Is Spinal Stenosis the Cause of Exercise-Induced Dyspnea? A case Report

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Spinal stenosis is a progressive degenerative condition characterized by the narrowing of the spinal canal, leading to compression of the spinal cord and nerve roots [1]. While commonly associated with aging, it can also develop following traumatic injuries that induce structural damage and chronic inflammation and contribute to canal narrowing [1]. Although spinal stenosis is primarily linked to neurogenic claudication, emerging evidence suggests that it may also impact respiratory function, particularly when it involves the cervical and thoracic spine [2].

We evaluated a 77-year-old male with a longstanding history of spinal stenosis and exertional dyspnea. We explored the potential connection between his symptoms and underlying spinal pathology, particularly in the absence of cardiopulmonary disease.

PATIENT DESCRIPTION

At the time of this study, YS was a 77-year-old Caucasian male (body mass index 32) with no history of smoking or significant past medical conditions other than a traumatic spinal injury sustained during a paratrooping jump 40 years ago. Imaging confirmed the presence of spinal stenosis. In recent years, he experienced progressive dyspnea while walking, limiting his endurance. He reported that his symptoms improved with lumbar flexion and rest.

In his youth, the patient was an active amateur athlete, primarily a long-distance runner and basketball player. At the time of this study, he would swim for 30 minutes 3–4 times per week without experiencing respiratory discomfort.

A comprehensive cardiovascular assessment revealed no abnormalities. Resting electrocardiogram showed a sinus rhythm with a heart rate of 82 bpm. Exercise electrocardiogram demonstrated no evidence of ischemia or arrhythmias. Echocardiography confirmed normal cardiac function at rest and during exercise, with no signs of pulmonary hypertension.

Complete pulmonary function tests were within normal limits. The percentage predicted values were as follows: forced expiratory volume in one second (FEV1) = 98%, forced vital capacity (FVC) = 101%, FEV1/FVC = 96%, total lung capacity = 88%, functional residual capacity = 71%, residual volume = 82%, and diffusing capacity of the lung for carbon monoxide (DLCO) = 89%. Chest computed tomography did not show any parenchymal abnormalities.

A cardiopulmonary exercise test (CPET) was conducted on a treadmill according to Bruce protocol [Figure 1A]. The patient reached a peak oxygen uptake (VO_2 peak) of 4.3 metabolic equivalent of tasks (METS) within 12 minutes, which was only 68% of the age-predicted value. His anaerobic threshold was within normal limits, maximal heart rate (133 bpm) was 92% of age-adjusted predicted value, the respiratory exchange ratio was 1.1 and O_2 pulse during exercise (9.9 ml/beat) was 86% of the predicted value, indicating adequate maximal effort and normal cardiovascular function. O_2 saturation remained 100% at peak exercise.

The patient's ventilatory efficiency was impaired. A high ventilatory equivalent for carbon dioxide (VE/VCO_2) of 45 reflected greater ventilatory requirement for eliminating the CO_2 produced by aerobic metabolism, while a low end-tidal carbon dioxide pressure ($EtCO_2$) of 28 mmHg, suggested hyperventilation. No re-

strictive breathing limitation was noted as tidal volume response was normal (700 ml at rest and 1800 ml during peak exercise). Although the breathing reserve during exercise was normal (27 L/min), the tidal flow-volume loop demonstrated inspiratory flow limitation, raising the possibility of an upper airway obstruction during exertion [Figure 1B].

COMMENT

This case highlights a patient with exertional dyspnea and impaired exercise capacity despite normal cardiopulmonary function. Given the absence of cardiac or pulmonary disease, an alternative explanation was considered. The presence of spinal stenosis, particularly its impact on neural control and musculoskeletal mechanics, provided a plausible link to the patient's symptoms.

