Neurological Disorders and Idiopathic Vocal Cord Palsies "Clinical, Radiological and Electrophysiological Correlates"

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ABSTRACT

Adult vocal cord palsy is a relatively common voice disorder, causes remain idiopathic. Some patients with idiopathic VCP may involve concomitant disseminated neuronal degenerative process. The aim of our study is to determine whether the patients with idiopathic VCP might have pre-existing neurological conditions and its plan of management. Twenty patients were included. From Al-Dar Hospital, Al-Madina Al-Munawwara through the period from June 2007 till June 2008 with age range 30-70 years old complaining of hoarseness of voice. Clinical history, examination, fibro-optic laryngoscope, M.R.I. brain "when indicated", E.M.G. and N.C. study when indicated, C.T scan chest, C.T scan neck, thyroid ultrasound, lab. indices were done for all patients. The study has shown that 20% of our patients with VCP were idiopathic. The majority of our patients have pre-existing neurological conditions 80%, 45% due to central lesions, the majority of them (30%) preceded by C.V. stroke either acute cerebral infarction or old stroke followed by multiple sclerosis (20%), the neurological conditions that preceded VCP seem to be associated with either the Brain-stem out flow end of neuraxis or the segmental. Laryngeal or esophageal anatomy, the remaining 35% were due to peripheral lesions (acute Guillain-Barré syndrome). The VCP in our case series was unilateral in 45% of cases, bilateral in 55% of patients. Finally, we therefore conclude that careful neurological examination and work-up should be considered for all patients with idiopathic VCP. (Egypt J. Neurol. Psychiat. Neurosurg., 2009, 46(1): 247-252)

INTRODUCTION

Adult vocal cord palsy is a relatively common voice disorder characterized by a malfunction of laryngeal muscle.

Causes remain idiopathic in approximately 12% of cases.¹,²

The vocal folds are concerned in the production of sound, and enclose two strong bands named the vocal ligaments. Each ligament consists of a band of yellow elastic tissue, attached in front to the angle of the thyroid cartilage, and behind to the vocal process of the arytenoids it is covered medially by mucous membrane, which is extremely thin and closely adherent to its surface.

The nerves are derived from the internal and external branches of the superior laryngeal nerve from the recurrent nerve, and from the sympathetic.

The internal laryngeal branch is almost entirely sensory.

Vocal fold paresis and paralysis result from abnormal nerve input to the laryngeal muscles.

Paralysis is the total interruption of nerve impulse resulting in no movement of the muscle; paresis is the partial interruption of nerve impulse resulting in weak or abnormal motion of laryngeal muscle(s).

Vocal fold paresis/paralysis can happen at any age – from birth to advanced age, in males and females alike, from variety of causes.

Vocal cord paralysis is the second most common congenital laryngeal abnormality.

The diagnosis can usually be made by flexible endoscopy at the bedside.

Once the diagnosis is made, the etiology of vocal cord paralysis must be determined.

The major causes of vocal cord paralysis in children include neurological conditions, birth
trauma, and idiopathic causes. The neurological conditions include CNS diseases e.g. cerebral dysgenesis, hypotonia, cerebral palsy, Charcot-marie tooth disease and Arnold–Chiari malformation with meningomyelocele.

Familial VCP exhibit a constellation of polyneuropathies and vocal fold motion impairment is one component of multiple system atrophy. This indicates that some patients with idiopathic VCP may involve concomitant disseminated neuronal degenerative process.

The aim of this study is to determine whether the patients with idiopathic VCP might have preexisting neurological conditions and its plan of management.

**Patients and Methods**

This study was carried out on 20 patients attending the outpatient clinic of neurology and ENT departments of Al-Dar Hospital, Al-Madina Al-Mounwar, KSA, through the period from June 2007 till June 2008 complaining of hoarseness of voice.

**Inclusion Criteria:**

Male or female patient aged from 30-70 years old presenting with sudden onset hoarseness of voice.

**Exclusion Criteria**

1. Patient with history of neck trauma in the last year prior to the study.
2. History of intubation.
3. Patient with neck swelling operated or not.
4. Patient with thyroid disease whether inflammatory or neoplastic.

The patients were subjected to the following:

1. Detailed history taking.
2. Thorough neurological exam.
3. Complete head and neck exam.
4. Fibro-optic laryngoscope
5. M.R.I brain "when indicated”.
7. C.T scan neck.
8. C.T scan chest.
9. Thyroid ultrasound.
10. Lab investigations: C.B.C, ESR, thyroid functions, NA, K, Ca, random blood sugar.

**Result**

This study included 20 patients who attended the Neurology and ENT department, Al-Dar private Hospital, Al-Madina, KSA in the period from June 2007 till June 2008, complaining of hoarseness of voice. To underpin the causes of this symptom, these patients underwent several procedures beginning with complete history, general and neurological examination then fibro-optic laryngoscope.

Thirteen patients were males. The hoarseness of voice was due to vocal cord palsy; 55% bilateral. As shown in figure (1), the age of patients varied; male patients were distributed as 25% slightly over 60 years, and from 20 – 60 there was an equal presentation of cases, 20%. Other wise female patients were over mostly between 20 and 40 years.

Figure (2) shows the distribution of the causes of vocal cord palsy in the 20 cases either idiopathic, central lesions, or peripheral. In figure 3, we tried to emphasis the neurological lesions that accompanied vocal cord palsy. They were multiple sclerosis, amyotrophic lateral sclerosis (motor neuron disease), syndromes (acute Guillian Barré & pseudo-bulbar), acute cerebral infarction, old stroke, Alzheimer's disease and other category includes patient with psychic condition, post-tonsillectomy case and patient with mild VII cranial nerve palsy.

