to confirm the diagnosis.

Results: Twenty-two cases showed a restrictive pattern in the pulmonary function tests (57%). The chest radiographs showed an elevated hemidiaphragm in 27 patients (71%). The neurophysiological studies confirmed phrenic nerve injury and/or muscle dysfunction of the hemidiaphragm in 33 of 38 (86%).

Conclusions: Diaphragmatic palsy is an uncommon reason for consultation, and imaging and pulmonary function tests have limitations in assessing disease severity. Therefore, neurophysiological phrenic nerve conduction studies and electromyography of the hemidiaphragms provide diagnostic and progression information on the disease.

Keywords: Diaphragmatic palsy; Phrenic nerve; Electromyography; Hemidiaphragm

Introduction

Diaphragmatic palsy secondary to phrenic nerve injury is a known cause of dyspnea and occasionally of respiratory failure [1-6]. In the health area served by University Hospital San Jorge (Huesca), the prevalence of this condition is 2 per 100,000 inhabitants/year (38 cases in 13 years in a population of 125,000 inhabitants).

The most common etiology for isolated diaphragmatic palsy is idiopathic, followed by underlying progressive neuromuscular disease [7-8]. However, phrenic nerve injury can be due to trauma, infections such as syphilis, neck surgery, chest surgery, cannulation of the internal jugular vein, hypothermia in the case of heart surgery due to the use of ice or cold solutions for pericardial lavage, and compression and/or infiltration by mediastinal or pulmonary tumors [9-26].

Material and Methods

We present a case series of diaphragmatic palsy in our center between 2009 and 2022, in which we retrospectively analyzed the etiology, outcome, radiological and neurophysiological findings, and pulmonary function tests. We excluded cases secondary to trauma due to accidents or multiple trauma and pulmonary neoplasia, although we included those iatrogenic cases due to surgical interventions that initially did not affect the diaphragm or its innervation.

Our cohort comprised 38 cases of unilateral or bilateral diaphragmatic palsy (7 patients), among 23 (60%) men and 15 (40%) women between 41 years and 83 years of age. The most common etiology was idiopathic (31%), followed by surgery (26%) and neuromuscular diseases (21%), which included demyelinating neuropathies, Parsonage-Turner syndrome, amyotrophic lateral sclerosis, myotubular myopathy, myopathy of unknown origin, and botulism. We also observed cases produced by various types of surgeries (valve replacement, pericardiectomy, radical prostatectomy, tonsillectomy), pulmonary embolization, cryoablation for atrial fibrillation, cervical injury (level C3-C4), compression by a fibrous tumor of the pleura, respiratory infections, pulmonary hypertension, and hernia with diaphragmatic eventration (Table 1) [27].

In our series, the most common initial clinical manifestation was dyspnea (81%), followed by postoperative radiological findings (5%) and abnormal laboratory test results (2.6%). During the consultation, 6 patients presented criteria of respiratory failure, 4 of them acute and 2 of them chronic (Table 2).

In terms of clinical progression, most cases had a slight improvement (57.8%), or remained stable (13.1%). In the other cases, the patients progressed to exit us (10%), or complications

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were observed due to multiple diseases or by mechanical ventilation (13.1%) (Table 3).

Table 1: Diaphragmatic palsy etiology.

Etiology	Number of patients
Idiopathic	12
Surgery	10
Neuromuscular disease	8
Infections	3
Others:	5
Total	38

Surgery: Cardiac valve replacement, radical prostatectomy, tonsillectomy, pericardiectomy, medullary tumor excision, cryoablation for atrial fibrillation

Neuromuscular disease: Demyelinating neuropathy, Motor Neuron Disease, botulism, Parsonage-Turner syndrome, myotubular myopathy, unknown myopathy, C3-C4 damage

Infections: respiratory infection, Influenza A infection

Others: pulmonary embolism, fibrous pleural tumor, unknown, pulmonary hypertension, diaphragmatic hernia with eventration

Table 2: Initial clinical manifestations of diaphragmatic palsy.

