

The reliability of differentiating neurogenic claudication from vascular claudication based on symptomatic presentation

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Background: Intermittent claudication can be neurogenic or vascular. Physicians use a profile based on symptom attributes to differentiate the 2 types of claudication, and this guides their investigations for diagnosis of the underlying pathology. We evaluated the validity of these symptom attributes in differentiating neurogenic from vascular claudication.

Methods: Patients with a diagnosis of lumbar spinal stenosis (LSS) or peripheral vascular disease (PVD) who reported claudication answered 14 questions characterizing their symptoms. We determined the sensitivity, specificity and positive and negative likelihood ratios (PLR and NLR) for neurogenic and vascular claudication for each symptom attribute.

Results: We studied 53 patients. The most sensitive symptom attribute to rule out LSS was the absence of "triggering of pain with standing alone" (sensitivity 0.97, NLR 0.050). Pain alleviators and symptom location data showed a weak clinical significance for LSS and PVD. Constellation of symptoms yielded the strongest associations: patients with a positive shopping cart sign whose symptoms were located above the knees, triggered with standing alone and relieved with sitting had a strong likelihood of neurogenic claudication (PLR 13). Patients with symptoms in the calf that were relieved with standing alone had a strong likelihood of vascular claudication (PLR 20.0).

Conclusion: The classic symptom attributes used to differentiate neurogenic from vascular claudication are at best weakly valid independently. However, certain constellation of symptoms are much more indicative of etiology. These results can guide general practitioners in their evaluation of and investigation for claudication.

Contexte : La claudication intermittente peut avoir une étiologie neurogène ou vasculaire. Les médecins utilisent un profil fondé sur les particularités des symptômes pour distinguer l'une de l'autre et ceci oriente leur choix des méthodes de diagnostic de la pathologie sous-jacente. Nous avons évalué la validité de ces particularités des symptômes utilisées pour distinguer la claudication d'origine neurogène de la claudication d'origine vasculaire.

Méthodes : Des patients atteints d'une sténose spinale lombaire (SSL) ou d'une maladie vasculaire périphérique (MVP) avérées qui se plaignaient de claudication ont répondu à 14 questions afin de caractériser leurs symptômes. Nous avons déterminé la sensibilité, la spécificité et les rapports de probabilité positifs et négatifs (RPP et RPN) à l'égard de la claudication neurogène ou vasculaire pour chacune des particularités des symptômes.

Résultats : Notre étude a regroupé 53 patients. La particularité des symptômes dotée de la sensibilité la plus élevée pour ce qui est d'écarter le diagnostic de SSL a été l'absence de « déclenchement de la douleur à la simple station debout » (sensibilité 0,97; RPN 0,050). Les données sur ce qui soulageait la douleur et sur la localisation des symptômes ont eu une faible portée clinique en ce qui a trait à la SSL et à la MVP. La présence d'une constellation de symptômes a donné lieu aux associations les plus solides : les patients qui manifestaient un signe du « panier d'épicerie » positif et dont les symptômes étaient localisés au-dessus du genou, déclenchés par la station debout seule et soulagés en position assise présentaient une forte probabilité de claudication d'origine neurogène (RPP 13). Chez les patients dont les symptômes étaient localisés au mollet et qui étaient soulagés par la station debout, on notait une forte probabilité de claudication d'origine vasculaire (RPP 20,0).

Conclusion : Considérés individuellement, les attributs classiques des symptômes utilisés pour distinguer la claudication d'origine neurogène de la claudication d'origine vasculaire sont au mieux faiblement valides. Toutefois, certaines constellations de symptômes éclairent bien davantage l'étiologie. Ces résultats peuvent guider l'omnipraticien dans son examen et dans son diagnostic de la claudication.

