Review Article

Ortner (Cardio-vocal) Syndrome: A Collective Review

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ABSTRACT

To understand the concept of Ortner's (cardio-vocal) syndrome (OCVS), it is necessary to present the up-to-date clinical features and potential management strategies of cardiovascular hoarseness. The medical literature on OCVS published between 1980 and 2011 was comprehensively retrieved and analyzed. The patients who had cardiovascular hoarseness included a total of 256 patients. Hoarseness was the only symptom in 91 (35.27%) patients. The secondary symptoms of patients varied greatly, with dyspnea and dysphagia being the most common manifestations. OCVS is uncommon. Aortic aneurysms of various etiologies are the most common risk factors leading to cardiovascular hoarseness. When a patient presents with hoarseness, one should never overlook the cardiovascular causes, so that a misdiagnosis can be avoided and an immediate therapy is started.

KEYWORDS: aortic disease, congenital, heart defects, hoarseness, mitral valve stenosis

INTRODUCTION

Numerous conditions ranging from the common cold to systemic disorders involving the larynx can cause hoarseness[1,2]. The cause may be neoplastic (32%), surgical (30%), idiopathic (16%), traumatic (11%), central (8%) or infectious (3%)[3]. In recurrent laryngeal nerve paralysis, the left recurrent laryngeal nerve (LRLN) was more commonly involved than the right (70% Vs 30%)[4,5]. The diagnosis is usually based on the patient's medical history, physical examination, and computed tomographic (CT) scan or magnetic resonance imaging (MRI) results[5].

Hoarseness due to LRLN palsy caused by cardiovascular disease is termed as OCVS as described by Ortner in 1897[5,6]. Subsequently, this condition was reported to be associated with series of cardiovascular disorders including congenital heart defects, aortic disease, mitral valve insufficiency, and pulmonary hypertension. In the past decades, more and more patients with hoarseness of voice caused by left vocal cord palsy resulting from cardiovascular disorders have been reported. To understand the concept of this peculiar entity, it is necessary to present an up-to-date review of clinical features and potential management strategies of cardiovascular hoarseness.

MATERIALS AND METHODS

Retrieval policies

The medical literature on OCVS published between 1980 and 2011 was comprehensively retrieved in the MEDLINE database and the Google and Highwire Press search engines. The secondary references cited in the articles obtained from these sources were screened. Articles published in Mandarin in mainland China journals were excluded from this study. An article published in the European Journal of Medicine, 1992, was considered to be a repetitive publication in terms of majority of their case series to an alternative article published by the same first author, and hence to be removed. However, the articles written in Mandarin published in the medical journals in Taiwan were included. The search ended on December 21, 2011.

Statistics

Data were expressed in mean ± standard deviation and student 't' test was used to evaluate intergroup differences. A p-value of < 0.05 was considered to be statistically significant.

RESULTS

By comprehensive literature collection, a total of 256 patients had cardiovascular hoarseness including

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245 cases from 172 reports[5-176] and 11 cases from the cited references of the report of Myojin *et al*[58].

Out of the patients whose gender was recorded, there were 101 male and 55 female patients with a male-to-female ratio of 1.84:1. Their mean age was 53.90 ± 22.76 years (range, from 6 days to 90 years) (n = 196). No age difference was found between male and female patients (55.59 ± 22.09 years, Vs 50.93 ± 23.76 years, p = 0.1686). Two hundred and eight patients had their age range recorded. There were 17 minors, children and infants (8.17%) ≤ 17 years old (age range, 6 days ~ 17 years; mean age, 5.21 ± 7.15 years; median age, 0.58 years) and the remaining 189 (91.83%) patients were ≥ 18 years old (age range, 18 ~ 90 years, mean age, 58.24 ± 18.11 years; median age, 62 years) . The patients' age distribution is shown in Fig. 1.