Clinical manifestations	Number of patients
Dyspnea	31
Asthenia	2
Radiologic finding	2
Coma	1
Lower limbs weakness	1
Alterations in blood test after surgery (hypoxemia and hypercapnia)	1
Total	38

Table 3: Clinical evolution of diaphragmatic palsy.

Clinical evolution	Number of patients
Improvement	22
Stable	5
Progression of respiratory insufficiency	2
NIMV (Bipap)/pluripathology complications*	5
Exitus	4
Total	38

NIMV: Non-Invasive Mechanical Ventilation

*Cardiac insufficiency, myeloproliferative syndrome

In all cases, diaphragmatic involvement was suspected after performing imaging tests (chest radiography, chest ultrasound, and/or computed tomography) and/or pulmonary function tests. Neurophysiological studies were requested, with nerve conduction studies of both phrenic nerves and electromyographic studies of both hemidiaphragms, to confirm the diagnosis.

In the pulmonary function tests, spirometry was performed with the Medisoft® BodyBox 5500 system with the patients at rest, obtaining the following parameters: Forced Vital Capacity (FVC), maximum expiratory volume in the first second (FEV1%), FEV1%/FVC (percentage of the Forced Vital Capacity that is expired in the first second of the maneuver), FEV1%/FVC (percentage of the slow vital capacity that is expired in the first second of the maneuver), Peak Expiratory Flow (PEF), PEF 25%-75% (mean expiratory flow, between 25% and 75% of the FVC), FEF50/FIF50 (coefficient of the Forced Expiratory Flow and Forced Inspiratory Flow at 50%) and total forced expiratory time (FET 100%). The diaphragm contributes to a high percentage of respiratory muscle strength, and therefore pulmonary function tests can detect a restrictive syndrome; however, there are often limitations in interpreting the test results due to inadequate patient collaboration or other concomitant pleuropulmonary or chest diseases (chronic obstructive pulmonary disease, asthma, productive pleurisy, kyphoscoliosis, etc.). Patients do not always tolerate the implementation of the test in sitting or supine decubitus positions, which allows spirometry to be performed to meet the quality criteria. The FVC in supine decubitus is usually reduced by more than 30% in unilateral diaphragmatic palsy and by up to 75% in bilateral diaphragmatic palsy [27-33]. When implementing pulmonary function tests, the recommendation is to follow the quality criteria of the European Respiratory Society/American Thoracic Society and those of the Spanish Society of Pulmonology and Thoracic Surgery (theoretical values for the Spanish population) [34,35].

The neurophysiological study was performed with a Natus® 8-channel electromyography system and was interpreted by 2 neurophysiologists (PZMJ, BAI) to avoid inter-examiner error. The electroneurography study of the phrenic nerve was performed using electrical stimulation at the cervical level, on the posterior edge of the sternocleidomastoid muscle, with recording using surface electrodes, the active electrode on the sternum, 1 cm from the xiphoid apophysis, located on the left or right side depending on the side to explore, and the reference electrode positioned at 16 cm on the costal margin of each side [36,37]. The parameters studied for considering abnormal findings were mainly the reduced/absent CMAP (compound Muscle Action Potentials) responses (in axonal damage), with reduced area, and in some cases increased latency.

The electromyographic study of both hemidiaphragms was performed with monopolar needles in the diaphragmatic crura (Podnar technique), 3 cm below the areola, in an imaginary line between the midclavicular line and the anterior axillary line, in the intercostal space, placing an adhesive reference electrode approximately 5 cm laterally [38-44]. Prior to this, patients had to sign an informed consent document that indicated the risks of the procedure. The patients' blood pressure and heart rate were monitored just before, immediately after, and at 20 minutes after the electromyography study of the hemidiaphragms. The podnar technique was performed without ultrasound support and consisted of the diaphragmatic crura approach (a safe technique, observing distal lung margins of the puncture area while guided by ultrasound) [38,41], with no complications in any of the patients. The abnormal parameter results considered abnormal were the presence of acute/chronic denervation signs (presence of fibrillation or positive waves for acute denervation and high amplitude, long duration potentials with an increase in the number of phases for chronic denervation) and a reduced recruitment of motor units.

