RÉSUMÉ : Chaque année, 100 000 Canadiens sont hospitalisés pour des syndromes coronariens aigus (SCA) (infarctus aigu du myocarde et angine instable); un aussi grand nombre de patients sont hospitalisés pour que soit finalement «écarté» le diagnostic de SCA. Le diagnostic de SCA doit être rapide et exact afin de réduire le taux de mortalité et de prévenir la progression de l’angine instable vers un infarctus du myocarde. En même temps, on doit limiter les coûts inutiles liés au traitement de ces patients. Malheureusement, aucune épreuve ou stratégie particulières ne permettent d’identifier de façon définitive tous les patients atteints de SCA. Les unités de douleur thoracique à l’urgence, de plus en plus populaires, permettent de réduire le nombre d’hospitalisations aux unités de soins critiques en appliquant des protocoles diagnostiques intensifs au département d’urgence. Mais ces unités diminuent-elles les coûts ou ne font-elles qu’augmenter la proportion de patients soumis à des épreuves? Plutôt que de soumettre tous les patients au même processus diagnostique, les urgentologues devraient classer les patients selon leur risque parmi l’une des trois catégories suivantes : ceux dont la probabilité de SCA est faible qui nécessitent un minimum d’épreuves à l’urgence; ceux qui présentent des signes évidents de SCA et qui doivent être hospitalisés; et ceux dont la probabilité de SCA est intermédiaire et qui doivent subir différentes épreuves.

Acute coronary syndromes: We must improve diagnostic efficiency in the emergency department

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Introduction

Almost every shift, emergency physicians agonize over patients who present with chest pain. Every year approximately 100 000 patients are hospitalized with acute myocardial infarction (AMI) or unstable angina (acute coronary syndromes [ACS]). Canadian registry data (FASTRAK II) suggest that at least another 100 000 patients are admitted with chest pain and subsequently have ACS ruled out. ACS are perhaps the most controversial issue in emergency medicine.

What are acute coronary syndromes?

ACS include ST-elevation AMI, non-ST-elevation AMI and unstable angina. These 3 diagnoses share similar pathophysiology and have many aspects of treatment in common. The culprit lesion is usually a ruptured atherosclerotic plaque in a coronary artery. The resultant exposed collagen, now unprotected by intact endothelium, is highly thrombogenic. A monolayer of platelets adheres to the open surface, and nearby platelets are activated to aggregate with each other and form a platelet plug. Soluble fibrinogen is rapidly converted to fibrin in a mesh that solidifies the platelet plug into a clot. Clots are dynamic and in constant flux between mechanisms that accelerate the clotting process and mechanisms that facilitate clot breakdown.

Clot causes obstruction to flow with resultant ischemia. The ischemia can cause transmural necrosis (ST-elevation AMI), partial necrosis (non-ST-elevation AMI) or no necrosis (unstable angina) depending on the duration and degree of occlusion, the amount of myocardium served by the blocked vessel and the extent of collaterals. Physicians should think of ACS as a ruptured plaque with an active clot occluding or threatening to occlude a coronary artery.

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Why is it important to make the diagnosis of acute coronary syndromes in the emergency department?

Significant short-term mortality exists in all ACS. Generally this mortality is highest for ST-elevation AMI, followed by non-ST-elevation AMI, followed by unstable angina. Unstable angina can progress to non-ST-elevation AMI, which can progress to ST-elevation AMI. Progression to a higher risk group is potentially avoidable; therefore, preventing progression is a primary goal of management. Mortality in each group can be reduced by early appropriate therapy. Although the benefit of rapid treatment is most clearly shown for ST-elevation AMI, it is also logical that early recognition and treatment of unstable angina and non-ST-elevation AMI will limit myocardial necrosis and improve outcomes.

Conversely, although it is important to make a rapid, accurate diagnosis of patients with ACS, every patient without serious illness who is admitted or subjected to a costly diagnostic work-up consumes health care resources that are then unavailable for other important health care needs. Diagnostic efficiency and cost-effectiveness are important. Therefore, in a perfect world, all patients with ACS would be recognized and treated appropriately in the ED, and those without ACS would be sent home or treated for the cause of their symptoms.

Why is it so hard to make a definitive diagnosis?

There are no tests that alert us to plaque disruption and early clot formation. As a result, no single ED test or combination of tests is sensitive for ACS. Clinical estimates of disease probability are driven mostly by the patient’s history and presenting electrocardiogram (ECG); however, atypical symptoms are relatively common, and patients without risk factors still suffer ACS. Electrocardiograms are helpful when positive, but in up to 50% of patients with ACS the results are normal or non-specific. Serum markers don’t rise until 4 to 6 hours after the onset of myocardial necrosis, and they remain normal in patients with unstable angina who have an unstable plaque and are in imminent danger of sudden coronary artery occlusion. Other investigations, including sestamibi nuclide scanning and stress tests, show promise but are frequently unavailable in the ED.

How do we currently decide whether to admit or discharge?

A patient with chest pain is seen by the emergency physician, who takes a history, orders an ECG and decides, based on his or her accumulated wisdom, hospital pattern of practice, and personality traits, whether the patient should be admitted and, if so, what therapies to initiate. As physicians gain experience they become more sensitive and less specific, discharging fewer patients with AMI but admitting more without ACS. This pattern is understandable because the physician’s primary concern for patient safety drives him or her to use a threshold that is maximally sensitive. However, admission practices vary widely, and in a large Canada/US study of serial cardiac markers, admission rates for patients with chest pain ranged from 49% to 87%. It is likely that individual physician disposition decisions are also extremely variable even within a single institution.

