NON CARDIAC CHEST PAIN - CURRENT CONCEPTS

Pages with reference to book, From 1 To 3

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Retrostemal chest pain usually causes anxiety both in patients and physicians because of possible fear of cardiac disease. Some such patients continüe to have chest pain despite the exclusion of coronary artery disease and remain persistently anxious, and some of them even adopt compromised life styles[1]. Are all retrosternal chest pains cardiac in origin? This question was partially answered by the introduction of electrocardiography and later by angiography and cardiac catheterization, which have shown normal coronary arteries in as many as 30% of patients with anginal syndrome[3]. The cause of recurrent noncardiac chest pain is a clinical dilemma. The role of oesophagus in this condition has been extensively studied recently, and it has been found that approximately 50% of these patients have demonstrable oesophageal abnormalities[2-4].

Chest pain evaluation should always begin with the exclusion of cardiac disease due to its serious prognosis. Although these two diseases may not be differentiated by history but features like age, family history and risk factors for cardiac disease may be helpful in differentiating cardiac from non-cardiac pain. In younger patients it can be excluded by a normal electrocardiogram, exercise tolerance test and echocardiogram. Older patients may need angiography or ergonovine test[5]. Non- cardiac chest pains are usually nonexertional but occasionally gastro-oesophageal reflux may be triggered by heavy exercise and may produce chest pain mimicking angina pectoris even during treadmill examination[6]. Other features in favour of oesophageal origin are pain continuing for hours, without lateral radiation, which interrupts sleep. It is often meal related which is relieved with antacids and the presence of other oesophageal symptoms like heartburn, dysphagia or refluxing[7].

Sometimes both problems may coexist and produce diagnostic difficulty. In one study 50% of the coronary patients were found to have oesophageal disease[8], , in another 58-75% of patients with microvascular ang?na had oesophageal motility disorders[9,10]. Patients with concomitant heart and reflux disease may develop pain and show electrocardiographic changes during acid perfusion test[11]. Later it was confirmed that acid reflux can lower the exertional angina threshold[12], probably due to the phenomenon of summation of similar pain stimuli from heart and oesophagus or it could be a generalized abnormality in smooth muscle function which produces pain in both ways.

Recently several oesophageal motility disorders have been recognised by newer technology but the significance of these tests are still controversial[13]. The best understood oesophageal motility disorder is “Achalasia”, in which there is incomplete relaxation of the lower oesophageal sphincter, and the absence of normal peristalsis in the distal oesophagus. “Diffuse oesophageal spasm” shows intermittent presence of simultaneous contractions in the distal oesophagus which impair bolus transport. “Nutcracker Oesophagus” has been diagnosed in 27-48% of patients with non cardiac chest pain[14-17] which shows a pattern of high- amplitude peristaltic contractions (180mm Hg) which rarely impairs oesophageal function[22]. “Hypertensive lower oesophageal sphincter” shows increased pressure of the lower oesophageal sphincter at rest associated with normal relaxation of the sphincter and oesophageal peristalsis. Half of these patients also have high amplitude peristaltic contractions. “Nonspecific Oesophageal Motility Disorder” shows broad spectrum abnormality but do not fit into any of the clearly defined categories, their clinical significance remains to be explained. Recent studies have shown that 20-60% of patients with non- cardiac chest pain show abnormal manometric findings[14,16-21] of whom the most frequent is the nutcracker oesophagus being 34% while only 16% have other

manometric abnormalities[14]. Importance of an oesophageal motility disorder particularly when chest pain is absent is controversial; thus the detection of abnormal motility does not prove its oesophageal origin and should be considered only a clue.

