

CASE DESCRIPTION

Our patient is a 52-year-old male with past medical history of hypothyroidism, who presented to outpatient clinic due to shortness of breath. Two years prior, he attempted a back flip, landed on his head and neck, and sustained two thoracic compression fractures. One week later, he noticed difficulty running due to dyspnea. He went to an urgent care center. Chest X-ray showed an elevated diaphragm on the right. He suffered multiple bouts of pneumonia the year following and eventually had a Sniff test which showed complete paralysis of the right hemidiaphragm.

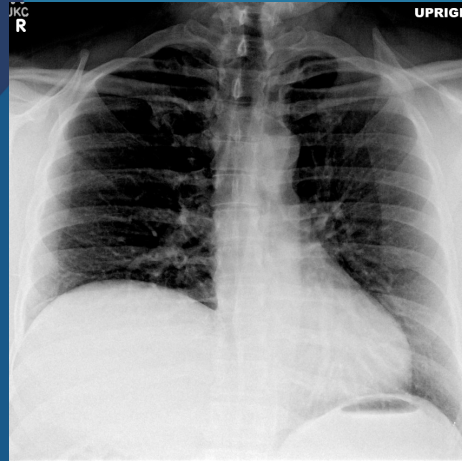
ASSESSMENT/RESULTS

He initially presented to outpatient clinic one year after onset of symptoms. During that time, he complained of dyspnea with exertional activities, such as lifting heavy objects or climbing stairs, inability to run due to severe shortness of breath, and orthopnea.

Results of nerve conduction study/electromyography (NCS/EMG) showed evidence of a right phrenic neuropathy with decreased motor units firing on EMG of the right hemidiaphragm and a reduced amplitude of response with direct stimulation of the right phrenic nerve. There was no evidence of an upper extremity peripheral neuropathy, right brachial plexopathy or right cervical radiculopathy. Diaphragm thickness measured by ultrasound was 0.23 cm on the left and 0.14 cm on the right.

Two days after initial visit, he underwent a right phrenic nerve grafting. He presented one year post-surgery for follow-up study. He reported since his surgery, he noticed a significant improvement in his breathing and functional abilities. He complained of mild dyspnea only when eating. He reported no limitations in daily activities and was able to run short distances.

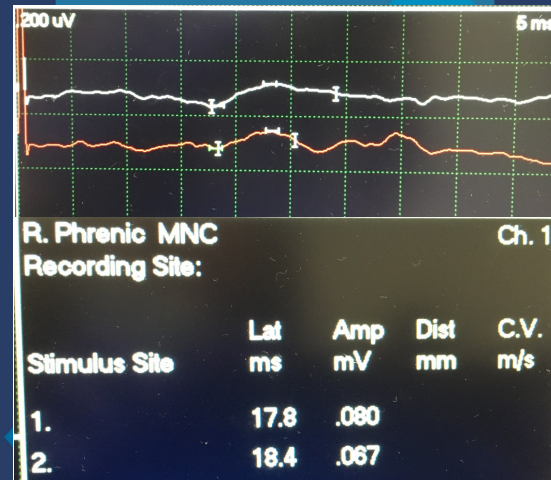
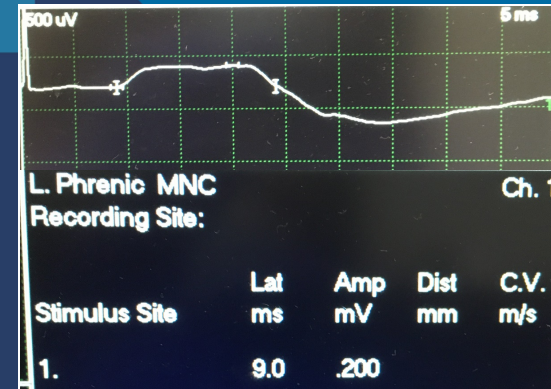
Results of repeat NCS/EMG showed improvement in the right phrenic nerve function in comparison to the previous study one year ago, with an increase in amplitude of response on direct stimulation. EMG of the right hemidiaphragm revealed polyphasic waves indicating some evidence of reinnervation. Diaphragm thickness measured by ultrasound was 0.23 cm on the left and 0.19 cm on the right.



Top Left: Upright chest X-ray revealing elevated right hemidiaphragm as compared to the left. Findings are consistent with a right phrenic nerve dysfunction.

Top Right: Normal left phrenic motor nerve conduction waveform. Note the amplitude of 0.200mV.

Bottom Right: Abnormal right phrenic motor nerve conduction waveforms. Note the decreased amplitudes of 0.080mV and 0.067mV, in comparison to the amplitude seen with the left phrenic nerve. This is indicative of a right phrenic neuropathy.



DISCUSSION

Diaphragmatic paralysis is most commonly caused by iatrogenic phrenic nerve injury. Patients can range from being asymptomatic to having severe dyspnea with minimal activity, and can suffer from complications such as recurrent pneumonias, progressive lung disease, or ventilatory failure. Treatment options includes surgical plication, ventilatory support, and/or diaphragmatic pacing. Extensive literature review reveals little to no reports of surgical nerve grafting for phrenic neuropathy in non-ventilatory-dependent patients.

CONCLUSION

While some patients remain asymptomatic, many suffer debilitating effects of diaphragmatic paralysis. Available treatment options may result in permanent lung dysfunction or dependence on ventilatory support. This unique case highlights the successful outcome of nerve grafting for a patient who suffered functional debilitation due to a unilateral phrenic neuropathy. Phrenic nerve grafting presents clinicians with an important treatment option that may result in more functional independence for their patients and improved quality of life.

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