Results

Table 4 shows the results of the imaging studies and pulmonary function tests, which revealed the elevation of one (24 of 38, 63%) or both hemidiaphragms (3 of 38, 7%), and the parameters recorded in the pulmonary function tests, which showed a restrictive pattern of variable intensity in 57% of cases.

Figure 1 shows the chest radiography of one of the patients, which showed an elevated right hemidiaphragm.

In the case of the neurophysiological studies, most cases showed a phrenic nerve injury [unilateral (25 of 38) or bilateral (8 of 38, normal in the remaining 5)], or electromyography studies of the pathological hemidiaphragm (29 of 38; electromyography was not performed in 4 due to oral anticoagulant therapy) (Table 5, Figure 2-6). Eight (21%) of the patients underwent check-up studies, with improvement in 6 of them, and with no favorable progression in the neurophysiological parameters in 2 of them.

Table 4: Chest Rx findings/Respiratory function tests

	Age	Gender	Chest Rx	FVC (L)		FEV1(L)		FEV/FVC (%)		MIP (cm H2O)		MEP (cm H2O)		Spirometry pattern
				Pre	%	Pre	%	Pre	%	Pre	%	Pre	%	
1	68	M	L hemidiaphragm elevation											
2	83	F	L hemidiaphragm elevation	0.57	30	0.51	43		90					Restrictive pattern
3	76	F	R hemidiaphragm elevation	1.91	67	1.48	76		78	17	22	92	36	Restrictive pattern
4	56	M	L hemidiaphragm elevation	2.58	60	1.91	59		74					Restrictive pattern
5	61	M	R hemidiaphragm elevation	3.1	65	2.72	78		73	79	104	126	103	Restrictive pattern
6	69	F	R hemidiaphragm elevation	1.73	63	1.51	78		87					Restrictive pattern
7	59	M	L hemidiaphragm elevation	5.39	60	3.47	81	64.2	87	114.3	90	88.5	49	N*
8	81	F	L hemidiaphragm elevation	1.28	60	0.94	67		66					Mixed pattern
9	62	M	N	4.18	78	3.19	82		73					Restrictive pattern
10	67	M	R hemidiaphragm elevation	2.63	44	1.9	33							FVC reduced in decubitus
11	41	M	L hemidiaphragm elevation	4.09	87	2.67	69		65					Mild obstructive pattern
12	56	F	R hemidiaphragm elevation	2.3	69	2.5	79		85					Mild restrictive pattern
13	61	M	Pulmonary infiltrate	2.5	46	2.27	57		91					Severe restrictive pattern
14	68	M	Both hemidiaprhram elevation	2.05	46	1.69	53		82	73	101	154	130	Moderate restrictive pattern
15	77	M	N	2.9	79	2.15	85		74					N
16	59	M	Both hemidiaprhram elevation	1.59	35	1.26	38		80					Severe restrictive pattern
17	65	M	R hemidiaphragm elevation	3.8	77	2.8	78		72					Mild restrictive pattern
18	73	F	R hemidiaphragm elevation	1.1	41	0.83	44		75	27	37	89	121	Moderate restrictive pattern
19	41	F	Fibrocicatricial tracts	1.6	42	1.13	38		71					Restrictive pattern
20	58	M	L hemidiaphragm elevation	1.79	45	1.55	53		86	58	71	136	107	Severe restrictive pattern
21	46	F	R hemidiaphragm elevation	3	80	2.23	77	74	97					N
22	74	F	Both hemidiaprhram elevation	1.22	44	0.96	51		79	33	44	40	45	Moderate restrictive pattern
23	80	M	N											
24	51	F	N	2.6	86	1.92	83		81					FVC reduced in decubitus
25	76	F	L hemidiaphragm elevation											
26	78	M	N											Severe obstructive pattern
27	68	M	N											
28	49	M	N	2330	64	3140	67		74	128	102	113	57	Restrictive pattern
29	62	F	L hemidiaphragm elevation	1.94	68	1.56	73	80.7	109					Moderate restrictive pattern
30	39	M	L hemidiaphragm elevation											
31	61	M	L hemidiaphragm elevation	2740	53	2150	55		78					Moderate restrictive pattern
32	71	F	N	2.15	88	1.54	80	71.6	91	27.71	35	43.8	37	N
33	56	F	N	1.37	40	0.97	37	71.1	91	15.99	18	30.8	28	Moderate restrictive pattern
34	57	M	L hemidiaphragm elevation	2.95	59	2.29	60	77.8	104	77.59	125.5	75.5	187.4	Restrictive pattern
35	64	M	L hemidiaphragm elevation											
36	53	M	L hemidiaphragm elevation	3.94	69	3.06	70		103					Mild restrictive pattern
37	72	M	R hemidiaphragm elevation	2.33	60	1.27	43	54.6	71					Mild restrictive pattern
38	79	F	R hemidiaphragm elevation	7.77	68	1.13	57	64.2	84					Mild restrictive pattern