The patient described dyspnea while walking. Dyspnea is a complex, multidimensional symptom defined as a subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity [3]. The underlying

Figure 1. A full respiratory dynamic response and the flow-volume loop during exercise

[A] A high Ve/VCO_2 slop with low $EtCO_2$ indicates a reduced ventilatory efficiency

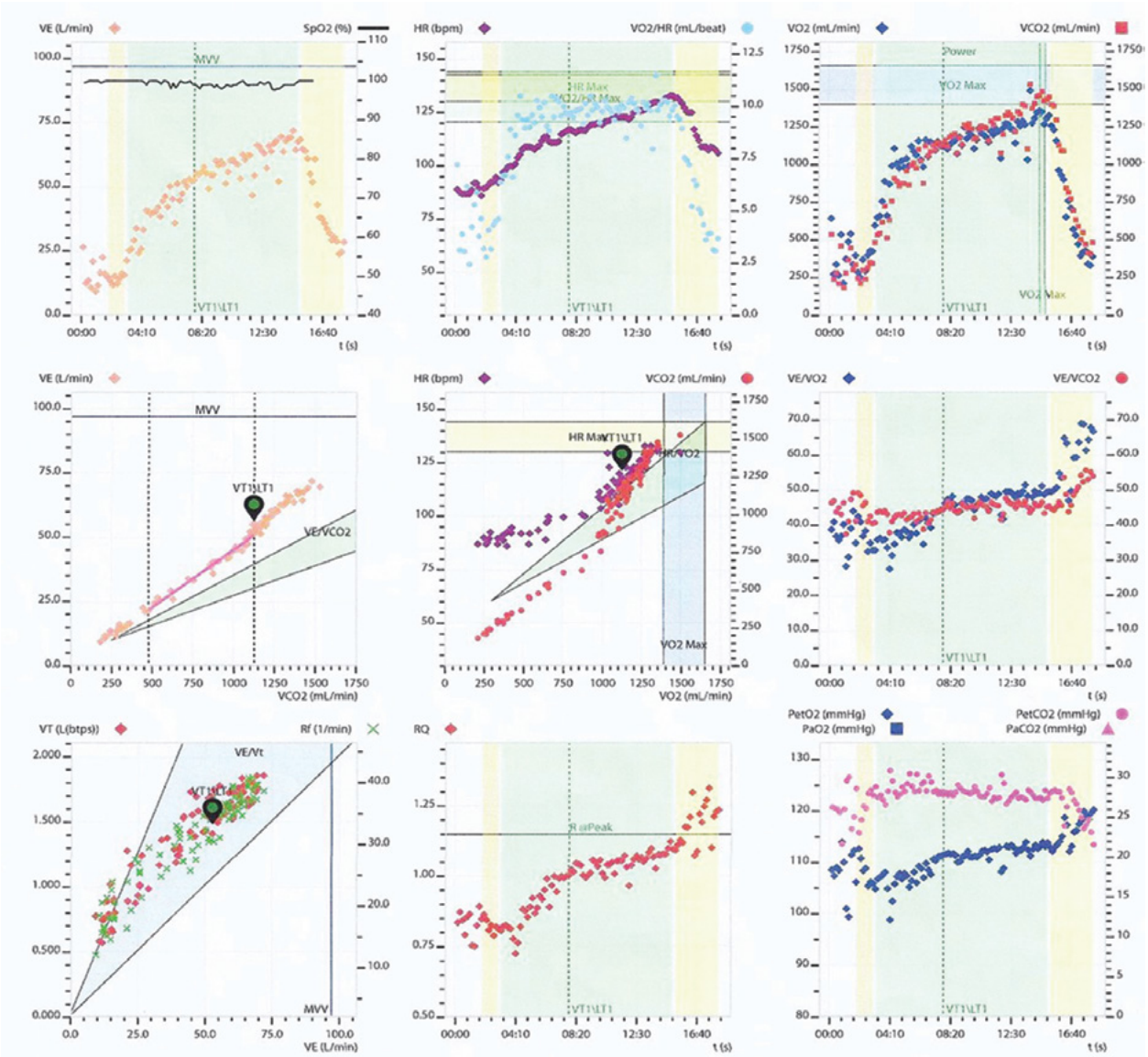
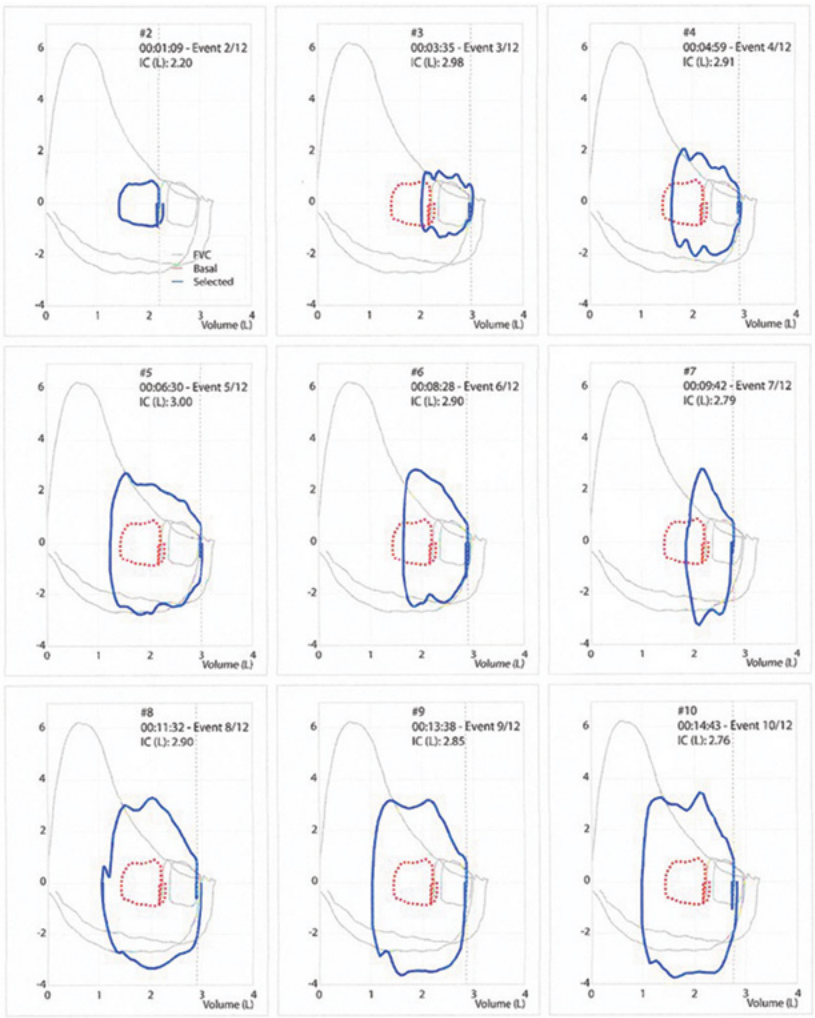