To get a close description of the cases, we distribute them according to vocal cord palsy either unilateral or bilateral (Fig. 4).

There were other accompanying conditions with vocal cord palsy such as; past history of diabetes mellitus, hypertension, hemiplegia or earache. Three cases of those who had bilateral palsy needed tracheostomy to prevent aspiration.
Fig. (1): Shows the percentage of patients according to both gender and age group that was grouped with 20 years interval.

Fig. (2): Shows the percentage distribution of the causes of vocal cord palsy.

Fig. (3): Shows the percentage of the causes of vocal cord palsy in the study.
Fig. (4): Shows the percentage distribution according to the cause and the type of vocal cord palsy.

**DISCUSSION**

In this series of adult patients; more than half of the patients with idiopathic vocal cord palsy presented with neurological conditions, our study confirms and extends evidence, for such an association. Several retrospective reviews have suggested an association in children between idiopathic VCP and underlying neurological conditions.

In a consecutive sample of 102 cases of pediatric VCP, 35% were classified as idiopathic and 16% as having underlying neurological conditions, seven patients had Arnold-Chiari malformation, exhibited peripheral neurological disease, had hereditary distal spinal muscular degeneration and had Horner syndrome ipsilateral to the VCP.

Of 113 children diagnosed with congenital VCP, excluding post surgical cases; 37% had idiopathic VCP and 25% had associated neonatal neurological diseases.

In adults the rate of idiopathic VCP is similarly high and is sometimes associated with a neurological condition.

In a series of (61) adult patients with bilateral VCP 39% of cases were idiopathic. Havas et al. classified etiologies of unilateral VCP in 108 patients. 45 cases were iatrogenic, 36 cases were idiopathic, of the remaining 27 cases, 6 had a C.N.S. disorder or systemic neurological disorder, 3 had vagus neuroma or neurofibroma, 2 had post-polio syndrome.

Ramadan et al. evaluated 98 patients with unilateral VCP, the cause was found to be neoplastic disease in 32% of the cases, idiopathic in 16% or caused by surgery (30%), trauma (11%), C.N.S disorder (8%) all 8 cases was due to stroke or infection (3%). Associated cranial nerve injuries were found in (9) patients.

Srrompatang et al. evaluated (90) patients with unilateral VCP, finding that 29% of cases were due to neoplasm, 21% inflammation 8% trauma from endotracheal intubations 5% C.N.S disorder 24% iatrogenic idiopathic.

In our series of patients with unilateral VCP, it is difficult to make a pathophysiological connection between idiopathic VCP and some diagnosis "e.g. Alzheimer's dementia".

The neurological condition that proceeded VCP seem to be associated with either the brainstem out flow end of the neuraxis or the segmental laryngeal or esophageal anatomy (the neuromuscular disorder, multiple cranial neuropathy, esophageal dysmotility, laryngeal spasms).

Similarly, it's unclear whether VCP potentially heralds any subsequent neurological diagnosis. The
subsequent neurological condition included hearing loss, vertigo, multiple cranial neuropathies. These conditions seem to suggest an association of idiopathic VCP with neuromuscular disorders.

The left vocal cord is more vulnerable to injury than the right as noted here, the left recurrent laryngeal nerve is longer. Typically, there's a 28% difference in length, it can vary from 5-15 cm.\(^{12}\) In addition there is pronounced variation in the way in which the 2 recurrent laryngeal nerves meet the larynx\(^{13}\). Whether, such anatomical differences and susceptibility to disease are evidenced by the 2 fold increased incidence of left sided VCP (in our study not proved).

The neurological disorders reported to be associated with VCP seem to be divisible into central nervous system disorders (cerebrovascular disease, multiple sclerosis, multiple system atrophy, Parkinson's disease, Arnold- Chiari malformation) (in our series 45%) and peripheral nervous system disorders in our series (35%) (Guillain-Barré syndrome, chronic motor axonal neuropathy, myasthenia gravis, charcot -marie- tooth disease). This observation is in agreement with Daya et al.\(^{7}\), de Gaudemar et al.\(^{8}\), Isozaki et al.\(^{15}\) and Marchant et al.\(^{17}\).

Although cases of Idiopathic VCP can occasionally be bilateral. They are usually unilateral\(^{16}\). Our series of patients were all adults with unilateral VCP 45% and bilateral VCP 55%. The majority of them have preexisting neurological conditions (80%).

In our case series, we are unable to perform extensive medical chart abstractions on an age – matched control group that would enable us to determine rates of the identified neurological conditions, in our general population for comparison with the rates seen among our patients with VCP.

The time between the diagnosis of a preexisting neurological conditions and the subsequent evaluation of VCP was not determined. Therefore, any cause and effect relationship between neurological conditions and Idiopathic VCP can not be established in our study.

So, our findings suggest a need for further prospective cohort study of patients with idiopathic VCP with comparison to an age-matched population. Based control group to a certain whether a valid epidemiological and temporal association exists between idiopathic VCP and neurological disorders.

**Conclusion and Recommendations**

1. The number of patients diagnosed VCP is rising so physicians must be aware of its prevalence.
2. Given the frequency of associated neurological conditions in adult patients with idiopathic VCP, a careful neurological examination should be considered for all patients.
3. A prospective study focusing on serial neurological evaluation in patients with idiopathic VCP should be mounted.

**REFERENCES**

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