THINKING ON YOUR FEET

Door-to-door-to-door

Interhospital transfer for urgent percutaneous coronary intervention

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A family physician practising in Perth County is just about to begin a Rourke Baby Record when a call comes in from the front desk of the clinic: a patient complaining of chest pain is on his way to the emergency department. Apologizing gently, the physician cuts the appointment short and jogs across the street to the hospital.

Arriving at the ambulance bay is a 59-year-old man who is diaphoretic, cyanosed and in clear distress. According to the man's wife, the two had been preparing to go for a walk when, about 15 minutes ago, he suddenly doubled over in pain, clutching his chest. The accompanying primary care paramedic helpfully clarifies that the chest pain is retrosternal, pressure-like in nature and radiating down the left arm beyond the elbow. Vitals obtained during transit reveal an elevated heart rate of 85 and mild hypotension (115/75). To nobody's surprise, the automated 12-lead ECG interpretation reads "Acute Inferior STEMI" (ST-elevation myocardial infarction).

INITIAL MANAGEMENT OF ACUTE CORONARY SYNDROME

Acute coronary syndrome (ACS) is a disease of coronary vessel obstruction, primarily due to the formation of clots secondary to atherosclerotic plaque rupture. Myocardial infarction (MI) refers to situations where cardiac tissue sustains irreversible damage, which can happen with as little as 15 minutes of ischemia.¹ As such, prompt assessment and management are key when ACS is strongly suspected. After airway, breathing and circulation have been stabilised, a cardiac-focused history and physical examination should be obtained and 12-lead ECG taken. Blood should also be drawn for evaluation of cardiac biomarkers, coagulation, glucose, lipid profile and standard chemistry.² Aspirin is indicated and to be continued indefinitely primarily for its antiplatelet properties, which have been exhaustively shown to reduce the incidence of negative outcomes in patients with all manner of cardiovascular disorders.³ Nitroglycerin is indicated for vasodilation of coronary and systemic vessels to improve blood flow to ischemic areas of the heart and reduce cardiac stress via decreasing preload and afterload. Since both these mechanisms may potentiate severe hypotension and hemodynamic decompensation, pre-existing hypotension, bradycardia and recent phosphodiesterase inhibitor use (as for erectile dysfunction) are important contraindications to nitroglycerin use.⁴ Morphine is used in this setting for its analgesic and anxiolytic effects,² but it must be carefully titrated because it causes systemic vasodilation and can worsen hypotension.⁵ Uniquely, inferior MIs may trigger the Bezold-Jarisch reflex, a phenomenon that also leads to vagal stimulation and hypotension. IV fluids or atropine can reverse this effect.⁶

Aggressive treatment and an immediate cardiology consult are necessary when ACS is complicated by cardiogenic shock, left heart failure, or sustained ventricular tachyarrhythmia. Relevant signs

include hypotension, tachycardia, confusion, cool, pale skin and pulmonary crackles.²

The physician, observing the ST-segment elevations in leads II, III and aVF characteristic for an inferior STEMI, quickly rules out relevant medical contraindications and orders STAT chewed aspirin and intravenous fluids. Sublingual nitroglycerin and intravenous morphine are ordered with the risk of hypotensive crisis in mind. Blood is drawn and immediately sent to the laboratory. An urgent consult to the internal medicine service at the nearest tertiary care centre is made due to concern about cardiogenic shock.

THROMBOLYSIS AND PERCUTANEOUS CORONARY INTERVENTION

Given the underlying pathology, there are two approaches to patients presenting with ACS: thrombolysis and percutaneous coronary intervention (PCI). Thrombolysis is induced via IV administration of fibrinolytic agents, and second-generation fibrinolytics like tenecteplase (TNK) preferentially activate clot-associated plasminogen to effect clot lysis while limiting the increase to systemic hypocoagulability. Such measures can restore coronary artery patency in 80 to 90% of cases overall, thereby reducing the risk of complications and death.⁷ Concomitant use of low-molecular weight heparins like enoxaparin decreases the risk of disease recurrence and improves vessel flow.⁸

PCI is an umbrella term that includes several non-surgical measures for restoring coronary blood flow. Generally, instruments are inserted through catheters in the inguinal femoral or radial artery and guided via angiography to sites of reduced perfusion. Balloon angioplasty compresses vessel obstructions to the sides of the lumen, while atherectomy mechanically disrupts the blockage for subsequent removal. Either technique may be augmented with the placement of an intracoronary stent that helps prevent the recurrence of stenosis.⁹

There is ample evidence that PCI is more effective than thrombolysis at reducing death, reinfarction and stroke,¹⁰ but this advantage is less certain where PCI is not available. The theoretical benefit of PCI over thrombolysis must be weighed against the delay in treatment incurred during transfer to a PCI-capable facility. Currently, the American College of Cardiology Foundation and American Heart Association recommend thrombolysis as a first-line treatment when it is anticipated that PCI cannot be performed earlier than 120 minutes after first medical contact (FMC). In addition, urgent transfer to a PCI-capable hospital for coronary angiography is recommended even if thrombolysis appears to be successful.¹¹