FVC: Forced Vital Capacity (L: measured in Liter); FEV1: Forced Espiratory Volume in the 1st second (L: measured in Liter); FEV/FVC: Forced Expiratory Volume/Forced Vital Capacity; MIP: Maximal inspiratory pressure (measured in H2O centimeters); MEP: Maximal expiratory pressure (cmH2O, measured in H2O centimeters); M: Male; F: Female, l: left, R: right, N: Non significative radiologic/spirometric alterations, *Control spirometry 3 years after

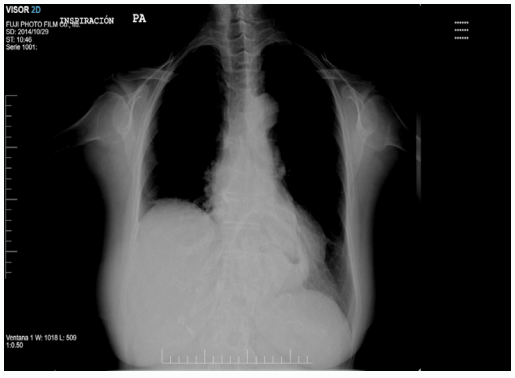

Figure 1: Elevation of right hemidiaphragm in chest Rx.

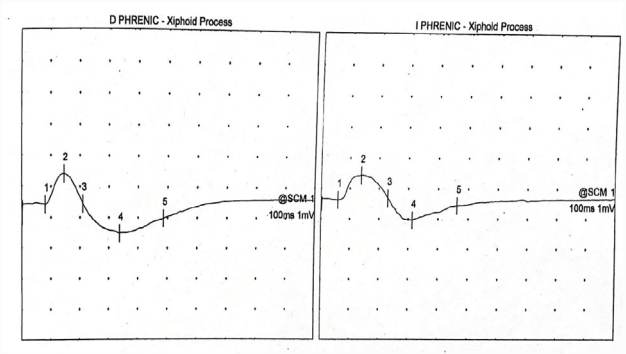

Figure 2: Normal bilateral phrenic nerve conduction study.

Discussion

Although diaphragmatic palsy is a frequent disease after multiple trauma and mediastinal or bronchopulmonary neoplasia, it is uncommon when the etiology is idiopathic or at the start of neuromuscular diseases. On numerous occasions, the initial suspicion is due to elevation of a hemidiaphragm in a chest radiograph performed during the study of recent-onset or increased dyspnea. In other cases, however, the radiological findings are not so obvious, and it is the presence of a restrictive syndrome not explained by any other cause that warns us when faced with this suspected diagnosis, especially if it is accompanied by reductions in maximal inspiratory (PImax) and

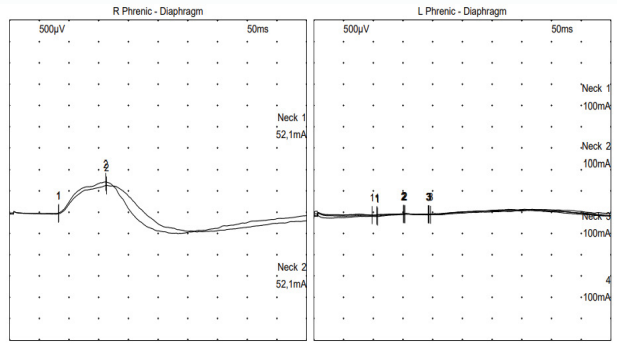

Figure 3: Unilateral phrenic nerve neuropathy (left phrenic nerve). Very low amplitude responses in left side, with normal responses in the right one.