Non cardiac chest pain of oesophageal origin may be confirmed by provocative tests like 'Acid Perfusion (Bernstein) test' which is useful in patients with reflux disease who present with chest pain rather than heartburn. The test yields a positivity of 7-27% and is specific for oesophageal chest pain[14,21]. 'Ergonovine test' is very rarely done due to its cardiac side effects. 'Endrophonim (Tensilon) test' is highly specific being positive in 24-34% in different doses[22,23] and does not occur in normal persons or in patients with irritable bowel syndrome. 'Bethanicol test' shows exceptionally high positivity upto 77% with two repeated doses[24] but unfortunately man; patients experience troublesome side effects[13] . 'Balloon distension' of oesophagus may also elicit chest pain in patients with noncardiac chest pain but it varies with individual's pain threshold, like that in rectal balloon distension in patients with irritable bowel syndrome[25].

Recently ambulatory pH monitors have shown a decrease in intraoesophageal pH to <4.0 at the time of typical chest pain[20] , which suggests that acid reflux can cause unexplained chest path. One study has shown that 12% of these pain events are associated with abnormal motility, 20% with reflux episodes and only 4% with both abnormal motility and reflux[26]. Thus standard oesophageal tests do not reliably predict the cause of chest pain and therefore the exact role of these tests in diagnosing non- cardiac chest pain remains unclear.

Psychiatric abnormalities may play a role in oesophageal motility disorder[27]. New studies using sophisticated psychometric instruments have detected psychiatric disorders like somatization disorder, anxiety and depression[28], while others have denied these[13] Therefore it has to be sorted out whether these patients have histories of high level of reinforcement for illness behaviour and if psychiatric abnormalities cause chest pain, or vice versa, as it has been found with duodenal ulcer studies[29]. Some of the features of painful oesophageal motility disorders mimic irritable bowel syndrome because it occurs predominantly in women and persons with low pain threshold. Detailed history has shown that 56% of patients with noncardiac chest pain have symptoms compatible with irritable bowel syndrome[30]. High frequency of lower gastrointestinal symptoms has been observed in patients with oesophageal motility disorders and vice versa[31]. Probably it is the severity of symptoms which dictates the primary focus of attention and that is why some patients complain of oesophageal symptoms while others of large bowel.

Management of oesophageal chest pain is difficult due to uncertainties about its specific diagnosis, intermittent nature of the symptoms, therapies that have serious side effects and the evolving concept that many patients improve spontaneouslywithout any treatment. However, before starting treatment, cardiac disease should be excluded first, followed by exclusion of musculoskeletal, peptic ulcer and biliary disease. Non-cardiac oesophageal chest pain due to gastro-oeso
phageal reflux unusually responds well to antirefluxing drugs alongwith H2 receptor antagonists. Patients with painful oesophageal motility disorder are usually difficult to treat; they usually respond well to nitrates[32], anticholenergic[33] psychotropic drugs[34] and calcium-channel-blocking agents[35-37]. In non responding cases oesophageal dilatation, pneumatic dilatation and oesophagotom[38] maybe considered but the latter should be reserved for patients with dysphagia associated with chest pain, achalasia and those with compromised life style who fail to respond to conservative measures. Finally confident reassurance is essential to create a better acceptance of symptoms and to assure that the symptoms are of non cardiac origin.

