BILATERAL ABDUCTOR VOCAL CORD PALSY

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INTRODUCTION

• Vocal cord paralysis is a sign of a disease
• It results from dysfunction of Recurrent laryngeal nerves on both sides
• Paralysis of abductors of both the cords causes vocal cords to lie in the midline/paramedian position
• This compromises airway and causes respiratory distress (aggravated by Bernoulli’s effect) - STRIDOR
• Patients usually have a good voice and cough
• Aspiration and dysphagia not seen until unless SLN is involved
• The severity of stridor depends on the exertion
ANATOMY

1. Muscles acting on vocal cords
   • Abductor - Posterior cricoarytenoid
   • Adductors - Lateral cricoarytenoid
     Transverse arytenoids
     Thyroarytenoids
     Cricothyroid (weak)
   • Tensors - Thyroarytenoids (vocalis)
     Cricothyroid
Action of posterior cricoarytenoid muscles
Abduction of vocal ligaments

Cricothyroid joint
(pivot point)

Action of cricothyroid muscles
Lengthening (increasing tension) of vocal ligaments

Action of transverse arytenoid muscle
Adduction of vocal ligaments

Action of vocalis and thyroarytenoid muscles
Shortening (relaxation) of vocal ligaments

Action of lateral cricoarytenoid muscles
Adduction of vocal ligaments
ETIOLOGY

• Iatrogenic (82.8%)
  Thyroidectomy
  Cervical oesophageal surgeries
  Tracheal surgeries
  Intubation injuries
  Radiation

• Malignancy (8.6%)
  Oesophageal cancer
  Thyroid cancer
  Cervical nodal metastasis
  Lung cancer

• Neurological (3.8%)
  Brainstem ischemia
  Arnold chiari malformation
  Head injuries
  PCF tumors

• Mechanical (2.7%)
  NG tube syndrome
  Stent placement in proximal oesophagus

• Idiopathic (2.2%)
  Viral infections
Position of vocal cords

- Median
- Paramedian
- Intermediate (cadaveric)
- Slight abduction
- Full abduction
WAGNER GROSSMAN THEORY

• “In complete paralysis of recurrent laryngeal nerve, the cord lies in paramedian position due to intact cricothyroid muscle which is a weak adductor of the cord”
SEMON’S LAW

• “In course of a gradually progressing organic lesion involving Recurrent laryngeal nerve, the fibres supplying the abductors of the vocal cords are involved much earlier than the adductors”
  - 1st stage – abductors damaged with weak adduction
  - 2nd stage – cords midline and immobile
  - 3rd stage – cords in cadaveric position
CLINICAL PRESENTATION

- Chief presenting complaint is typically shortness of breath or stridor
- Degree of airway compromise is variable and can range from mild stridor on exertion to life threatening airway obstruction
- No hoarseness of voice due to close proximity of immobile cords
- Voice – monotone
- Speech – short hurried phrases interrupted by stridorous inspiration
CLINICAL HISTORY

Symptoms of etiology like H/O
- Trauma
- Fever with evening rise
- Weight loss
- Reduced appetite
- Swelling in neck
- Symptoms of hypo/hyperthyroidism
- Limb weakness / numbness
- Previous surgeries
- Prolonged intubation
- Radiation exposure
- Smoking
CLINICAL EXAMINATION

- Examination of cranial nerves
- Indirect laryngoscopy
- Direct laryngoscopy – to rule out local causes and to assess the CA joint fixity

Vocal cords are in paramedian position
INVESTIGATIONS

For diagnosis and morbidity assessment

- Rigid endoscopy (30° / 70°)
- Fibre optic laryngoscopy (FOL)
- Laryngeal electromyography (LEMG)
  - Differentiate between VCP, synkinetic activity, CA joint fixation
  - Determining the prognosis for recovery
  - Differentiate muscular and neurological causes
# LEMG

<table>
<thead>
<tr>
<th>Classes</th>
<th>Spontaneous activity</th>
<th>Recruitment of motor units</th>
<th>Individual motor unit morphology</th>
<th>Interpretation (prognosis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Absent</td>
<td>Normal</td>
<td>Normal MUAPs</td>
<td>Normal</td>
</tr>
<tr>
<td>II</td>
<td>Absent</td>
<td>Reduced</td>
<td>Small polyphasic MUAPs</td>
<td>Reinnervation</td>
</tr>
<tr>
<td>III</td>
<td>Absent</td>
<td>Reduced</td>
<td>Very large MUAPs</td>
<td>Old injury</td>
</tr>
<tr>
<td>IV</td>
<td>Present</td>
<td>Reduced</td>
<td>Polyphasic MUAPs</td>
<td>Equivocal</td>
</tr>
<tr>
<td>V</td>
<td>Present</td>
<td>Absent</td>
<td>Fibrillations, etc.</td>
<td>Denervation</td>
</tr>
</tbody>
</table>

MUAP, motor unit action potential.
Routine blood investigations

- CBP
- ESR
- RBS
- Rheumatoid factor
- Parathormone levels
- VDRL
- Thyroid profile
Radiological

