BY CHARLES BRUEN, MD

The number of chest pain presentations you see in a week or a month can be overwhelming. They crowd your emergency department and claim scarce inpatient beds. But your ED can achieve relief as several advancements converge. Better risk stratification models can identify low-risk ED patients, and improved troponin assays allow us to rule out myocardial infarction.

You can safely and quickly discharge patients when the ED has accelerated diagnostic protocols in place, too, which means that all that is left is updating your low-risk chest pain protocol.

Risk models designed to predict adverse outcomes in chest pain patients are not new; many have been developed over the decades. They usually incorporate features of the patient’s presentation, medical history, ECG, and initial troponin. TIMI is the most widely known and used, but was originally developed to predict in-hospital mortality and has poor predictive capability.

Newer models developed specifically for ED patients include the HEART score, Manchester ACS (MACS), the Vancouver Chest Pain Rule, and the Emergency Department Assessment of Chest Pain (EDAP) study. Dr. Backus in the Netherlands developed a two-hour protocol. Independently, Simon A. Mahler, MD, at Wake Forest validated a pathway that used the HEART score (developed by Dr. Backus in the Netherlands) and a three-hour delta troponin measurement for early discharge of chest pain patients. This has been implemented with great success at the University of Maryland along with a risk visualization tool and shared decision-making.

Better risk stratification models can identify low-risk ED patients, and improved troponin assays rule out myocardial infarction

Score (ED-ACS). Physicians developed the risk scores using patients just like you see every day in your ED — the undifferentiated patient with chest pain. Patients scored as low risk with these models have 30-day adverse outcomes of less than two percent, and the HEART score and ED-ACS have been validated in follow-up studies.

Measuring Troponin

Our troponin assays are also getting better. No high-sensitivity troponin test is approved for use in the United States, but our contemporary assays have continually improved. Sensitive contemporary assays are available with lower detection limits (though not as good as high-sensitivity assays) and improved precision. Detecting smaller amounts of troponin allows for earlier detection of increased precision, which the assay manufacturers refer to as coefficient of variance, that is most important for rapid chest pain assessment in the ED. (Read more about troponins at http://emn.online/Aug15SponCirc.)

Troponin level was detectable only above the 99 percent upper limit of normal (ULN) in older assays. This fit with the definition of myocardial infarction, which is a rise or fall in cardiac troponin with at least one value above this 99 percent level. Troponins, with these more sensitive assays, may be reported above the level of detection (the absolute minimum amount of troponin detectable with the assay) but lower than the 99 percent level required for an MI.

Within this range, a delta troponin that is less than 99 percent ULN or a change of 20 percent or 9 ng/mL from the initial level indicates a myocardial infarction. A negative troponin with these assays imply a much lower troponin level than with the older assays and reduced risk.

Modern Protocols

Using these new tools, researchers developed accelerated diagnostic protocols for rapid disposition of chest pain patients. A group of Australian researchers developed a two-hour rule-out protocol studied with a large cohort of patients using the TIMI risk score and high-sensitivity troponins published in a series of studies such as RATPAC and ASPECT. Using a subset of the same patients along with new subjects, the authors’ ADAPT study substituted a contemporary troponin, which showed low-adverse events in low-risk patients with a two-hour delta troponin that was negative. (Detailed discussion at http://emn.online/Aug15SponCirc.)

This is being incorporated across Queensland, Australia, with the ACRE project. The same group of researchers also developed the ED-ACS risk score and validated it using a similar two-hour protocol. Independently, Simon A. Mahler, MD, at Wake Forest validated a pathway that used the HEART score (developed by Dr. Backus in the Netherlands) and a three-hour delta troponin measurement for early discharge of chest pain patients. This has been implemented with great success at the University of Maryland along with a risk visualization tool and shared decision-making.

How can you adapt these protocols to your emergency department? After the history and physical, ECG, and initial troponin, risk-stratify your patient as low, moderate, or high risk. Risk models such as TIMI, the HEART score, ED-ACS, or the Vancouver Chest Pain Rule can assist the physician’s judgment. If the outcome is low risk, discuss with the patient the concerning features of history and exam, evaluation results, risk stratification, and assessment of 30-day risk of major adverse cardiac events.

You should offer the patient hospital observation for further evaluation (including a realistic expectation of duration), the opportunity to remain in the emergency department for a second troponin measurement in two to three hours, or no further testing and follow-up. Visual aids are useful in these
Symptoms: Knee Pain, Shortness of Breath, Toothache

BY WHITNEY BARRETT, MD

A 59-year-old woman with a history of chronic alcohol abuse and no other past medical history presented with knee pain and shortness of breath. She reported that her symptoms had grown worse over the previous few days. She also mentioned having a dental infection for a few weeks.

Her oxygen saturation was 85% on room air, she was tachycardic to the 120s, and febrile to 38.2°C. She had diminished breath sounds in bilateral bases and a right knee that was warm with a moderate effusion but no erythema. The left side of her neck had focal swelling with fluctuance, but it was not appreciably tender or erythematous. She had normal range of motion of her neck and poor dentition but no tongue elevation. Her posterior oropharynx was normal.

What is on your differential given this constellation of symptoms and exam findings?

Find the diagnosis and case discussion on p. 27.

Protocol

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In Brief

ED to Divert Drunk Patients

The administration at Kent and Canterbury Hospital in the United Kingdom is instructing its staff to send drunk patients to a hospital 17 miles away, according to the Daily Mail. (http://dailym.ai/20tpOtTn.)

The department treats patients with acute medical illnesses, such as those who have suffered heart attacks and stroke and those with minor injuries including fractures and sprains.

A spokesman for the hospital said he considered the move part of bigger plans to divert patients with nonemergent complaints. He emphasized, however, that all patients will still be assessed.

“We never turn patients away and all patients who come to us for treatment will continue to receive the care they need as they always have done. The changes we are making will ensure patients receive the right care in the right setting,” he told the Daily Mail.

Nuffield Trust health care think tank representatives reported the number of people attending an emergency or urgent care center with probable alcohol poisoning doubled over the past six years, and more than one million patients are admitted for alcohol-related illness and injury every year.

Antidepressant Use Linked to Microbleeds

Antidepressant use was associated with a higher incidence of cerebral microbleed than nonuse, according to a study published in Stroke. (2016;47[1]:251.)

When stratified by affinity for the serotonin transporter, the authors found intermediate serotonin affinity antidepressant use was associated with an increased risk of developing microbleeds (odds ratio, 3.07; 95% confidence interval, 1.53-6.17).

The authors studied 2,559 participants 45 years and older of the population-based Rotterdam Study, who were all without microbleeds at baseline and who had repeated brain magnetic resonance imaging between 2005 and 2013 to determine microbleed incidence. They assessed antidepressant use between baseline and follow-up scan. Additional analysis concluded antidepressants were classified as low, intermediate, or high affinity for the serotonin transporter, and alternatively as selective serotonin reuptake inhibitors or non-selective serotonin reuptake inhibitors.

“Our results may support findings from previous clinical studies about increased intracranial and extracranial bleeding risk in antidepressant users,” the authors said.

Dr. Bruen is a cardiac intensivist and emergency physician at Regions Hospital in St. Paul, MN, and an assistant professor at the University of Minnesota with a special interest in emergency cardiovascular care. He completed a combined emergency medicine/internal medicine residency and fellowships in critical care and emergency cardiology at Hennepin County Medical Center. Visit his website, http://resusreview.com, follow him @resusreview, and read his past columns at http://emn.online/SponCircEMN.