What are chest pain units and why are they so popular in the US?

ACS are lethal, difficult to diagnose and often require observation and serial testing over time. Chest pain units are therefore a logical solution. Many US hospitals have already established such units to address diagnostic uncertainty, and many more units are currently under development. Hospitals and physicians want to limit the risk associated with inappropriate discharges, and patients want to have confidence that the hospital they attend is providing the “best” care.

Chest pain units are designated areas, often within the ED, that have 2 main functions. First, patients who need urgent stabilization or initiation of anti-ischemic therapy are rapidly recognized and treated before being transferred to a critical care unit or catheterization laboratory. Second, patients with undiagnosed or obscure chest pain of possible cardiac origin are held in chest pain units, where they typically undergo an expensive, protocol-driven battery of tests to rule out ACS. Diagnostic protocols, which can take anywhere from 9 to 24 hours to complete, prevent some critical care unit admissions but extend ED lengths of stay.

Diagnostic protocols in chest pain units include combinations of: clinical observation, frequent serum markers, continuous ST-segment monitoring or serial ECGs, echocardiography, nuclide scanning and stress tests. Despite expensive work-ups there is little good evidence that the application of these protocols improves patient outcomes.

Let’s not forget about clinical judgement

Chest pain units help us focus on more efficient methods of ruling out ACS. Unfortunately, they will encourage work-
ups on more patients — even those with very low clinical likelihood of disease. And although chest pain units are cost-effective compared with admitting patients to a critical care unit for 1 to 2 days, increased numbers of patients subjected to intensive investigation could actually increase the overall diagnostic costs for all patients presenting with chest pain.

We should not forget that clinical judgement is a powerful tool. Many experienced emergency physicians rapidly identify low-risk patients from their history, ECG and brief observation. In a large study of diagnostic serum markers in patients with chest pain, the ED discharge rate by centre was as high as 51%. Of patients discharged after a limited ED evaluation, fewer than 1% experienced ischemic complications within a week and no deaths occurred due to unrecognized ischemia — strong evidence that physicians can be reasonably specific with their initial assessment. Unfortunately, we don’t understand the process that good physicians use in making these assessments, nor has this decision process been scientifically evaluated.

Defining three groups of patients with chest pain

Patients who present with chest pain (or symptoms consistent with ACS) can be placed in 1 of 3 categories. Each category requires a different diagnostic and therapeutic approach, and each has an urgent research agenda.

Patients in the first (high-risk) group have objective signs of myocardial ischemia, including ST-elevation AMI, ST-depression or T-wave inversion signifying unstable angina or non-Q-wave AMI, or elevated serum markers signifying myocardial necrosis (including micro-infarcts or high-risk unstable angina). For this group, ongoing research is aimed at optimal reperfusion strategies, fibrinolytic agents, antiplatelet therapies, thrombin inhibitors and strategies to shorten the time to reperfusion in appropriate patients.

Patients in the second (moderate-risk) group have no objective signs of ischemia, but have a clinical presentation compatible with ACS. These patients should enter a rapid diagnostic track in an actual or “virtual” chest pain unit to determine whether an acute coronary syndrome is the cause of their symptoms. The most efficient and effective diagnostic protocol has yet to be defined and will require further research.

Patients in the third (low-risk) group can be discharged from the ED after a brief assessment and observation period. These patients should not undergo expensive diagnostic tests. The difficult challenge we face is identifying which patients fall into this category.

A clinical prediction rule for low-risk patients?

Our time-honoured diagnostic approach is to identify patients with disease and let the others fall out. This is particularly difficult when there are no sensitive tests to determine who has disease. Therefore, a promising approach is to reverse our logic and identify patients without disease, much like we use the Ottawa Ankle Rules to identify patients who do not require an x-ray. An ACS prediction rule that would identify low-risk patients based on common clinical criteria would be of great value in helping emergency physicians determine which patients are safe to discharge.

Clinical prediction rules have been developed and tested in emergency medicine in a more rigorous manner than any other specialty. The Ottawa Ankle Rules tell us which extremity injuries can be managed without radiography, and similar methodology is now being used to help us make more complicated decisions, such as which patients need computed tomography after “minor” head trauma. A practical rule to identify patients with chest pain who can safely be discharged early would have enormous implications. Such a rule would identify a significant portion of the more than 100 000 patients admitted each year for negative diagnostic work-up. The daily direct nursing cost of a Canadian critical care unit bed is approximately $665 per day, excluding tests, consultations and institutional overhead (costing database, St. Paul’s Hospital, Vancouver, 1998 data). If only 25% of “low-risk” patients could be saved 1 day’s stay in hospital, the savings in nursing care alone would approximate $16 625 000 per year, and the true cost savings are likely to be far greater.

Conclusions

The complicated and difficult diagnostic issues that surround patients who present to the ED with possible ACS can and must be simplified. As emergency physicians we need to objectify our early decisions in patients with chest pain. A clinical tool should be developed to answer the question, Who can be safely discharged after a brief early assessment?

Patients at low risk should be identified as safe for discharge and should not undergo intensive investigation. Patients at moderate risk should undergo a rapid and inexpensive diagnostic protocol. At any time in this diagnostic process, objective evidence of ischemia must be rapidly recognized and urgently treated if we are to provide the best and most cost-effective care to patients presenting with acute coronary syndromes. We must commit ourselves to understanding how we diagnose and dispose of ED patients with ACS. Determining how to do it better is our challenge.
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References

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