Intermittent claudication, recognized as pain in the legs associated with walking, is commonly reported among elderly patients.¹⁻⁴ Two common pathologies produce intermittent claudication: lumbar spinal stenosis (LSS) resulting in “intermittent neurogenic claudication” (sometimes referred to as LSS syndrome) and peripheral vascular disease (PVD) leading to “intermittent vascular claudication.” Although both produce activity-limiting symptoms in the legs, the pathogenesis of each differs. For neurogenic claudication, an extended lumbar posture narrows a degenerative stenotic spinal canal to a critical threshold, leading to direct mechanical compression or indirect vascular compression of the nerve roots and/or cauda equina.^{5,6} In vascular claudication, arterial vessel narrowing restricts blood flow to levels insufficient to match the metabolic demands of the lower extremity musculature.⁷ Verbiest⁸ first described the symptom similarities for patients with these 2 types of claudication in 1954. Since that time, many authors have attempted to distinguish characteristics of these 2 patient groups.⁹⁻¹²

Despite today’s noninvasive imaging technology, physicians must continue to rely on history and physical examination to guide their diagnosis. This is because the presence of LSS on magnetic resonance imaging (MRI) or computed tomography (CT) scans has been shown to poorly correlate with lower extremity symptoms.¹²⁻¹⁸ Similarly, not all patients with PVD, demonstrated by their arterial brachial index (ABI) measurement on a Doppler ultrasound, have claudication symptoms.^{19,20} Moreover, both LSS and PVD can coexist in the same patient, and imaging studies are unhelpful in deciding which must be treated for relief of their claudication symptoms.^{21,22} Finally, the cost of MRI and CT, if performed for every patient reporting intermittent claudication, would become an unreasonable financial burden to our health care system.

Historically, physicians have used different symptom attributes to differentiate neurogenic from vascular claudication. They consist of symptom triggers and alleviators, predictability of onset and time for relief, location and nature of the symptoms and association with posture.^{9,11,12,23-26} Despite being theoretically consistent with the pathogenesis of either claudication type, these associations have not, to our knowledge, been clinically validated in the literature.

The purpose of our study was to establish the validity of these symptom attributes in differentiating intermittent neurogenic claudication from vascular claudication. To do so, we calculated the sensitivity, specificity, positive likelihood ratio (PLR) and negative likelihood ratio (NLR) for these symptoms. We hypothesized that a subset of these symptom attributes would reliably differentiate the 2 types of claudication. We hope the results will guide physicians in determining the etiology of intermittent claudication based on symptomatic presentation without relying on multiple expensive investigations.

METHODS

This was an observational cohort study approved by our institution research ethics review board. A research associate explained the study and provided a letter of information to every patient who fulfilled the inclusion and exclusion criteria described in the next section. We obtained written consent from each patient enrolled in the study.

Selection and description of participants

We recruited patients attending the spine or vascular clinics at a tertiary care centre between July 2008 and July 2011 who reported intermittent claudication. Our inclusion criteria were a 6-month history of intermittent claudication symptoms (defined as “induction of pain in the legs and/or buttocks with walking, alleviated with standing or sitting”) and either clinically important LSS or PVD diagnosed by a fellowship-trained orthopedic spine surgeon or vascular surgeon, respectively. The exclusion criteria were concurrent PVD and LSS, nondegenerative LSS (e.g., congenital, traumatic), mechanical back pain equal to or greater than claudication symptoms, radicular pain due to foraminal stenosis, previous back or vascular surgery, type 1 diabetes, lower extremity peripheral neuropathy, symptomatic hip or knee osteoarthritis, total hip or knee arthroplasty, inability to read/write English, inability to provide informed consent, substance abuse and/or mental illness.

To confirm that every patient had a single pathology, either LSS or PVD, each patient underwent MRI of the lumbar spine and had their ABI measured with Doppler ultrasonography. A fellowship-trained orthopedic spine surgeon (C.S.B.) reviewed the MRI scans in a blinded fashion to determine if severe central canal stenosis (LSS) was present. Certified ultrasonography technicians performed the ABI measurements. Values less than 0.9 were labelled as positive for PVD.^{19,20} Patients with findings positive for one test and negative for the other were enrolled in the appropriate group. No patients had positive results for both LSS and PVD.