Figure 1. A full respiratory dynamic response and the flow-volume loop during exercise

[B] The inspiratory flow overlaps the maximal inspiratory flow



mechanisms involve altered respiratory mechanics, increased neural respiratory drive, and impaired gas exchange [3]. The etiology of dyspnea varies, including cardiovascular, pulmonary, neuromuscular, and psychological disorders [3]. Dyspnea is often associated with hyperventilation that is evident during CPET and is a compensatory response to hypoxia, acidosis, or increased respiratory drive, which ultimately contributes to exercise intolerance.

With no cardiovascular or pulmonary limitation, but reduced ventilatory efficiency (high VE/VCO_2 with low $EtCO_2$), the reason underlying the limited work capacity might be multifaceted. One possible explanation is the role of neurogenic claudication in limiting exercise capacity. Lumbar spinal stenosis often leads to pain, weakness, and gait disturbances, all of which contribute to deconditioning and reduced oxygen uptake [4]. Patients frequently assume a forward-flexed posture to relieve pressure on the spinal cord, which could also influence respiratory mechanics. The patient's improvement in symptoms with lumbar flexion supports this hypothesis. Chronic pain and neurological dysfunction associated with stenosis may further contribute to exercise intolerance by promoting biomechanical inefficiencies and neuromuscular fatigue [4].