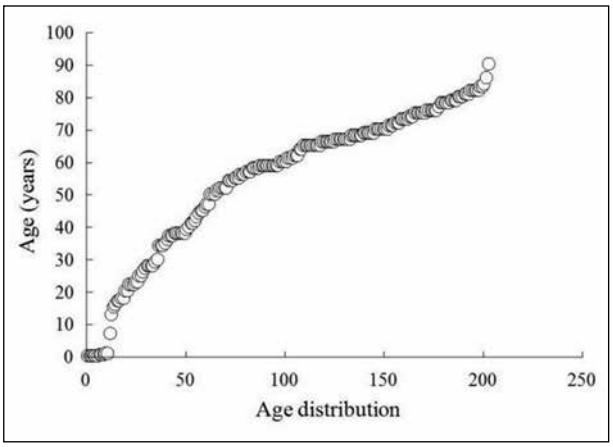

Fig. 1: Age distribution of patients with cardiovascular hoarseness

All patients in this patient setting presented with hoarseness. The nature of hoarseness was not described in majority of the reports, while in a few, it was described as gradual[40] or progressive[55]. Hoarseness was the only symptom in 91 (35.27%) patients. Besides, the secondary symptoms of the patients varied greatly, with dyspnea and dysphagia being the most common manifestations (Table 1). In addition, 61 patients had a past medical history (Table 2). The duration of hoarseness was 10.09 ± 19.66 (range, -2 ~ 120) months (n = 120) (minus means the onset of hoarseness developed after admission), with no difference between males and females (8.60 ± 17.57 months Vs 13.84 ± 23.83 months, p = 0.1846), or between the minors and adults (3.59 ± 3.61 months Vs 10.82 ± 20.47 months, p = 0.2691). No significant relationship was found between the patients' age and the duration of hoarseness ($Y = 0.002X + 10.101$, $R^2 = 5\times10^{-6}$) (Fig. 2).

Left vocal cord paralysis was verified by laryngoscopy in 140 (54.26%) patients and bilateral vocal cord paralysis in one (0.39%) patient, of which the resting positions of the vocal cord were recorded

Table 1: Major symptoms except for hoarseness in 81 patients

Major symptoms	n (%)
Dyspnea with / without cough	26 (32.10)
Dysphagia	11 (13.58)
Dyspnea, palpitation	3 (3.70)
Hemoptysis / hemosputum	5 (6.17)
Chest pain	6 (7.41)
Chest pain, hemoptysis	4 (4.94)
Cough	1 (1.23)
Dyspnea, dysphagia	2 (2.47)
Inspiratory stridor	2 (2.47)
Arthralgia	1 (1.23)
Central cyanosis	2 (2.47)
Fever/malaise/anorexia	2 (2.47)
Headache, blurred vision	1 (1.23)
Hemoptysis, dyspnea, chest pain	1 (1.23)
Neck swelling	3 (3.70)
Palpitation	1 (1.23)
Chest pain, inspiratory stridor	1 (1.23)
Syncope	1 (1.23)
Tachypnea	1 (1.23)
Wheeze	2 (2.47)
Weight loss	1 (1.23)
Neck pain	1 (1.23)
Seizure	1 (1.23)
Headache	1 (1.23)
Pain and burning sensation of the right great toe	1 (1.23)

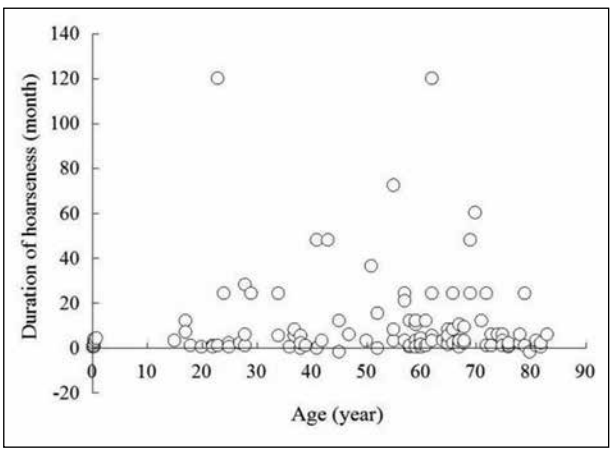

Fig. 2: Correlation between patients' age and duration of hoarseness

in 32 patients: the vocal cords were resting in the paramedian position in 27 (84.38%) patients and in the median position in 5 (15.63%) patients. Vocal cord position and movement under laryngoscopy in 141 patients with OCVS are shown in Table 3.

In the early days, angiogram, surgical exploration or autopsy was the diagnostic method for OCVS. Nowadays, non-invasive modalities including echocardiography, CT scan and MRI have mostly substituted the invasive catheterization as the diagnostic means. However, angiogram is still an adjunct on some occasions (Table 4).