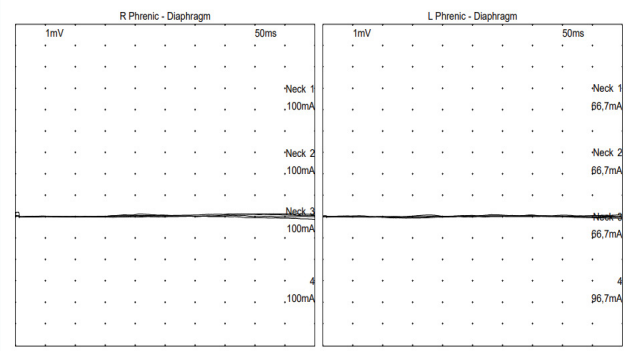

Figure 4: Bilateral phrenic nerve neuropathy. There is no response in both sides.

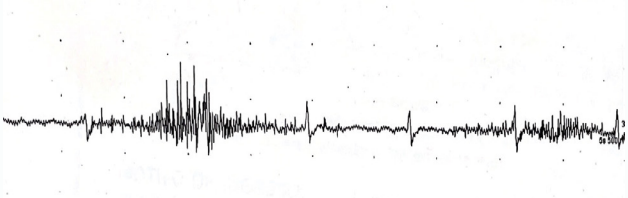

Figure 5: Normal EMG pattern in left hemidiaphragm. Sweep speed: 5s, Sensitivity: 500µV.

Figure 6: Pathological EMG pattern in left hemidiaphragm, with discrete recruitment pattern showing loss of motor units. Sweep speed: 5s, Sensitivity: 500µV.

expiratory (PEmax) pressures, although its reproducibility is less than that of measuring FEV1% or FVC using spirometry. In our series, spirometry that met the quality criteria could not be performed in 7 of the 38 patients due to a lack of collaboration or the presence of dyspnea, which hindered an appropriate maneuver; in 22 of the 38 patients, PImax/PEmax readings could not be obtained for the same reason. We can therefore see the importance of neurophysiological studies for confirming the diagnosis, although not all hospital centers have all the techniques for respiratory function studies and neurophysiological studies of the phrenic nerve/diaphragmatic muscles or ultrasound.

For neurophysiological studies, some centers perform the electromyography study by ultrasound-guided direct puncture at the central level of the chest wall [39]. In our center, the electromyography studies are performed with a diaphragmatic crura approach, using the Podnar technique, with the need for imaging support (ultrasound; our center does not have neuromuscular ultrasound) [38].

In our experience, we have not had any case of iatrogenic pneumothorax or any other complication as a result of the electromyography study.

Performing pulmonary function tests and diagnostic techniques that confirm the clinical findings are essential for managing and following-up these patients.

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Table 5: Neurophysiological studies (Phrenic nerve conduction studies and EMG of both hemidiaphragms).