Outcome measures

Patients completed a questionnaire (see the Appendix, available at cma.ca/cjs) pertaining to their claudication symptoms. This was completed with the assistance of a research associate or surgical resident to ensure proper understanding of the questions. All questions addressed different symptom attributes, including symptom triggers and predictability of onset, symptom alleviators and time for relief, symptom location, nature of the symptoms and association between symptoms and body posture. Each question elicited a response of “yes” or “no.”

Statistical analysis

We tested each question for its sensitivity, specificity, PLR and NLR (Table 1) for both the neurogenic and vascular groups. If a test item (i.e., symptom attribute) has a high sensitivity, a negative response can be useful in ruling out a disease; conversely, if a test item (or symptom attribute) has a high specificity, a positive response indicates a high probability of the presence of the disease. Because the prevalence of each pathology is unclear, sensitivity and specificity are less valid in determining clinical relevance. On the other hand, likelihood ratios are not affected by prevalence, and are therefore more valid in this context. They indicate how much the odds of having the disease increase when the test is positive (PLR) or decrease when the test is negative (NLR; Table 2).^{27,28} We began by calculating these values for individual symptom attributes. This yielded weak clinical significance only; therefore, we performed the same statistical analysis using combinations of symptom attributes. To do this, we combined in a stepwise fashion the attributes with the highest PLR values whose 95% confidence interval (CI) did not include 1 until we reached a PLR greater than 10, indicating a strong evidence for the pathology in question (LSS or PVD).

RESULTS

We enrolled 53 patients (12 women and 18 men, with an average age of 65 ± 7.6 yr in the neurogenic group and 8 women and 15 men with an average age of 61 ± 8.1 yr in the vascular group). The sensitivity, specificity, PLR, and NLR are provided for each symptom attribute classically associated to the neurogenic and vascular groups (Tables 3 and 4, respectively). We analyzed every attribute for both groups. All PLRs obtained were less than 5 for any of the individual symptom attributes investigated (Tables 3 and 4). This implies that a single symptom attribute represents weak evidence for one type of claudication over the other (Table 2). This result led us to create constellations of symptom attributes; these are presented in Tables 3 and 4.

Neurogenic claudication

The symptom attribute best able to rule out intermittent neurogenic claudication (i.e., high sensitivity) was the absence of “triggering of pain with standing alone” (sensitivity 0.97, NLR 0.050; Table 3). The 3 largest PLR values were the alleviation of symptoms when sitting (PLR 3.8), triggering of symptoms when standing alone (rather than walking; PLR 3.2) and symptoms located above the knee (PLR 2.3; Table 3). In addition, the NLR values for these 3 symptom attributes were the smallest (0.21, 0.04 and 0.31, respectively), meaning that not only are these attributes most closely associated with neurogenic claudication, they are also the ones least closely associated with vascular claudication. Furthermore, despite small sample numbers, the 95% CI for the PLR and NLR values of each of these 3 symptom attributes did not include 1. However, because the PLR and NLR values were less than 5, they individually represented only weak evidence of neurogenic claudication.

Certain constellations of symptoms yielded stronger associations and, therefore, more clinically relevant results. The constellation consisting of triggering of symptoms with standing, relief with sitting, symptoms located above the knees and a positive shopping cart sign yielded a PLR of 13 (Table 2).

Vascular claudication

The absence of the triggering of pain with walking was the most sensitive symptom with which to rule out vascular claudication in this cohort (sensitivity 0.96). However,

Table 2. Interpretation of likelihood ratio values for clinical application²⁷

Likelihood ratio	Interpretation
> 10	Strong evidence to rule in the disease
5–10	Moderate evidence to rule in the disease
2–5	Weak evidence to rule in the disease
0.5–2	No significant change in the likelihood of the disease
0.2–0.5	Weak evidence to rule out the disease
0.1–0.2	Moderate evidence to rule out the disease
< 0.1	Strong evidence to rule out the disease