Table 2: Past history of 61 patients with Ortner (cardiovocal) syndrome

Past history	n (%)
Acute lymphoblastic leukemia, ankylosing spondylitis, hepatitis B, cocaine abuse	1 (1.64)
Atrial fibrillation, stroke	1 (1.64)
Behçet's disease	1 (1.64)
Blunt chest trauma (traffic accident, falling from height)	13 (21.3)
Chronic atrial fibrillation	2 (3.28)
Chronic obstructive pulmonary disease	2 (3.28)
Chronic obstructive pulmonary disease, s/p coronary artery bypass grafting	1 (1.64)
Chronic renal failure	1 (1.64)
Coronary artery disease	5 (8.20)
Coronary artery disease/myocardial infarction, hypertension	2 (3.28)
Diabetes, pneumonia, upper lung abscess	1 (1.64)
Human immunodeficiency virus and hepatitis C virus infections, hemophilia	1 (1.64)
Hypertension	6 (9.84)
Hypertension, chronic obstructive pulmonary disease, chronic renal failure	1 (1.64)
Hypertension, coronary artery disease, s/p percutaneous transluminal coronary angioplasty	1 (1.64)
Hypertension, gouty arthritis	1 (1.64)
Hypertension, hypertensive renal disease	1 (1.64)
Lung cancer invasion	1 (1.64)
Myocardial infarction, s/p percutaneous coronary interventions	1 (1.64)
Osteomyelitis of the right tibia	1 (1.64)
Raynaud's phenomenon, menorrhagia, systemic lupus erythematosus	1 (1.64)
Retrotracheal aorta with aberrant left carotid artery	1 (1.64)
Rheumatoid arthritis, peripheral vasculitis	1 (1.64)
Adrenalectomy, unrepaired aortic abdominal aneurysm (s/p)	1 (1.64)
Coronary artery bypass grafting (s/p)	1 (1.64)
Heart transplantation (s/p)	1 (1.64)
Hemicolectomy for colon cancer, fistula in the esophageal wall (s/p)	1 (1.64)
Mitral valve replacement (s/p)	1 (1.64)
Patent ductus arteriosus ligation (s/p)	1 (1.64)
Repair of coactation of the aorta 34 years earlier (s/p)	1 (1.64)
Schistosomiasis mansoni	1 (1.64)
Schistosomiasis, urinary and intestinal	1 (1.64)
Superior vena cava syndrome	1 (1.64)
Tuberculosis, pulmonary	3 (4.92)
Tuberculosis, military	1 (1.64)

s/p: status post

Table 3: Vocal cord situation and movement under laryngoscopy of 141 patients with Ortner (cardiovocal) syndrome

Situation and movement	n (%)
Situation	127 (100)
Completely paralyzed	7 (5.51)
Paralyzed	80 (56.74)
Palsy	33 (25.98)
Subtle palsy	1 (0.79)
Paresis	2 (1.57)
Partially paralyzed	2 (1.57)
Closure	1 (0.79)
Deviation	1 (0.79)
Movement	14 (100)
Immobile/fixation	9 (64.29)
Slightly movable	2 (14.29)
Reduced mobility	1 (7.14)
Negligible movement	1 (7.14)
Standstill	1 (7.14)

Table 4: Diagnostic methods in 174 patients

Diagnostic method	n (%)
CT	39 (22.41)
Echocardiography	31 (17.82)
Angiogram	31 (17.82)
CT + angiogram	27 (15.52)
CT + echocardiography	9 (5.17)
CT + MRI	8 (4.60)
Echocardiography + angiogram	5 (2.87)
MRI	3 (1.72)
CT + echocardiography + angiogram	3 (1.72)
CT + MRI + angiogram	2 (1.15)
CT + MRA	2 (1.15)
Surgical exploration	4 (2.30)
Autopsy	2 (1.15)
Barium swallow examination	1 (0.57)
CT + CTA	1 (0.57)
CT + CT-guided needle biopsy	1 (0.57)
CT + MRI + MRA	1 (0.57)
CTA	1 (0.57)
Echocardiography + MRI	1 (0.57)
Mediastinoscopy	1 (0.57)
Upper gastrointestinal endoscopy	1 (0.57)

CT = computed tomography, CTA = computed tomographic angiogram, MRA = magnetic resonance angiogram, MRI = magnetic resonance imaging

Fourteen patients had their C-reactive protein (CRP) measured. It was reported that one patient had a normal CRP value without giving any quantitative result. The remaining 13 patients had a CRP of 10.20 ± 11.02 (range, 0.1 - 30.63, median 5) mg/dl.

