Integrated narrative and evidence based case report

Case report of paroxysmal atrial fibrillation and anticoagulation

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Evidence based medicine and narrative based medicine rarely meet, and they may conflict,[1 2] but in clinical care they are complementary.[3–5] Some attempts to integrate the two approaches have not considered the doctor's story, which is often critical to understanding a case.[6 7]

This paper tries to bridge gaps between a patient's and doctor's narratives and the evidence. It is the story of a patient in our practice (see box) who has produced written narratives about her illness and shared them with her family physician (SR). We use the narratives to decide on the best evidence based treatment for paroxysmal atrial fibrillation after corrected tetralogy of Fallot.

Search

We searched Medline between December 1999 and May 2000 with the keywords atrial fibrillation (paroxysmal), tetralogy of Fallot, atrial arrhythmia, anticoagulation, primary care, family medicine, general practice, and ambulatory medicine. We also searched www.goldenhour.co.il (an Israeli website that includes evidence based medicine).

We found no important evidence on the treatment of paroxysmal atrial fibrillation plus tetralogy of Fallot. Our discussion is based on the evidence available on paroxysmal atrial fibrillation alone.

Deliberations

Risk factors for thromboembolism in paroxysmal atrial fibrillation are cumulative.[8–13] In patients without risk factors who are not treated for thromboembolism, the annual risk of stroke is about 1%. For patients at low risk of thromboembolism, aspirin or no drug is recommended.[8 11–14] The presence of one risk factor puts patients at moderate risk of thromboembolism (2-5% annual risk of stroke), and the treatment recommendation is aspirin or warfarin. Patients at high risk—with a 6-12% annual risk of stroke—should be given warfarin. Articles based in primary care lean towards less aggressive diagnostic and therapeutic approaches.[15–18]

Patient's risk

Hypertension is an obvious risk factor in our patient, and corrected tetralogy of Fallot can be considered as valvular disease. The patient's syncope might be considered as equivalent to a transient ischaemic attack. Her risk score is at least one (probably more)—she is at least at moderate risk but probably at high risk.

Case history

A 56 year old married mother of one child

Partial list of problems
- Tetralogy of Fallot (corrected in 1976 at age 30, leaving residual mild pulmonary stenosis, regurgitation, and right bundle branch block)
- Labile hypertension (started in 1990)
- Paroxysmal atrial fibrillation (since 1996)
- Syncope (first episode in 1999):
  Hospitalised for one day
  High blood pressure
  Started on atenolol 25 mg a day and low dose aspirin

Investigations
- Echocardiography (no atrial or ventricular enlargement)
- Holter monitoring (normal)

Evidence based best recommendation

The evidence suggests that anticoagulants are justified in our patient. Aspirin is definitely recommended if she is not given anticoagulants.

Integrating patient's and doctor's narratives

Deliberations between the patient and doctor came to a head in March 2000. A cardiologist recommended starting the patient on anticoagulation with warfarin.

Patient

"What an embarrassment to have fainted! It just confirms my mother's claim, 'You're a frail child!' I didn't dance at parties and eventually stopped going to them altogether because it was boring … after the Fallot's full correction, life changed.

"One evening, almost by accident, I find myself dancing, without knowing the steps. When I sit down, my heart starts palpitating. What should I do? It lasts for a few minutes and then stops. I went too far …

"I'm attending a lecture, suddenly my heart starts palpitating … the whole room seems to be spinning wildly. I don't remember ever having felt this way

before … In the emergency room, they decide to hospitalise me. The following day, they let me out, and I go straight to my family doctor. He is tired—I see his eyes glaze over; I urge my husband, 'Time to go home' …

"A few days later, I meet my cardiologist, who supports the pills for blood pressure and clot prevention. How many times was my father hospitalised to stop his present medication and then start all over again with different ones … I don't want to slide down this slope … will my doctor help me with this?"

Doctor

"It is reasonable to keep her on a small dose β blocker and a low dose aspirin, yet the cardiologist favours warfarin, and the evidence is lacking. I feel quite anxious thinking about a stroke—what should I do? On the other hand, the decision is hers. Narrative and evidence—the lived experience of palpitations and their meaning. What about my story? My ego is flattered in this patient's care. She is an 'interesting case'; she writes to me, and I find much to be proud of in what she writes. Is this influencing my decisions? Reality is quite messy, even when the story sounds good. I also remember my own tiredness during the late night consultation: I couldn't have cared less about the evidence at that point."

Patient

"The pills I've been taking for three months now have to continue. I took the medication because I was afraid of what might happen during the trip abroad, but when I got back, you continued to scare me, and you asked me to carry on. With every pill I put in my mouth, I feel I'm poisoning myself … I felt that in the consultation with the cardiologist my feelings and wishes were not even considered … You did say, however, that in the end it *is* my decision … I will have to take charge of and live my life with no pills for as long as I can … no chance that, if my situation stays the way it is now, I will take more or stronger medications …

"I feel quite depressed now that I'll eventually, once again, become the same weak person I was at the beginning of my life … and I simply do not want and will not accept this."

Doctor

"Three family meetings were held during the course of one month. We shared information. We discussed our feelings. I respect that personal 'health belief model,' which is definitely different from my own. She wants to be guided by her inner feelings rather than by fear, and she finally decides no to warfarin, yes to atenolol, and yes to aspirin.

"Fully aware of the possible risks in this course of action, her husband shares her decision. I feel I can live with it too."

One year later

We return to the story one year later, when the patient is taking atenolol and aspirin; no palpitations have been felt or documented, and her blood pressure is controlled. A second literature search produces one new relevant article, which suggests that the patient is at high risk.[19] Further literature about the anticoagulation issue is identified [14 20]: warfarin probably is indicated.