Table 1. Statistical calculations used for analysis²⁷

Term	Definition	Formula
Sensitivity	Likelihood that the diagnostic test will indicate the presence of disease when the disease is actually present	$T+ \div (T+ + F-)$
Specificity	Likelihood that the diagnostic disease will indicate the absence of disease when the disease is actually absent	$T- \div (T- + F+)$
Positive likelihood ratio	Indicates how much more likely it is to get a positive test in a person with than without the disease	$\frac{\text{Sensitivity}}{(1 - \text{Specificity})}$
Negative likelihood ratio	Indicates how much more likely it is to get a negative test in a person without than with the disease	$\frac{(1 - \text{Sensitivity})}{\text{Specificity}}$

F+ = false positive; F- = false negative; T+ = true positive; T- = true negative.

Table 3. Symptom attributes for neurogenic intermittent claudication

Attribute*	Measure (95% CI)			
	Sensitivity	Specificity	PLR	NLR
Single symptom attributes				
Trigger				
Standing (1)	0.97 (0.81–1.0)	0.70 (0.47–0.86)	3.2 (1.7–5.9)†	0.04 (0.0067–0.34)†
Walking (2)	0.90 (0.72–0.97)	0.04 (0.0023–0.24)	0.94 (0.81–1.1)	2.30 (0.12–43)
Alleviator				
Sitting (3a)	0.83 (0.65–0.94)	0.78 (0.56–0.92)	3.80 (1.7–8.5)†	0.21 (0.083–0.44)†
Posture				
Shopping cart sign (4)	0.80 (0.61–0.92)	0.52 (0.31–0.73)	1.70 (1.1–2.7)†	0.38 (0.17–0.85)†
Walking uphill (7)	0.23 (0.11–0.43)	0.78 (0.55–0.92)	1.07 (0.39–2.9)	0.98 (0.79–1.2)
Nature				
Numbness (8)	0.75 (0.55–0.89)	0.41 (0.21–0.63)	1.30 (0.84–1.9)	0.61 (0.28–1.3)
Cramping (9)	0.53 (0.35–0.71)	0.35 (0.17–0.57)	0.82 (0.52–1.3)	1.30 (0.78–2.3)
Burning pain (10)	0.62 (0.42–0.79)	0.52 (0.31–0.73)	1.30 (0.78–2.2)	0.73 (0.42–1.3)
Weakness (11)	0.43 (0.25–0.63)	0.59 (0.37–0.79)	1.00 (0.54–2.0)	0.97 (0.66–1.4)
Location				
Above the knees (5)	0.80 (0.61–0.92)	0.65 (0.43–0.83)	2.30 (1.3–4.1)†	0.31 (0.14–0.66)†
Time for relief				
> 10 min	0.30 (0.15–0.50)	0.78 (0.56–0.92)	1.40 (0.53–3.6)	0.89 (0.69–1.1)
Constellation of symptom attributes				
Triggered with standing (1), alleviated with sitting (3a)	0.80 (0.61–0.92)	0.87 (0.65–0.97)	6.10 (2.1–18)†	0.23 (0.11–0.48)†
Triggered with standing (1), alleviated with sitting (3a), located above the knees (5)	0.67 (0.47–0.82)	0.91 (0.70–0.98)	7.70 (2.0–30)†	0.37 (0.22–0.61)†
Triggered with standing (1), alleviated with sitting (3a), located above the knees (5), positive shopping cart sign (4)	0.57 (0.38–0.74)	0.96 (0.76–1.0)	13.00 (1.9–91)†	0.45 (0.30–0.68)†

CI = confidence interval; NLR = negative likelihood ratio; PLR = positive likelihood ratio.
 *Numbers in brackets represent the corresponding question number in the questionnaire (see the Appendix, available at cma.ca/cjs).
 †Numbers whose values represent clinical significance.