Out of the young patients ≤ 17 years of age, in infants, congenital heart disease prevailed as an underlying cause of hoarseness including atrial or ventricular septal defect (ASD and VSD), patent ductus arteriosus (PDA), total anomalous pulmonary venous connection, double outlet right ventricle, left main coronary artery arising from the pulmonary artery, or idiopathic pulmonary artery hypertension. In older children, rheumatic mitral stenosis was the most common. Inclusive of a 16-year-old patient who had a traffic accident which caused aortic arch rupture, aortic disorders were the most common underlying causes of OCVS in 142 (55.47%) patients. Aortic aneurysms of various etiologies showed overwhelming predilections leading to left vocal cord palsy and hoarseness in 132 (51.56%) patients. Moreover, left atrial lesions presented in 20 (7.81%), and congenital heart defects presented in 14 (5.47%) patients, respectively. Ductal aneurysms, either patent or non-patent, accounted for up to 22 (8.59%) patients (Table 5).

Table 5: Primary disorders responsible for cardiovascular hoarseness

Primary disorders	n (%)
Congenital heart defects	14 (5.47)
Patent ductus arteriosus + pulmonary artery hypertension	3
Patent ductus arteriosus	1
Atrial septal defect	2
Ventricular septal defect + patent ductus arteriosus	1
Ventricular septal defect	1
Double outlet right ventricle	1
Ebstein's anomaly	1
Mitral atresia	1
Double outlet right ventricle with mitral atresia	1
Ensenmenger's syndrome, large ventricular septal defect	1
Total anomalous pulmonary venous connection	1
Left atrial disorders	20 (7.81)
Mitral stenosis	9
Mitral regurgitation + mitral stenosis	2
Mitral regurgitation + aortic regurgitation	1
Mitral stenosis with pulmonary hypertension	1
Mitral periprosthesis insufficiency with severe pulmonary hypertension	2
Mitral vlave prolapse	3
Left atrial myxoma	1
Chorda tendinae rupture	1
Aortic diseases	142 (55.47)
Aortic rupture (traumatic)	1
Penetrating aortic ulcer	2
Infectious aortitis	1
Giant cell artiritis of the aorta	1
Aortic fistulae	5
Esophago-broncho-aortic fistula	1
Aortopulmonary and/or aortoesophageal fistula	1
Aortobronchial fistula	1
Acute aorto-pulmonary artery fistula	1
Aortotracheal fistula	1
Aortic aneurysm	132
Marfan: Root (1)	1
Atherosclerotic : Aorta (8), arch (11), proximal descending aorta (1)	20
Degenerative : Aorta (4), arch (5), descending aorta (1), arch + descending aorta (1)	11
Embryologic: Arch (1)	1
Tuberculosis : Arch (2)	2
Syphilitic	2
Mycotic	10
Iatrogenic	3
Inflammatory: Arch + right subclavian artery + infrarenal abdominal aorta (1)	1
Not described: Aorta (16), arch (16) [one of them ruptured into the pulmonary artery], isthmus (2), descending aorta (11)	45
Traumatic : Aorta (8), arch (4), proximal descending aorta (6), arch + descending aorta (1)	19
Dissecting: Aorta (posttraumatic) (5), arch (6), descending aorta (5), ascending + arch + descending (1)	17
Supraaortic vessel disorders	16 (6.25)
Aberrant right subclavian artery aneurysm	1
Aneurysm of the common carotid artery (Takayasu's arteritis)	1
Aneurysm of the innominate artery	2
Aneurysm of the right subclavian artery	1
Aneurysm of the right subclavian artery + ascending aorta dilation	1
Anomalous left carotid artery arising from a retrotracheal arch	1
Left subclavian artery pseudoaneurysm	1
Pseudoaneurysm of the innominate artery (mycotic)	1
Pseudoaneurysm of the right subclavian artery	1
Pseudoaneurysm of the left common carotid artery	1
Right subclavian artery aneurysm (tuberculosis)	1
Right subclavian artery aneurysm	1
Right subclavian artery pseudoaneurysm	1
Ruptured innominate artery	1
Tortuosity of the three aortic branches	1
Other cardiovascular malformations	54 (21.09)
Aneurysm of diverticulm of the ductus arteriosus	7
Chronic cor pulmonale	2
Dilated cadiomyopathy	1

Continued...

