Clinical Case Files: Real Stories of Diagnosis, Treatment, and Recovery of Common Internal Disorders

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Introduction

Illness rarely announces itself with a tidy label. It arrives as a story—of fatigue that lingers, shortness of breath climbing the stairs, a strange flutter in the chest, a headache unlike any before. This book collects real-world clinical case files to show how those stories are translated into diagnoses and care plans. By following patients through evaluation, decision points, and recovery, we illuminate the practical reasoning that connects symptoms to causes and guides safe, coordinated treatment.

Each chapter begins with a clear, lay-language summary of the patient’s presentation and the key questions it raises. We then open the diagnostic toolbox: what the team considered first, what they deliberately ruled out, which tests were chosen and why, and how results were interpreted in context. Rather than memorizing lists, you will see thinking in motion—how a differential diagnosis narrows, how probabilities shift with each clue, and how clinicians balance urgency with caution when red flags appear.

Because internal disorders rarely respect the borders of a single specialty, the cases highlight multi-disciplinary care in action. Cardiologists, internists, endocrinologists, gastroenterologists, pulmonologists, neurologists, nurses, pharmacists, dietitians, physical and respiratory therapists, and social workers all make appearances. You will see how information flows among team members, how responsibilities are shared, and how the plan adapts as patients respond to therapy—or fail to. Care coordination is not a footnote; it is the backbone of safe outcomes.

Patients and families read the medical system as much as the medical record reads them. To support that partnership, every chapter calls out red flags that should trigger prompt medical attention, along with practical lessons for navigating similar situations: preparing for appointments, understanding test options, recognizing medication side effects, and planning follow-up. We also include moments when uncertainty remained and how the team and patient made decisions in the face of it—because ambiguity is common, and transparency builds trust.

These cases are drawn from real encounters and have been carefully de-identified; in some instances, details are blended to preserve privacy while preserving clinical truth. They are presented for education, not as medical advice for any one person. Health concerns are individual, and readers should use what follows to ask better questions, not to self-diagnose or delay care. When in doubt, seek timely, in-person evaluation.

Finally, a note on how to use this book. You can read straight through or select chapters by symptom or condition. Consider pausing at each decision point to ask, “What else could this be? Which data would most change my mind?” If you are a

student or trainee, try drafting your own differential and plan before reading further. If you are a patient or caregiver, focus on the summaries, red flags, and practical lessons at the end of each case. However you approach it, the goal is the same: to demystify clinical reasoning and to empower safer, more informed participation in care.

Medicine is a human enterprise. Behind every lab result is a person with goals, values, fears, and constraints. These pages aim to honor that reality. By foregrounding stories and the thoughtful work that connects them to science, we hope to make the path from first symptom to recovery a little clearer—and, when clarity isn’t possible, to make the journey more navigable and compassionate.

CHAPTER ONE: Acute Coronary Syndrome in a Middle-Aged Runner: When Chest Pain Isn't Indigestion

Marcus Ellison was forty-six years old, in the kind of shape that most primary care physicians would greet with a nod and a quick glance at the chart. He ran four days a week, logged between twenty-five and thirty miles, kept his weight at a respectable one hundred seventy-eight pounds on a six-foot frame, and had completed two marathons and a handful of half-marathons over the past decade. He ate what most sports medicine literature would approve of: lean proteins, whole grains, vegetables, the occasional post-run beer. He did not smoke. His blood pressure, last checked at a pharmacy kiosk, read 128 over 82, which he figured was close enough to normal. He had no family history of heart disease that he knew of, unless you counted a grandfather who had died at seventy-nine of a stroke. By every conventional metric, Marcus was the picture of health.

It started on a Tuesday morning in late October. The temperature had dropped sharply overnight, and a thin sheet of frost covered the running trails along the river near his home. Marcus pulled on a long-sleeve technical shirt, laced up his usual shoes, and headed out for what was supposed to be a moderate six-miler. He had a race on the calendar in six weeks and was trying to stay disciplined about his training plan. The first three miles felt fine. His legs were loose, his breathing was steady, and his Garmin watch was recording a pace that he considered respectable for a Tuesday effort.