Table 4. Symptom attributes for vascular intermittent claudication

Attribute*	Measure (95% CI)			
	Sensitivity	Specificity	PLR	NLR
Single symptom attribute				
Trigger				
Walking (2)	0.96 (0.76–1.00)	0.10 (0.03–0.28)	1.10 (0.92–1.2)	0.43 (0.04–5.3)
Symptom onset				
Predictable (2a)	0.87 (0.65–0.97)	0.37 (0.21–0.56)	1.37 (1.0–1.9)	0.36 (0.11–1.1)
Alleviator				
Standing (3)	0.78 (0.56–0.92)	0.90 (0.72–0.97)	7.80 (2.6–23)†	0.24 (0.11–0.53)†
Nature				
Numbness (8)	0.59 (0.37–0.79)	0.25 (0.11–0.45)	0.79 (0.52–1.2)	1.60 (0.80–3.3)
Cramping (9)	0.65 (0.43–0.83)	0.47 (0.29–0.65)	1.20 (0.78–1.9)	0.75 (0.40–1.9)
Burning pain (10)	0.47 (0.27–0.69)	0.38 (0.21–0.58)	0.77 (0.46–1.3)	1.37 (0.83–2.3)
Weakness (11)	0.41 (0.21–0.63)	0.57 (0.37–0.75)	0.95 (0.49–1.8)	1.00 (0.69–1.5)
Location				
Calves (6)	0.78 (0.56–0.92)	0.73 (0.54–0.87)	2.90 (1.6–5.5)†	0.30 (0.13–0.66)†
Time for relief				
1–2 min (11a)	0.57 (0.35–0.76)	0.57 (0.38–0.74)	1.30 (0.76–2.2)	0.77 (0.46–1.3)
Constellation of symptom attributes				
Alleviated with Standing (3), located in the calves (6)	0.65 (0.43–0.83)	0.97 (0.81–1.0)	20.00 (2.8–140)†	0.36 (0.21–0.63)†

CI = confidence interval; NLR = negative likelihood ratio; PLR = positive likelihood ratio.
 *Numbers in brackets represent the corresponding question number in the questionnaire (see the Appendix, available at cma.ca/cjs).
 †Numbers whose value represent clinical significance.

the specificity of this attribute was 0.10, and both the PLR and NLR were very close to 1 (PLR 0.99; NLR 1.1), indicating that patients with neurogenic claudication experience pain with walking almost as frequently as those with vascular claudication. Therefore this symptom cannot be used as a positive discriminator for vascular claudication.

The alleviation of pain with standing alone (i.e., standing still/no walking) represents moderate evidence for vascular claudication (PLR 7.8). Symptoms located below the knees had a weak association with this etiology (PLR 2.9). The 95% CIs for both values did not include 1.

As in the neurogenic group, combination of symptom attributes led to stronger associations. The constellation consisting of alleviation of symptoms with standing alone, and symptoms located below the knees yielded a PLR of 20, which represents strong evidence for vascular claudication.

DISCUSSION

In 1858, Charcot²⁹ described vascular claudication as “weakness, numbness, cramping and stiffness of the legs with walking, and prompt relief with rest.” It was not until almost a century later, in 1954, that Verbiest recognized the clinical syndrome of LSS.⁸ He claimed “the most typical symptoms are tiredness and loss of power in the legs, anaesthesia and a feeling of numbness in the sacral dermatomes, and bilateral sciatica.” Relevant to the findings of our study, he stated, “the symptoms are present only on standing or walking.” He noted the resemblance to vascular claudication and recognized the clinical equipoise that this posed.⁸ Subsequent publications have attempted to differentiate neurogenic from vascular claudication, but relied on clinical impression rather than quantitative assessment.³ This has led to the classic associations of certain symptom attributes with either neurogenic or vascular claudication, yet the validity of these associations remains unclear. The present study established the validity of specific symptoms in differentiating neurogenic from vascular claudication.