Patient

"It makes my heart sink. Yes, I know that to be a Fallot is more risky, and again the statistics. How many times do I have to tell you that I am a person—not a statistic? I'm blessed with a supportive family—thank God for that; that in itself is a great remedy for Fallot.

"I understand how my doctor must feel—anxious, afraid to lose his patient, and highly responsible for her good care. Statistics always frighten doctors: too many stress factors in their own lives. What medications should my doctor be taking himself? I don't want to lose him either."

Doctor

Personal—"Am I anxious and fearful? Am I coming across to the patient as such? We always share a few laughs, which I like. Anyway, no anticoagulation for now. I should see my own doctor though—I haven't for a long time, my cholesterol level is high, and with my family history …"

Reflective—"The patient's beliefs, preferences, and family members' input were much more powerful than the rational evidence. I took part in the process without a sense of professional compromise. The final appropriate decision for the specific dilemma with that particular patient can be contrary to the apparent evidence. Looking for evidence and sharing narratives served our relationship. Progress was made on the way to a more mindful practice."[21]

Discussion

Clinical care is no easy task. Doctors are not inert vehicles that transmit therapeutic alternatives. Patients' values and contexts[4] and the relationships between patients and doctors play a crucial role; evidence is sometimes the minor player. The personal history between the doctor and patient and their relationship evolve to create a bond that affects decisions and healing.

Easy solutions do not exist, and evidence based medicine is no exception. A combination of evidence based medicine and narrative based medicine can enhance shared decision making and patient-doctor bonds, provide explicit deliberations, and enhance the doctor's personal and professional development. Addition of the physician's narrative can support Frank's idea that "when a patient's and provider's narrative meet, medicine can become a healing profession for both."[22]

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Lesson of the week

Wound botulism associated with subcutaneous drug use

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Wound botulism in drug users is a rare but important cause of weakness and neuropathy

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Botulism has long been considered a foodborne infection, the causative agent, *Clostridium botulinum* usually being transmitted in preserved foods. Yet *C botulinum* is widely distributed in soil and may be transmitted through wounds. An important distinction is that infections from food are limited to the amount of toxin ingested whereas in infections from wounds the toxin can be produced in situ until infection is eliminated from the wound.[1]

A new mode of presentation is occurring in drug users, the organism entering at sites of subcutaneous injection. The first reported case due to subcutaneous injection was in New York in 1982.[2] As drug use has increased so has the number of people exposed to *C botulinum*. Over 90% of cases of wound botulism have been reported in the United States (75% in California), with only a small number of cases in the United Kingdom and elsewhere in Europe.[1 3–6]

Botulism manifests as an acute descending paralysis, with involvement of autonomic and cranial nerves. *C botulinum* produces a potent exotoxin that binds irreversibly to the presynaptic membrane causing failure of transmission at the neuromuscular junction, autonomic ganglia, and parasympathetic nerve terminals. Diagnosis is based on clinical and neurophysiological findings, serology, and by identifying the organism or toxin. An antitoxin is available, but treatment remains largely supportive. We report a case of wound botulism in a subcutaneous heroin user who posed diagnostic difficulties.

Case report

A 50 year old man presented with a two week history of a flu-like illness and four days of dysarthria, dysphagia, shortness of breath, and neck discomfort. On the day of admission he developed diplopia and generalised weakness and collapsed at home. He was an intravenous and subcutaneous drug user (mainly heroin), and he was positive for hepatitis C.

In the emergency department he was restless, and his verbal response was indistinct. He had generalised weakness, depressed reflexes, and flexor plantars. Within 12 hours he was hypotensive, oliguric, tachypnoeic, increasingly drowsy, and confused. Within 18 hours arterial blood gases showed severe respiratory failure. He was intubated, ventilated, and transferred to the intensive care unit.

Neurological examination 48 hours later showed that he was conscious and able to obey commands but profoundly weak. He had complete ptosis, limitation of eye movements in all directions, and nystagmus on lateral gaze. His pupils were 4 mm in diameter and responded sluggishly to light. He had global facial weakness and severe bulbar dysfunction. He had symmetrical, mainly proximal, weakness with no movement at the shoulder, hip, or knee. Reflexes were depressed and plantars flexor.

Investigations showed a neutrophilia, increased concentration of C reactive protein, and normal routine biochemistry. Blood cultures grew *Staphylococcus aureus*. A drug screen was positive for opiates and benzodiazepine. A chest *x* ray film showed consolidation of the right lower lobe. An echocardiogram appeared normal. Computed tomography and magnetic resonance imaging of the head gave normal results as did computed tomography of the cervical spine. Lumbar puncture showed an opening pressure of 12 cm of water (reference range 5-20 cm of water), no white cells, 17 red cells, normal glucose levels, protein 0.49 g/l (reference range 0.2-0.4 g/l), and negative bacteriology and virology results. A Tensilon test was weakly positive.

He was treated with antibiotics, thiamine, and B vitamins. Neurophysiological studies could not be performed on the intensive care unit where he was admitted, and it was not possible to transfer him to the regional neurosciences centre until 10 days after admission. He was therefore treated empirically with intravenous immunoglobulin, the presumed diagnosis being Miller-Fisher variant of the Guillain-Barré syndrome.

He remained systemically stable but unchanged neurologically. On day 4 several raised, erythematous areas were noted on his buttocks.

On day 10 he was transferred to the neurosciences centre. Neurophysiological studies showed normal sensory responses, noticeably attenuated motor