Many factors complicate the estimation of this timeframe in the setting of a rural hospital. In Huron County, for example, 6 ambulances cover an area of 3400 km² while in Middlesex-London, 22

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units cover a similar area (and 8 of 13 ambulance stations are in the city of London).¹²⁻¹⁴ This illustrates the greater distances that must be covered between responding to an emergency call, making FMC and arriving at a local hospital and the fewer number of emergency response vehicles available to do so in a rural setting. In certain areas and times of the year, factors such as road accidents and inclement weather further increase the uncertainty of any estimation of FMC-to-PCI time.¹⁵

Returning to the case, the physician barks out an order for TNK and enoxaparin while waiting for the local cardiologist to pick up the phone. Several frustrating minutes of call routing later, a general internist on the other end of the line agrees that the patient would benefit from PCI, but gently rejects the request for transfer as the lone PCI suite was unavailable. Eventually, a transfer is secured to another hospital, 83 km away.

INTERHOSPITAL TRANSFER

Interhospital transfer of critically ill patients carries substantial risk—patients in ground and air ambulances can become unstable when jostled and are in a small space that is ill-equipped for medical manoeuvres and cut off from outside help. As such, it is usually advisable to ensure patients are hemodynamically and otherwise stable before departure.¹⁶ That said, stabilization efforts would be carried out at the expense of PCI timeliness, and thus, some guidelines for interhospital transfer from PCI-incapable hospitals do not recommend allowing patient instability to delay transport.¹⁷

Telemetry should transition seamlessly from the emergency room to the ambulance, if possible. Invasive blood pressure monitoring is additionally recommended except for hemodynamically stable patients with short predicted travel times. Where possible, electrical equipment should be battery-powered and IV fluids and medications delivered by syringe or infusion, rather than by gravity.¹⁸

Ideally, a pair of physicians trained in airway management, advanced cardiac life support (ACLS) and critical care should accompany an unstable patient during transport; realistically, this standard is seldom met.¹⁵ In rural southwestern Ontario, essentially all emergency department physicians are ACLS-trained, but formal emergency medicine training is rare. Moreover, limited staff numbers at these centres can make the formation of an ideal transport team impossible.^{19,20}

In the present case, the medical team manages to correct the earlier hypotension somewhat while the consults were made. The patient is quickly bundled back into the ambulance he had arrived in and the physician climbs into the back with him. The second emergency physician on call is contacted and, leaving some home renovations unfinished, joins the 2 nurses left to staff the emergency department. With that, the ambulance speeds off with lights and sirens active.

After half an hour of travel, the patient develops an atrioventricular (AV) block that rapidly progresses to ventricular tachycardia (VT) with cardiac arrest.

ARRHYTHMIC COMPLICATIONS OF STEMI

Electrolyte imbalances, hypoxia, myocardial damage and abnormal sympathetic discharge secondary to MI together contribute to a high risk of peri-infarction arrhythmias. Indeed, such events

occur in approximately 90% of all patients with myocardial infarction and are especially common in STEMI.²¹

Both first-degree and second-degree (Mobitz) type I AV blocks are potentiated by right coronary occlusion and these occur most commonly with inferior MI.²² Neither usually requires treatment if cardiac output remains adequate. Mobitz type II AV blocks, on the other hand, indicate a poor prognosis and should be treated immediately with transcutaneous pacing or atropine, if possible. Caution should be used with atropine, however, as an increase in heart rate can improve cardiac output but decompensate the heart.²¹

All types of sustained VT require urgent intervention because they severely decrease cardiac output, not infrequently to the point of cardiac arrest, and have a tendency to progress to life-threatening ventricular fibrillation. Monomorphic VT should be treated immediately with synchronized electrical cardioversion starting at 100 J, unless it is hemodynamically well-tolerated, in which case IV amiodarone may be tried. Polymorphic VT is less common during acute STEMI, but should it occur, it should be treated with immediate unsynchronized electrical defibrillation starting at 120 or 200 J.²³ Synchronized cardioversion can resolve VT with lower energy and therefore expose the heart to less electrical damage, but it is contraindicated in polymorphic VT because delivery of a charge during a T wave can provoke ventricular fibrillation.²⁴

As the patient monitor alarms go off, the driver parks the ambulance at the side of the highway and offers to help manage vitals. Meanwhile, the physician abandons a vial of atropine half-drawn up and reaches for the defibrillator instead. Though telemetry revealed a mostly monophasic pattern, defibrillation at 120 J was deemed safer and thus administered.

Post-shock, the patient alternates between normal sinus rhythm and Mobitz type II. The rhythm is of concern, but the patient seizes and bites his tongue, which begins to bleed freely and compromises his airway. A nasopharyngeal tube is successfully inserted despite the cramped setting and the physician, never forgetting the ABCs, switches to providing bag-valve-mask ventilation to preempt a drop in O₂ saturation. Without having to be told, the paramedic returns to the driver's seat and sets off. The remainder of the journey is mercifully uneventful.

The patient is received by a six-person team at the destination interventional cardiology suite, just over 180 minutes after the 911 call was made. Our intrepid physician catches a ride back to the clinic with the same ambulance and quietly prepares for the afternoon's appointments.

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