Another potential mechanism involves impaired respiratory control due to cervical or thoracic spinal involvement. The phrenic nerve, which originates from the cervical spine (C3–C5), innervates the diaphragm, and any compromise at this level may disrupt normal breathing patterns [2]. Similarly, spinal cord compression in the thoracic region can affect the intercostal muscles, which play a key role in chest wall expansion. The patient's ability to alleviate symptoms by leaning forward suggests that reducing spinal cord compression may improve diaphragmatic excursion.

The inspiratory flow limitation observed during CPET raises concerns about upper airway dysfunction as a contributing factor. In some cases, spinal pathology can alter upper airway muscle coordination or lead to neuromuscular impairment, causing dynamic airway collapse during exertion [5]. While the exact mechanism remains unclear, this phenomenon could explain the patient's sensation of breathlessness despite normal lung function.

In terms of clinical implications, this case underscores the need for a multidisciplinary approach. Physical therapy focused on spinal mobility and core stability may help improve posture and overall endurance. In addition, breathing retraining techniques and inspiratory muscle training may optimize ventilatory efficiency.

CONCLUSIONS

Spinal stenosis can be an overlooked contributor to exercise-induced dyspnea, particularly when cardiopulmonary function remains intact. The combination of neurogenic claudication, altered respiratory mechanics, and potential upper airway dysfunction provides a reasonable explanation for the patient's symptoms. A comprehensive evaluation involving neurology, pulmonology, and rehabilitation medicine may provide valuable insights into effective management strategies.

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Capsule

Thiorphan reprograms neurons to promote functional recovery after spinal cord injury

Niekerk and colleagues previously identified an embryonic shift in the corticospinal motor neuronal transcriptome after spinal cord injury associated with successful axonal regeneration. Exploiting this transcriptional regenerative signature, the authors used in silico screens to identify small molecules that generate similar shifts in the transcriptome, and identified thiorphan–a neutral endopeptidase inhibitor–as a lead candidate. In a new adult motor cortex neuronal in vitro screen, thiorphan increased neurite outgrowth 1.8-fold ($P < 0.001$). Then they infused thiorphan into the central nervous system beginning 2 weeks after severe C5 spinal cord contusions and, when combined with a neural stem cell graft, thiorphan elicited significant improvements in forelimb function ($P < 0.005$) and corticospinal regeneration ($P < 0.05$). Extending clinical relevance, thiorphan significantly increased neurite outgrowth in primary cortical neuronal cultures from a 56-year-old human. These findings represent a new path for drug discovery, starting from in silico screens to proof-of-concept in adult human brain cultures.

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Capsule

Fixed-duration versus continuous treatment for chronic lymphocytic leukemia

Treatment of chronic lymphocytic leukemia (CLL) currently consists of two main approaches: continuous therapy with Bruton's tyrosine kinase inhibitors and fixed-duration regimens combining venetoclax with either CD20 antibodies or Bruton's tyrosine kinase inhibitors. Comparisons of these two therapeutic approaches are lacking. **Al-Sawaf** et al. conducted an investigator-initiated, phase 3, randomized trial involving patients with previously untreated CLL. A total of 909 patients were assigned to venetoclax–obinutuzumab (303 patients), venetoclax–ibrutinib (305 patients), or ibrutinib (301 patients). The median follow-up was 34.2 months. In this prespecified interim analysis, 3-year progression-free survival was 81.1% in the venetoclax–obinutuzumab group, 79.4% in the venetoclax–ibrutinib group, and 81.0% in the ibrutinib group (hazard ratio for venetoclax–obinutuzumab vs. ibrutinib = 0.8, 98.3% confidence interval [CI] 0.54–1.41; hazard ratio for venetoclax–ibrutinib vs. ibrutinib = 0.84, 98.0% CI 0.53–1.32). The results for each comparison met the criterion for noninferiority. After the end of treatment, minimal residual disease response in peripheral blood was undetectable in 73.3% of the patients in the venetoclax–obinutuzumab group, 47.2% in the venetoclax–ibrutinib group, and 0% in the ibrutinib group. Three-year overall survival was 91.5%, 96.0%, and 95.7%, respectively. The most common adverse events were infections, gastrointestinal disorders, and cytopenias.

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