REFERENCES

1. Ockene, 1.S., Shay, M.J, Alpert, J.S., Weiner, B.H. and Dalen,J.E. Unexplained chest pain in patients with normal coronary arteriograms; a follow-up study of functional status. N. EngI. 3. Med., 1980; 303:1249.
2. DeMeester,T.R., O'Sullivan, G.C., Bermudez, 0., Midell, A.L, Cimochowski, G.E. and O'Drobinak, J.O. Esophageal function in patients with angina-type chest pain and nonnalcoronaryangiograms. Ann. Surg., 1982; 196:488.
3. Kline, M., Chesne, It., Sturdevant, R.LA. and McCallum, R.W. Esophageal disease inpatientswith angina.like chest pain. Am. J. Gastroenterol., 1981; 75:116.
4. Davies, H.A., Jones, D.B., and Rhodes, J. Esophageal angina as the cause of chest pain. JAMA., 1982; 248:2274.
5. Cannon, R.O. HI, Bonow, R.O., Bacharach, S.L, Green, M.V., Rosing, D.R, Leon, M.B., Watson, RM. and Epstein, S.E. Left ventricular dysfunction in patients with angina pectoris, normal epicardial coronary arteries, and abnormal vasodilator reserve. Circulation, 1985; 71:218.
6. Schofield, P.M., Bennet, D.H., Whonveli, PJ., Brooks, N.H., Bray, C.L., Ward, C. and Jones, P.R Exertional gastro- oesophageal reflux; a mechanism for symptoms in patients with angina pectoris and normal coronary angiograms. Br. Med.J. (Clin. Res.), 1987; 294:1459.
7. Davies, H.A., Jones, D.B., Rhodes, J. and Newcombe, R.J. Angina- like esophageal pain: differentiation from cardiac pain byhistory. 3. Clin. GastroenteroL, 1985; 7:477.
8. Svensson, 0., Stenport, G., Tibbling, L. and Wranne, B. Oesophageal function and coronary angiogram in patients with disablingchestpain.ActaMed.Scand., 1978; 204:173.
9. Ducrotte, P.11., Berland, M.J, Denis, P.11., et al. Coronary sinus lactate estimation and esophageal motor anomalies in angina with normal coronaiyangiograms. Dig. Dis.Sci., 1985; 29:305.
10. Cattau, E.L, Hirzel, it, Benjamin, S.B. and Cannon, R.O. Esophageal motility disorders in patients with abnormalities of coronary flow reserve and atypical chest pain [Abstract]. Gastroenterology, 1987; 92: 1339.
11. Mellow, M.H., Simpson, A.G., Watt, L., Schoolmeester, L and Hays, O.L. Esophageal acid perfusion in coronary artery disease. Induction of myGcardial ischemia. Gas. troenterology, 1983; 85:306.
12. Davies, 11.A., Page, Z., Rush, E.M., Brown, A.L, Lewis, M.J and Patch, M.C. Oesophageal stimulation lowers exertional angina threshold. Lancet, 1985; 1:1011.
13. Richler, J:E, Bradley, L.A. and Castell, D.O. Esophageal chest pain: Current controversies in pathogenesis, diag nosis, and therapy. Ann. Intern. Med., 1989; 110:66.
14. Katz, P.O., Dalton, GB., Richter, J.E., Wu, W.C. and Castell, D.O. Esophageal testing of patients with noncardiac chest pain or dysphagia. Results of three years' experience with 1161 patients. Ann. Intern. Med., 1987; 106:593.
15. (louse, RE and Staiano, A. Contraction abnormalities of the esophageal body in patients referred for manometry; a nay approach to manometric classification. Dig. Dis. Sci., 1983; 28:784.
16. Herrington, J.P., Burns, T.W. and Balart, L.A. Chest pain and dysphagia in patients with prolonged peristaltic contractile duration of the esophagus. Dig. Dis. Sci., 1984; 29: 134.
17. Orr,W.C. and Robinson, M.G. Hypertensive peristalsis in the pathogenesis of chest pain: further exploration of the nutcracker esophagus. Am. J. Gastroenterol., 1982; 77: 604.
18. Brand, D.L, Martin, D. and Pope, C.E II. Esophageal manometrics in patients with angina-like chest pain. Am. J. Dig. Dis., 1977; 22:300.
19. Benjamin, S.B., Richter, J.E, Cordova, C.M., Knuff, T.E. and Castell, D.O. Prospective manometricevaluationwith pharmacologic provocation of patients with suspected esophageal motility dysfunction. Gastroenterology, 1983; 84 (5 pt.1) :893.
20. deCaestecker,J.S., Blackwell,J.N., Brown,J. and Heading, RC. The esophagus as a cause of

