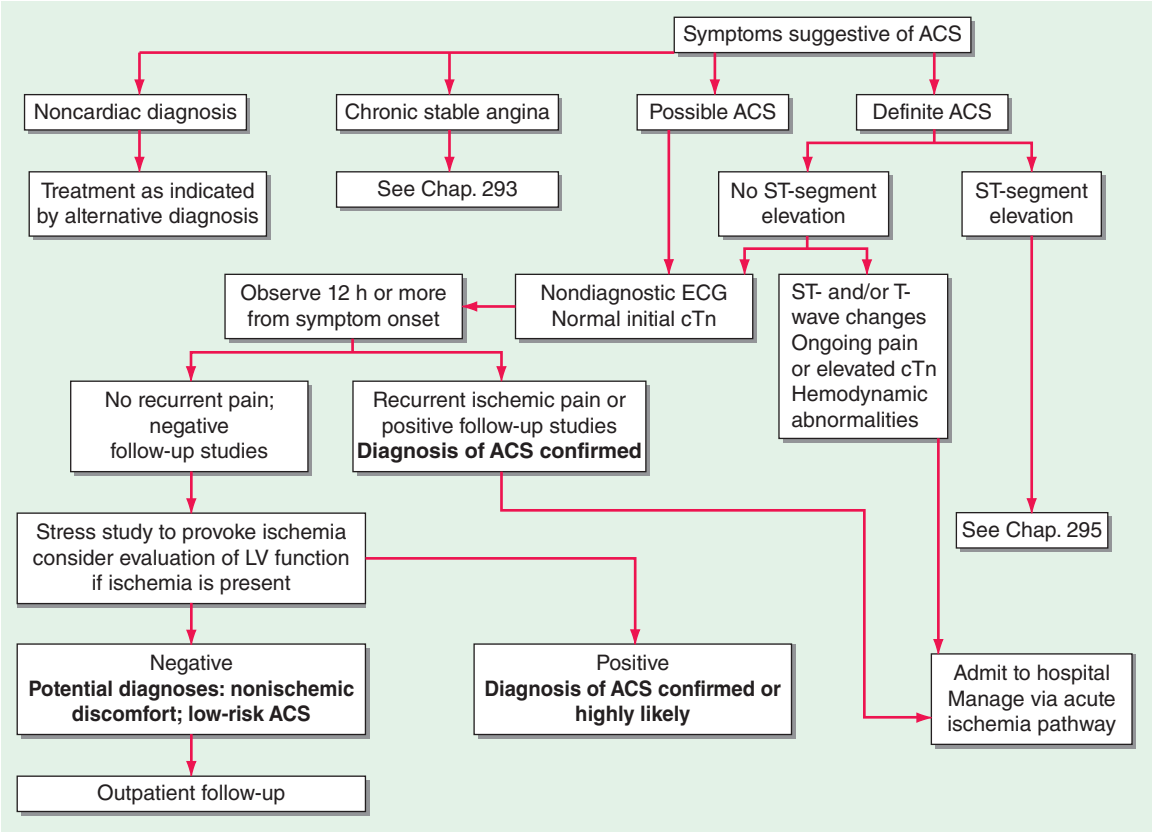

FIGURE 294-3 Algorithm for evaluation and management of patients with suspected acute coronary syndrome (ACS). Follow-up studies refer to ST deviations and elevation of troponin levels. cTn, cardiac troponin; ECG, electrocardiogram; LV, left ventricular. *(Modified from JL Anderson et al: J Am Coll Cardiol 61:e179, 2013.)*

(2) detect rest ischemia (using serial or continuous ECGs); and (3) detect significant coronary obstruction at rest with CCTA and myocardial ischemia using stress testing **(Chap 270e)**.

Patients with a low likelihood of ischemia are usually managed with an emergency department–based critical pathway (which, in some institutions, is carried out in a "chest pain unit") **(Fig. 294-3)**. Evaluation of such patients includes clinical monitoring for recurrent ischemic discomfort and continuous monitoring of ECGs and cardiac markers, typically obtained at baseline and at 4–6 h and 12 h after presentation. If new elevations in cardiac markers or ST-T-wave changes on the ECG are noted, the patient should be admitted to the hospital. Patients who remain pain free with negative markers may proceed to stress testing to determine the presence of ischemia or CCTA to detect coronary luminal obstruction (Fig. 294-2).

RISK STRATIFICATION

Patients with documented NSTE-ACS exhibit a wide spectrum of early (30 days) risk of death, ranging from 1 to 10%, and a recurrent ACS rate of 5 to 15% during the first year. Assessment of risk can be accomplished by clinical risk scoring systems such as that developed from the Thrombolysis in Myocardial Infarction (TIMI) Trials, which includes seven independent risk factors **(Fig. 294-4*A*)**. The presence of an abnormally elevated cTn is especially important, as is its peak level, which correlates with the extent of myocardial damage **(Fig. 294-4*B*)**. Other risk factors include diabetes mellitus, left ventricular dysfunction, renal dysfunction, and elevated levels of B-type natriuretic peptides and C-reactive protein. Multimarker strategies are now gaining favor, both to define more fully the pathophysiologic mechanisms underlying a given patient's presentation and to stratify the patient's risk further. Patients with ACS without elevated levels of cTn (infrequently encountered with the new sensitive troponin assays) are considered to have UA and have a more favorable prognosis than those with cTn elevations (NSTEMI).

Early risk assessment is useful both in predicting the risk of recurrent cardiac events and in identifying patients who would derive the greatest benefit from an early invasive strategy. For example, in the TACTICS-TIMI 18 Trial, an early invasive strategy conferred a 40% reduction in recurrent cardiac events in patients with an elevated cTn level, whereas no benefit was observed in those without detectable troponin.

TREATMENT NON-ST-SEGMENT ELEVATION ACUTE CORONARY SYNDROME (NON-ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION AND UNSTABLE ANGINA)

MEDICAL TREATMENT

Patients should be placed at bed rest with continuous ECG monitoring for ST-segment deviation and cardiac arrhythmias. Ambulation is permitted if the patient shows no recurrence of ischemia (symptoms or ECG changes) and does not develop an elevation of a biomarker of necrosis for 12–24 h. Medical therapy involves simultaneous anti-ischemic and antithrombotic treatments and consideration of coronary revascularization.

ANTI-ISCHEMIC TREATMENT (TABLE 294-1)

To provide relief and prevention of recurrence of chest pain, initial treatment should include bed rest, nitrates, beta adrenergic blockers, and inhaled oxygen in the presence of hypoxemia.

Nitrates These should first be given sublingually or by buccal spray (0.3–0.6 mg) if the patient is experiencing ischemic pain. If pain persists after three doses given 5 min apart, intravenous nitroglycerin (5–10 μg/min using nonabsorbing tubing) is recommended. The rate of the infusion may be increased by 10 μg/min every 3–5 min until symptoms are relieved, systolic arterial pressure falls to