- CECT skull base to aortic arch
- Gadolinium enhanced MRI Brain
- Barium swallow
- CT chest
- Targeted FNAC/biopsy (if any lesion detected)
SURGICAL MANAGEMENT

• AIRWAY MANAGEMENT
  1. Tracheostomy

• CORD PROCEDURES
  STATIC PROCEDURES
  1. Posterior corpectomy
  2. Arytenoidectomy
  3. Arytenoidopexy

  DYNAMIC PROCEDURES
  1. Nerve pedicle transfer
  2. Pacing of vocal cords
  3. Botox injection
• FRAMEWORK SURGERIES
  1. Type II thyroplasty
  2. Posterior cricoid lamina split

• OTHERS
  1. Gene therapy
  2. Stem cell therapy
TRACHEOSTOMY

- Warranted in acute respiratory embarrassment
- Temporary management
- Immediate relief of airway obstruction
- Indicated – Expecting spontaneous recovery
  - If surgical expertise/facilities not available
  - If dynamic/framework procedures are planned that do not provide immediate relief from airway obstruction
- Complications – infections, granulations, aphonia (can be overcome with a speaking valve)
POSTERIOR CORDECTOMY

• Irreversible procedure
• 2 approaches
  1. Laryngofissure
  2. Microlaryngoscopy and CO$_2$ LASER cordectomy (KASHIMA procedure)
• Dissection focussed on submucosa, preserving mucosa to avoid scarring and voice disturbance
• C shaped wedge of posterior vocal fold excised
• Susceptible to granulation formation, rough (when LASER not used) and breathy voice
ARYTENOIDECTOMY

- Permanent and irreversible
- Glottis expanded in transverse axis
  - Woodman’s approach (Lateral neck approach)
  - Anterior laryngofissure procedure
- Expose arytenoids → resect except vocal process → sutures through remaining arytenoid to thyroid lamina
• Endoscopic (Thornell’s, Ossoff’s technique, Crumley’s ELMA)

• CO\textsubscript{2} LASER excision of arytenoid body and vocal process

• Used alone or in combination with partial vocal fold resection
ARYTENOIDOPEXY

- Vocal cord abduction by suture lateralization
- Combined endoscopic and open approach
- Reversible – used as temporary management when spontaneous recovery is expected
- DLscopy → CA joint assessed → TA muscle released
- Sutures passed around vocal process and posterior third of vocal fold → fixed to thyroid ala / strap muscles
- Greater glottal opening, better phonatory closure, low risk of aspiration
Vocal cord lateralization (laterofixation / cordopexy)
LARYNGEAL PACING

- Functional electrical stimulation (FES) of paralysed muscle – Potential therapy
- If spontaneous recovery does not occur within a year
- Electrical pacing is delivered to Posterior cricoarytenoid muscle with EMG feedback signals from ipsilateral cricothyroid muscle through needle electrodes
- Trigger signals are produced as a product of feedback from Diaphragmatic EMG/ Intercostal EMG / temperature difference in respiratory cycle
• Greater ventilatory improvement than any other approach, no voice compromise
• Still experimental
• Very expensive (not available in India)
• Device has to be replaced every 5-10 years
REINNERVATION

• Prerequisites – CA joint not fixed
  Nerve for graft functioning
  EMG should reveal functioning muscle

• 2 types
  1. Nerve muscle transfer (Ansa cervicalis – Anterior belly of omohyoid)
  2. Nerve pedicle transfer (Ansa cervicalis/Phrenic nerve – RLN anastamosis)
TYPE II THYROPLASTY

- II a – lateral approach
- II b – medial/midline approach

Complication – breathy
POSTERIOR CRICOID SPLIT

• Posterior cricoid split + costal cartilage graft
• 2 approaches
  - Endoscopic
  - Partial laryngofissure (external)
BOTULINUM TOXIN

• Neurotoxin produced by Clostridium botulinum
• There are 7 types (A – G), type A and B most frequently used clinically
• Prevents release of acetylcholine from nerve terminals → flaccid paralysis
• Botox injection into bilateral adductor muscles found to improve ventilation
• Results found acceptable but temporary (≈ 3 months) therefore requires multiple injections
GENE THERAPY

• Genes encoding neurotrophic growth factors injected into damaged RLN and PCA muscle
• Stimulates cellular proliferation and differentiation
• Absorbed by the muscle cells / neurons by retrograde axonal transport transduced into nucleus produce peptides
• Animal studies using IGF – 1 gene ( PCA muscle), GDNF ( RLN) have shown improvement
• Major drawback is it does not prevent synkinesis
• Only animal studies performed so far
STEM CELL THERAPY

- Autologous muscle derived stem cells isolated, cultured to critical mass and re-implanted into PCA
- This attenuated overall muscle atrophy
- Drawback – cannot prevent synkinesis
- No human trials so far
CONCLUSION

• Bilateral abductor vocal cord palsy can range from exertional stridor to potentially fatal condition
• Therefore requires prompt and accurate diagnosis followed by adequate treatment
• Iatrogenic causes which are the leading need to be avoided
• Almost all the treatment modalities may produce a poorer voice quality as increasing the airway will always create a phonatory gap
THANK YOU