Table 5 Continues ...

Primary disorders	n(%)
Ductal aneurysm	22
Left ventricular aneurysm	1
Pulmonary artery aneurysm	3
Pulmonary artery dilation	8
Pulmonary artery hypertension	8
Schistosomal pulmonary hypertension	1
Pericardial cyst	1
Extracardiac lesions	10 (3.91)
Massive hepatic hemangiomatosis (extracardiac vascular abnormality leading to dilation of the left atrium)	1
Cystic fibrosis of the lung	1
Tumor of the Botallo's lymph node invading the aortic arch	1
Mediastinal fibromatosis	1
Mediastinal bronchial artery aneurysm	1
Lung cancer	1
High altitude	3
Adenocarcinoma of the thymus	1

Out of the total patients, 16 (6.25%) patients died. The management of the underlying primary disorders responsible for the hoarseness in 90 patients was surgical in 41 (45.56%), interventional in 12 (13.33%), and follow-up / conservative treatment in 37 (41.11%). Two (2.22%) patients refused surgery, and two (2.22%) patients refused interventional treatment. Out of three patients who received treatment for the hoarseness, two had thyroplasty and one patient received injection and the voice recovered. The remaining patients did not. Forty patients had a follow-up of two weeks ~ 6 years (mean, 8.51 ± 12.13 months; median 5.5 months). The outcomes of hoarseness were recorded in 58 patients: resolved in 26 (44.83%), improved in 17 (29.31%), persisted in 13 (22.41%), and exacerbated in two (3.45%) patients, respectively.

DISCUSSION

Anatomy

The LRLN courses between the aorta and pulmonary artery. Cadaveric studies illustrated that the distance between the aorta and pulmonary artery within the aortic window is only 4 mm. Therefore, the primary mechanism of injury to the LRLN involves compression of the nerve between the left pulmonary artery and aorta[177]. Ari *et al*[178] found that the LRLN was compressed between an enlarged left pulmonary artery and aorta near the ligamentum arteriosum in patients with mitral stenosis. However, it may have anatomical variance in certain individuals. Odegard *et al*[179] observed during video-assisted throracoscopic surgery for PDA, LRLN had an unexpected location in 8 / 59 (13.56%) patients: in direct contact with the PDA on its superior surface in 5 / 59 (8.47%), on its lateral surface in 2 / 59 (3.39%) and beyond this region in 1 / 59 (1.69%) patients, respectively. In 29 / 59 (49.15%) patients, it was located inferiorly and medially to the ductus with close proximity to the position for placing a clip and thus vulnerable to injury.

Symptomatology

Hoarseness caused by cardiovascular pathology might be the only symptom for the patients to consult a clinician[134]. Alternative symptoms may include dyspnea, wheezing, or hemoptysis resulting from laryngeal nerve impingement, or chest pain caused by hemorrhage or compression of the intrathoracic structures[180]. The ductal aneurysm may compress the left main bronchus thereby explaining the wheeze[53]. Most fistulas are related to the thoracic aortic aneurysm, and massive hemoptysis has been reported in these patients[181]. Hoarseness can be the initial manifestation of a painless aortic pseudoaneurysm formed by an aortotracheal fistula[157]. When the patients with OCVS syndrome presents with hemoptysis, lethal aorto-pulmonary fistula should be highly suspected[157]. The movement and status of the vocal cord may have close correlation with the severity of the symptoms. Resting in a paramedian position for the LRLN may result in an absence of vocal fold opposition, weak phonation, stridor and feeding difficulties in infants due to poor swallowing coordination or aspiration through an incompetent larynx[182]. Vocal cord fixation may mean that the vocal cord becomes atrophic, hoarseness becomes exacerbated, or aspiration may be associated with vocal cord adduction; when the vocal cord becomes movable, symptoms such as hoarseness and aspiration could be alleviated[5].