Somewhere around mile three and a half, he felt it. Not pain exactly, but a sensation of pressure, a heaviness sitting right behind his sternum, as though someone had placed a firm palm flat against his chest. It was not sharp, and it did not radiate anywhere. He slowed to a walk, expecting it to pass the way a side stitch does when you ease off the pace. It did not pass. He stood on the side of the trail, resting his hands on his knees, and waited. The pressure remained, unchanged in character, unchanged in intensity. He tried taking a deep breath. That did not change it either.

Marcus debated for about ninety seconds whether to call someone. He lived alone. His phone was in the pocket of his shorts, and he stared at the screen as though the act of looking at it might resolve the question. He started walking back toward his car at a brisk pace, and the pressure did not worsen, but it did not improve. By the time he reached the parking lot, he was breathing harder than the walk warranted, and there was a thin film of sweat on his forehead despite the cold air.

He drove himself to the emergency department. In retrospect, he would later tell his

cardiologist that he almost had not gone. Every instinct he had, built from years of pushing through discomfort on long runs, told him to wait it out, to give it an hour, to see if it resolved on its own. What finally pushed him through the doors was not dramatic. It was a quiet, stubborn thought that he did not want to take the chance of being wrong.

The triage nurse asked the standard questions. On a scale of one to ten, how would he rate the pain? He said four. It was not really pain, he explained, more pressure. It was located centrally, did not move, and had lasted about fifteen minutes so far. The nurse checked his vitals. His blood pressure was 156 over 94, up from whatever baseline it had been that morning. His heart rate was 98. His oxygen saturation was 95 percent on room air. Those numbers were not catastrophic, but they were not reassuring either, especially in a man his age with chest discomfort that had been exertional in onset and was persistent at rest.

An electrocardiogram was done within twelve minutes of his arrival. The emergency medicine physician, Dr. Patel, reviewed it carefully. The tracing did not show the classic ST-segment elevation that most people associate with a heart attack. But there were subtle changes in the lateral leads, a slight depression of the ST segment and some T-wave flattening that, while not dramatic, were not normal either. Dr. Patel's differential diagnosis at that moment included several possibilities. The most concerning was acute coronary syndrome, which is a broad term encompassing unstable angina and the two main types of heart attack: one caused by a complete blockage of a coronary artery, known as ST-elevation myocardial infarction, and one caused by a partial blockage, known as non-ST-elevation myocardial infarction. The ECG findings were more consistent with the latter. But Dr. Patel also considered other causes of chest pain in a middle-aged runner: musculoskeletal strain from the cold weather, gastroesophageal reflux triggered by the pre-run coffee Marcus had consumed, and costochondritis, an inflammation of the cartilage connecting the ribs to the sternum.

What made the clinical picture lean toward a cardiac cause were several factors working together. The pain was exertional. It was central and pressing rather than sharp or pleuritic, which would have suggested a lung-related origin. It did not respond to rest in the way a simple muscle cramp would. And the ECG, while not diagnostic on its own, showed changes in a distribution consistent with ischemia affecting the lateral wall of the heart. Dr. Patel ordered serial troponin levels, blood tests that measure a protein released by damaged heart muscle cells, and she called the on-call cardiologist to discuss the case.

The cardiologist, Dr. Nguyen, arrived in the emergency department within the hour. She reviewed the ECG, the clinical history, and the initial troponin result, which was slightly elevated at 0.08 nanograms per milliliter. The normal range in that particular laboratory was below 0.04. The elevation was small, but in the context of chest

discomfort and suggestive ECG changes, it was significant. Dr. Nguyen made the decision to admit Marcus to the hospital and to proceed with coronary angiography, a procedure in which a thin catheter is threaded through an artery in the wrist or groin to the coronary arteries, where dye is injected so that blockages can be visualized directly.