	Age	Gender	EMG	Lat RP	Lat LP	Amp RP	Amp LP	Area RP	Area LP	EMG RHD	EMG LHD	Cont EMG	Impr cont EMG
1	68	M	PU	4.3	NR	1.4	NR	7.5	NR	N	↓MU	No	
2	83	F	PU	7.05	7.45	1.2	0.01	6.7	0.2	N	↓MU Sub den	No	
3	76	F	PU	9.4	5.85	0.3	1.2	2.6	1.5	Cron den ↓MU	N	Yes	No
4	56	M	PU	6.35	NR	1.1	NR	5.8	NR	N	↓MU	No	
5	61	M	PU	8.75	8.35	0.5	1.1	4.6	6.8	Cron den ↓MU	N	Yes	Yes
6	69	F	N	7.15	6.75	0.5	0.4	4.8	3.2	N	N	No	
7	59	M	PU	7.7	NR	0.9	NR	4.3	NR	N	↓MU	No	
8	81	F	PU	7.35	17.35	0.6	0.1	3.5	1.1	No EMG AC	No EMG AC	No	
9	62	M	PU	7.1	6.95	0.3	0.1	2.8	0.9	N	Cron den	Yes	Yes
10	67	M	PU	18	6.95	0.1	2.1	0.9	6.6	Cron den ↓MU	N	Yes	Yes
11	41	M	PU	7.7	14.2	1.1	0.8	8.8	8.1	N	Cron den	No	
12	56	F	PU	6.25	5.7	0.2	0.4	3.2	5.5	No EMG AC	No EMG AC	Yes	
13	61	M	PU	8	5.9	1.9	1.5	7.4	8.1	↓MU	N	No	
14	68	M	PB	7.8	7.15	0.9	0.8	4.7	4.9	Cron den	Cron den	No	
15	77	M	N	7.7	8.15	1.5	1.4	0.9	1.5	N	N	No	
16	59	M	PB	12.6	13.15	0.1	0.1	0.3	0.9	↓MU	↓MU	Yes	No
17	65	M	PU	9.45	6.3	0.1	1.1	0.5	7.4	↓MU	N	No	
18	73	F	PU	NR	7.8	NR	0.2	NR	2.1		N	No	
19	41	F	N	5.35	5.05	0.9	0.7	6.1	5.5	N	N	No	
20	58	M	PU	8.5	14.1	0.4	0.1	3.4	0.5	No EMG AC	No EMG AC	No	
21	46	F	N	5.7	5.75	0.6	0.9	5.7	7.7	N	N	No	
22	74	F	PB	7.35	6.95	0.01	0.01	0.1	0.5	↓MU	↓MU	No	
23	80	M	PU	10.05	NR	0.7	NR	3.1	NR	No EMG	No EMG	No	
24	51	F	PB	NR	NR	NR	NR	NR	NR	N	N	No	
25	76	F	PU	7.2	7.2	0..40	0.2	2.2	0.8	N	↓MU	No	
26	78	M	N	8.8	8.8	2.1	2.1			N	N	No	
27	68	M	PB	NR	NR	NR	NR	NR	NR	No FT ↓MU	No PF ↓UM	Yes	Yes
28	49	M	PB	2.5	3.02	0.1	0.01	0.2	0.1	↓MU	↓MU	Yes	Yes
29	62	F	PU	7.5	9.38	0.5	0.1	5	0.3	N	↓MU	No	
30	39	M	PU	3.6	7.7	0.1	0.01	0.5	0.1	N	↓MU	No	
31	61	M	PU	9.2	12.3	0.1	0.1	1.4	0.3	N	↓MU	No	
32	71	F	PU	6.15	8.28	0.7	0.2	4.9	1.3	Acute + cron den			
33	56	F	PB	2.95	3.45	0.4	0.6	5.7	10.7	↓MU	↓MU	No	
34	57	M	PU	8.28	10.63	0.7	0.1		0.1	No EMG AC	No EMG AC	No	
35	64	M	PB	NR	NR	NR	NR	NR	NR	↓MU	↓MU	Yes	Yes
36	53	M	PU	8.07	14.95	0.4	0.1	4.7	1.4	↓MU	N	No	
37	72	M	PU	10.1	5.28	0.4	0.7	3.6	4.8	↓MU	N	No	
38	79	M	PU	8.9	7.3	0.5	1	4.1	7.4	↓MU	N	No	

M: Male; F: Female; EMG: Electromyogram; Lat RP: Right Phrenic Nerve Latency; Lat LP: Left Phrenic Nerve Latency; Amp RP: Right Phrenic Nerve Amplitude; Amp LP: Left Phrenic Nerve Amplitude; Area RP: Right Phrenic Nerve Area; Area LP: Left Phrenic Nerve Area; EMG RHD: Right Hemidiaphragm EMG; EMG LHD: Right Hemidiaphragm EMG; Cont EMG: Control EMG Study; Impr cont EMG: Improvement in Control EMG Study; PU: Pathological Unilateral; N: Normal; PB: Pathological Bilateral; NR: Non Responses; ↓MU: Reduction in Motor Units; No FT: No Functional Test; Cron den: Chronic Denervation; Acute den: Acute Denervation; Sub den: Subacute Denervation. No EMG AC: no EMG study because of treatment with anticoagulant drugs

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