Etiology

In OCVS, the LRLN was injured by compression or traction resulting from the cardiovascular anatomical changes[134]. Clinical observations revealed that the aortic aneurysms were located in the aortic arch, proximal part of descending aorta, or distal part of the arch. All of them had close proximity to the aortic arch / isthmus[5] and the LRLN was stretched over the aneurysm[34,143]. The shapes of the aortic aneurysms leading to OCVS varied, being saccular, fusiform, cystic

or dissecting[120]. Stoob *et al*[5] described that the shapes of the aneurysms did not correlate with the clinical outcomes. In addition, aortic aneurysm complicating aorto-bronchopulmonary and aorto-esophageal fistulae may also present with hoarseness[76].

Pseudoaneurysms are frequently post-traumatic, atherosclerotic, inflammatory (vasculitic) and infectious (mycotic) in origin[183]. Pseudoaneurysm formation in Behcet's disease was taken as obliteration of the vasa vasorum by an inflammatory process, thereby interrupting the nutrient flow to the aortic wall[59].

In pulmonary artery hypertension, compression of the nerve between the enlarged and upwardly displaced pulmonary artery and aorta was responsible for the LRLN palsy and the left vocal cord paralysis[72]. Apart from the primary pulmonary artery hypertension, several conditions may have secondary pulmonary artery hypertension, including schistosomiasis infection[54], high altitude[100], mitral periprosthesis insufficiency[56] and left main coronary artery arising from the left pulmonary artery[103], *etc*. Within the context of mitral stenosis[99,178] mitral valve prolapse with left atrial dilation and cardiomegaly[66], atrial myxoma[29], and cardiomegaly from high output failure caused by massive hepatic hemangiomatosis leading to left atrial dilation[20], the enlarged left atrium pushes the laryngeal nerve upwards compressing it against the aortic arch.

In extreme cases, cardiovascular hoarseness resulted either from traction by the collapsed lung, or by direct pressure from enlarged mediastinal lymph nodes in patients with cystic fibrosis and complete pulmonary collapse[184] and the tumor-eroding of the lung apex, which might compress the recurrent laryngeal nerve and cause hemoptysis[174].

TREATMENT

Early diagnosis of OCVS may be helpful in starting prompt treatment, restore the vocal cord function and avoid permanent damage[100]. Surgical repair is often indicated for symptomatic chronic post-traumatic pseudoaneurysm[136]. Endovascular stent grafting can provide definitive treatment for both the aortic pathology and LRLN palsy[153]. LRLN paralysis could be alleviated spontaneously in patients receiving surgical treatment of the primary cardiovascular disorder[5].

Summary

Clinical features of cardio-vocal hoarseness that are germane to the contemporary classification are described. Aortic aneurysm near the aortic arch is the major predisposing factor for this special entity, followed by left atrial dilation, and pulmonary artery hypertension. Some cardiovascular disorders secondary to extra-cardiac anomalies should be included in this syndrome. However, the so-called "iatrogenic" OCVS, which does not conform to one of the main elements of OCVS - "cardiovascular disease" as the underlying etiology of hoarseness, defined by Ortner in 1897. The "iatrogenic" OCVS reported in the literature was actually an 'immediate hoarseness' complication following cardiovascular operation, intervention or maneuver. On the contrary, a hoarse voice caused by late complications secondary to cardiovascular operations like aortic pseudoanerysms[86,158] should be termed as iatrogenic OCVS. Instead, the hoarse voice occurring following cardiovascular operation, intervention or maneuver should be defined as an "immediate hoarse complication", "iatrogenic LRLN palsy" or "iatrogenic left vocal cord paralysis". Therefore, the usually mentioned "iatrogenic left vocal cord paralysis" was a wrong concept and should be excluded from the domain of OCVS. The bias in this concept should be realized by all concerned.

CONCLUSIONS

OCVS is uncommon. Aortic aneurysms of various etiologies are the most common risk factors leading to cardiovascular hoarseness. The location of the aneurysms near the aortic arch correlated significantly with the development of cardio-vocal hoarseness. When the patient presents with hoarseness, one should never overlook the cardiovascular causes so that a misdiagnosis is avoided and an immediate therapy is started.

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