What is the diagnostic approach to a patient with leg cramps?

Evidence-Based Answer

A careful history targeting possible peripheral vascular disease, arthritis, and medication use (particularly diuretics, statins, and long-acting bronchodilators) is likely helpful in evaluating a patient with leg cramps. (SOR: **B**, observational studies). Laboratory evaluation is not known to be helpful (SOR: **C**, extrapolated from studies in runners).

A 1999 cross-sectional study of adults 65 years of age and older (N=365) investigated the prevalence of leg cramps and their association with specific disease conditions.[1] Data were collected from patient questionnaires administered over a 6-month period. The overall prevalence of leg cramps was 50%. Leg cramps were most prevalent at night (62%).

Patients with leg cramps showed a strong association with peripheral vascular disease (OR 2.9; 95% CI, 1.9–4.6), arthritis (OR 2.3; 95% CI, 1.5–3.5), and female sex (OR 2.0; 95% CI, 1.3–3.0) compared with individuals without leg cramps.[1]

A 2012 sequence symmetry analysis sought to determine if treatment for muscle cramps increased during the year after initiation of statin, diuretic, or long-acting β2-agonist (LABA) therapy.[2] By searching healthcare databases in British Columbia and Canada, the number of new quinine prescriptions written (initiated for treatment of cramps) were identified before and after the initiation of treatment with the index medications, in adults 50 years or older.

Adjusted sequence ratios (ASR) showed initial quinine prescriptions were significantly more likely to follow rather than precede new prescriptions for all index medications: diuretics (ASR 1.5; 95% CI, 1.3–1.6), statins (ASR 1.2; 95% CI, 1.0–1.3), and LABAs (ASR 2.4; 95% CI, 2.0–2.9). Both thiazide-like diuretics (ASR 1.5; 95% CI, 1.3–1.7) and loop diuretics (ASR 1.2; 95% CI, 1.0–1.4) were associated with increased quinine prescriptions. Thus in patients presenting with complaints of leg cramps, the possibility of diuretics, statins, and LABAs as etiologic agents should be explored.[2]

A 2003 individual cohort trial evaluated the development of exercise-associated muscle cramping (EAMC) in 72 long-distance runners.[3] The runners were divided into those with and without a history of EAMC. Lab studies (Na, K, Ca, Mg, and hemoglobin) and weight were measured pre-race, immediately post-race (56 km), and 60 minutes post-race.

The only laboratory measurements showing statistically significant differences between the groups were a lower immediate post-race serum sodium concentration in the cramp group versus the control group (mean 140 vs 142 mEq/L; *P*=.004) and higher immediate post-race magnesium level (1.7 vs 1.6 mg/dL; *P*=.03).

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1. Abdulla AJ, et al. *Int J Clin Pract.* 1999; 53(7):494–496. **[STEP 3]**
2. Garrison SR, et al. *Arch Intern Med.* 2012; 172(2):120–126. **[STEP 3]**
3. Schwellnus MP, et al. *Br J Sports Med.* 2004; 38(4):488–492. **[STEP 2]**

Is there an increased rate of infectious endocarditis in pacemaker patients?

Evidence-Based Answer

Yes. The estimated risk of infectious endocarditis (IE) after first pacemaker implantation is roughly 0.1% per year or a 1% risk in 10 years. Although rare, IE is significantly more common among patients with pacemakers than among the general population (SOR: **B**, cohort studies and a heterogeneous systematic review)

The best estimates of the incidence of IE in the general population vary from 1.4 to 7.9 per 100,000 population per year (or 0.0014%–0.0079% per year).[1,2]

An inception cohort of more than 46,000 Danish patients, who had a pacemaker implanted between 1982 and 2007, evaluated the incidence of pacemaker infection after insertion.[3] Removal due to infection after first implantation was 0.18% per pacemaker per year. The investigators made the assumption that infections requiring device removal within the first year were most likely due to surgical-site infections, whereas those that occurred after 1 year were more likely to be due to endocarditis. By this definition, the weighted incidence of IE in pacemakers was 0.102% per year or 102 per 100,000 pacemaker patients per year.

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