The angiogram revealed a high-grade stenosis, a narrowing of approximately 85 percent, in the left anterior descending artery. In everyday language, this is the artery most people have heard of, sometimes called the "widow-maker" because of its critical role in supplying blood to a large portion of the heart muscle. The blockage was caused by a ruptured atherosclerotic plaque. Plaque is a buildup of cholesterol, inflammatory cells, calcium, and other substances within the wall of an artery. When a plaque ruptures, the body responds by forming a blood clot at the site, and that clot can suddenly and severely narrow or completely occlude the vessel.

Marcus's case illustrates a point that bears repeating: atherosclerosis is not simply a disease of the elderly, the sedentary, or the overweight. It is a process that begins quietly, often decades before symptoms appear, driven by a combination of genetic predisposition, metabolic factors, and environmental exposures. Marcus had one risk factor he did not know about. His LDL cholesterol, checked at a health fair two years earlier, had come back at 168 milligrams per deciliter, a number that his primary care provider at the time had called borderline and suggested he recheck with a fasting panel. Marcus had meant to schedule the fasting panel but never did. The post-race celebrations after his last marathon had featured plenty of beer and barbecue. Between work demands and family obligations, the follow-up appointment had simply slipped away. That single missed follow-up meant that a modifiable risk factor went unidentified and unmanaged for two critical years.

The interventional cardiologist, Dr. Kim, performed a percutaneous coronary intervention during the same procedure. A drug-eluting stent, a tiny mesh scaffold coated with medication to prevent re-narrowing, was deployed across the blockage, restoring blood flow to the affected portion of Marcus's heart muscle. The procedure took forty-five minutes. Marcus spent one night in the cardiac care unit and was moved to a regular floor the next morning.

His recovery followed a path that is familiar to many patients admitted with acute coronary syndrome but unfamiliar to Marcus, who had never before spent a night in a hospital. He worked with a physical therapist on graded movement, starting with short walks in the hallway. He met with a cardiac dietitian who reviewed his typical eating patterns and identified a few areas where modest changes could meaningfully reduce his cardiovascular risk. He had long conversations with a pharmacist about the medications he would now need to take, including a high-intensity statin to lower his LDL cholesterol, a low-dose aspirin to reduce the risk of further clot formation, a beta-blocker to decrease the heart's workload, and a medication to manage his newly

diagnosed mild hypertension.

The practical lessons from Marcus's case are worth dwelling on, not because his story is unusual, but because it is common. Acute coronary syndrome remains one of the leading causes of death worldwide, and it does not discriminate by fitness level. Several red flags from his presentation deserve emphasis. First, chest discomfort that occurs during exertion and persists at rest should be evaluated promptly, regardless of age or fitness level. The fact that Marcus was a runner did not protect him. Second, any new chest discomfort that is accompanied by shortness of breath, sweating, or nausea warrants immediate medical attention. Third, borderline lab results, such as an elevated LDL or a blood pressure reading in the high-normal range, deserve follow-up rather than dismissal. Fourth, a family history of premature cardiovascular disease, typically defined as a first-degree relative affected before age 55 in men or 65 in women, is a significant risk factor even if no other red flags are present.

For patients navigating similar situations, preparation before an emergency can make a meaningful difference. Knowing your own baseline numbers, such as blood pressure, cholesterol, and blood glucose, helps you and your care team interpret new symptoms in context. Keeping an updated medication list and a short summary of your medical history in your wallet or phone can save valuable time in urgent settings. And understanding that the emergency department prioritizes cases based on severity, which means that a patient with chest pain may or may not be seen instantly, can help manage expectations during an anxious wait.

Marcus returned to running four months after his procedure. He completed a 10-kilometer race the following spring, not at his previous pace, but with a chest that felt, in his words, completely clear. His cardiologist cleared him for the activity gradually, and his cardiac rehabilitation program provided the structured pathway back to full training that he needed. The experience changed him in ways that extended beyond his running. He became an advocate for regular health screenings in his running club, a small but meaningful act that, as he put it, might save someone else from the assumption that fitness equals invulnerability.

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