Our study demonstrates that use of symptom attributes in isolation to differentiate between neurogenic and vascular claudication is weak at best. These attributes include the location of the symptoms (above the knees for neurogenic claudication; below the knees for vascular claudication) as well as the symptom alleviators (sitting for neurogenic claudication; standing for vascular claudication). However, alleviation of symptoms with standing alone has a moderate correlation with vascular claudication; its presence could direct physicians toward a vascular workup rather than neurogenic investigations.

Certain constellations of symptom attributes are more strongly associated with each type of claudication. We found that the presence of symptoms that are triggered with standing, relieved with sitting, located above the knees and have a positive shopping cart sign represent strong evi-

dence that a patient has intermittent neurogenic claudication rather than vascular claudication. On the other hand, a patient with symptoms that are relieved with standing alone and located below the knees is much more likely to have vascular than neurogenic claudication.

In 1978, Hawkes and Roberts¹² compared the clinical history and examination between patients with degenerative LSS and those with PVD. They found that the vascular group had relief of symptoms when standing and a constant claudication distance and that the neurogenic group had pain with standing and variable claudication distance. Our study demonstrated similar findings regarding the dependence of symptoms with respect to standing, but we found that symptom onset with “constant claudication distance” was not predictive of the claudication type.

Dodge and colleagues²¹ reviewed a series of patients with LSS who were treated with decompressive lumbar spine surgery. They found that some patients had persistent symptoms postoperatively due to a secondary vascular etiology contributing to their claudication. They noted that a cramping type of discomfort can be associated with both types of claudication, but that a motor deficit can usually be attributed to LSS. While our study supports their observation with respect to the cramping nature of the pain, we did not find a greater prevalence of subjective weakness in the neurogenic group, which differs from their suggested association with objective weakness.

Four recent studies³⁰⁻³³ have examined symptomatic presentation of patients with LSS, and the results of these studies were reviewed in a meta-analysis ($n = 741$) by Suri and colleagues.⁴ The study population was not limited to patients with specific reports of intermittent claudication; rather, patients presenting with LBP and/or lower extremity pain were included. The meta-analysis concluded that the symptoms most commonly associated with LSS were absence of pain when seated, improvement of symptoms when bending forward and presence of bilateral buttock or leg pain. They also suggested that most of the symptom attributes classically associated with LSS were not specific to neurogenic claudication. Importantly, our data are in agreement with the results of the meta-analysis with respect to the positional influence of symptoms in patients with LSS.

Strengths and limitations

Strengths of our study include its prospective design and the fact that all patients were assisted by 1 of 2 health care staff members in a single institution, ensuring proper, consistent interpretation and understanding of every item on the questionnaire. Also, our cohorts comprised patients whose mainly reported lower extremity claudication symptoms rather than nonspecific low back pain or lower extremity symptoms.

Our study is limited by the size of our patient cohorts.

Despite this limitation, we did obtain statistically significant results, as demonstrated by 95% CIs. Furthermore, it would have been useful to include physical examination findings in our analysis.

CONCLUSION

Our results indicate that most symptom attributes in isolation have limited reliability in diagnosing neurogenic or vascular claudication. However, there is a specific constellation of symptoms that can significantly increase the likelihood of accurately predicting the underlying etiology of claudication. For neurogenic claudication, the constellation consists of symptoms that are triggered with standing, relieved with sitting and located above the knees and that have a positive shopping cart sign. For vascular claudication, the constellation consists of symptoms that are relieved with standing alone and located below the knees. Our results can guide physicians and surgeons in their evaluation of patients reporting intermittent claudication and encourage the appropriate use of health care resources.^{34,35}

Competing interests: None declared.

Contributors: M. Nadeau, M. Rosas-Arellano, K. Gurr, S. Bailey and C. Bailey designed the study. M. Nadeau, M. Rosas-Arellano, K. Gurr, S. Bailey, D. Taylor, K. Lawlor and C. Bailey acquired the data, which M. Nadeau, M. Rosas-Arellano, K. Gurr, R. Grewal and C. Bailey analyzed. M. Nadeau and C. Bailey wrote the article, which all authors reviewed and approved for publication.

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