recurrent chest pain; which patients should be investigated and which tests should be used? Lancet, 1985; 2:1143.
21. Janssens, J., Vantrappen, 0. and Ghillebert, 0. 24-hour recording of esophageal pressure and pH in patients with noncardiac chest pain. Gastroenterology, 1986; 90:1978.
22. Richter,J.E., Hackshaw, B.T., Wu, W.C. and Castell, D.O. Edrophonium; a useful provocative test for esophageal chestpain. Ann. Intern. Med., 1985; 103:14.
23. Lee, C.A, Reynolds, J.G, Ouyang, A, Baker, L. and Cohen, S. Esophageal chest pain. Value of high-dose provocative testingwith edrophonium chloride in patients with normal esophageal manometries. Dig. Dis. Sci., 1987; 32:682.
24. Nostrant, T.T., Saves, J. and Haber, T. Bethanechol increases the diagnostic yield in patients with esophageal chest pain. Gastroenterology, 1986; 91: 1141.
25. Ritchie, J. Pain from distention of the pelvic colon by inflating a balloon in the irritable colon syndrome. Gut, 1973; 14:125.
26. Peters, U., Maas, L.C., Petty, D., et al. Spontaneous non-cardiac chest pain: evaluation by 24 hour ambulatoiy esophageal motility and pH monitoring. Gastroentemlogy, 1988; 94:878.
27. (louse, RE and Lustman, P.J. Psychiatric illness and contraction abnormalities of the esophagus. N. EngI. J. Med., 1983; 309:1337.
28. Anderson, K.O., Dalton, C.B., Bradley, LA. and Richter, J.E. Stress; a modulatorof esophageal pressures in healthy volunteers and non-cardiac chest pain patients. Dig. Dis. Sd., (In press).
29. Feldman, M., Walker, P., Green, J.L and Weingarden, K. Life events, stress and psychosocial factors in men with peptic ulcer disease. A multidimensional case-controlled study. Gastroenterology, 1986; 91: 1370.
30. McMahan, T.P. and Richter, J.E Non-cardiac chest pain (NCCP) and irritable bowel syndrome (IBS): part of a continuum? (Abstract). Gastroenterology, 1986; 90:1546A.
31. Clouse, RE. and Eckert, T.C. Gastrointestinal symptoms of patients with esophageal contraction abnormalities. Dig. Dis. Sei., 1986; 31:236.
32. Kikendall, J.W. and Mellow, M.H. Effect of sublingual nitroglycerin and long-acting nitrate preparations on esophageal motility. Gastroenterology, 1980:79:703.
33. Blackwell,J.N., Dalton, C.B. and Castell, D.O. Oral pirenzepine does not affect esophageal pressures in man. Dig. Dis. Sci., 1986; 31:230.
34. Clouse, RE., Lustman, PJ., Eckert, T.C., Ferney, D.M. and Griffith, U.S. Low-dose trazodone for symptomatic patients with esophageal contraction abnormalities. A double-blind, placebo- controlled trial. Gastroenterology, 1987; 92: 1027.
35. Bortolotti, M. and Labo, G. Clinical and manometric effects of nifedipine in patientswith esophageal achalasia. Gastroenterology, 1981; 80:39.
36. Richter, i.E., Dalton, C.B., Buice, RG. and Castell, D.O. Nifedipine; a potent inhibitor of contractions in the body of the human esophagus: studies in healthyvolunteers and patients with the nutcracker esophagus. Gastroenterology, 1985; 89: 549.
37. Richter, i.E., Spurling, TJ., Cordova, GM. and Castell, D.O.Effectsoforalcalcium blocker,diltiazem,onesophageal contractions. Studies in volunteers and patients with nutcrackeresophagus. Dig. Dis. Sci., 1984; 29:649.
38. Horton, M.L and Goff, J.S. Surgical treatment of nutcracker esophagus. Dig. Dis. Sci., 1986; 31:878.