Truthfulness and Personal Disclosure in the Physician–Patient Relationship

TO THE EDITOR: I enjoyed the recent essay by Agrawal (1) but remain concerned about half-truths and outright lying to patients. What we do in our personal lives really should have no part in the consultation.

I was away from my practice for 8 months due to critical illness after bypass surgery in September 2007. My patients had missed me. Now I am returning, and many consultations begin with “How are you, doctor?” It is difficult for me to reply honestly because my role is to be “the doctor.” Most of my patients are not really interested to learn of my chronic lung disease, physical infirmity, and generalized anxiety with posttraumatic stress disorder. Nor do they wish to hear about my constant aches and pains and how I’ve had an interrupted sleep pattern since January. But these are important to me, and sometimes I feel that bits and pieces of my frailty should be shared.

However, one patient booked an appointment specifically to check on me and reassure me that he has never felt better since his bypass 24 years ago. He was surprised and grateful that I wished to discuss his cholesterol treatment; perhaps I was a little too dismissive of his overture to help me.

Trust is something we must earn—outright lying to our patient cannot be a good thing. However, being economical with the truth need not betray that trust.

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Reference

TO THE EDITOR: Regarding the recent article by Agrawal (1), I understand the short-term benefit of her lie, but if the patient ever found out the truth, that great relationship she built with her patient will be forever ruined. There will be no trust in a person who lies. I have always found that if patients could not deal with the truth of any situation, they and you are better off separating. As Agrawal said in her article, one lie leads to another. And then where does it end?

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IN RESPONSE: I agree that physicians must be truthful to earn the trust of their patients, and that trust is the bedrock of a healthy physician–patient relationship. This is precisely the reason for my distress with the patient interaction I described.

However, the issue of patient and physician perception in regard to sexual orientation is complicated. A 1994 national survey of gay and lesbian physicians revealed that 67% felt they “would jeopardize their practices if their colleagues learned they are lesbian, gay, or bisexual” and 75% felt they “would jeopardize their practices if their patients learned they are lesbian, gay, or bisexual” (1).

At least 2 further studies of patients’ attitudes on this issue seem to support this apprehension on the part of physicians. Lee and colleagues (2) found that many of the 502 patient respondents in a national survey indicated that they would change health care providers if they found out their provider was gay or lesbian (30.4%) or would change practices if gay or lesbian providers were employed there (35.4%). Another study, published in 1998, found that 11.8% of respondents would refuse to see a gay or lesbian physician, the most common reasons being fears of incompetence and worries about feeling “uncomfortable” (3).

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Estimates suggest that several hundred thousand gay or lesbian providers practice in the United States. Therefore, this issue can affect many patients, families, and their providers. I hope my essay, your letters, a continuing dialogue, and further studies will foster greater understanding among patients, physicians, communities, and society in regard to this issue.

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Potential Financial Conflicts of Interest: None disclosed.

References

Does Lower Diabetes-Related Numeracy Lead to Increased Risk for Hypoglycemic Events?

TO THE EDITOR: In reviewing the findings of Cavanaugh and colleagues (1), we were impressed that the association between low numeracy and poor glycemic control seemed much stronger among insulin users than among persons taking oral medications. In addition, their finding that insulin users with low numeracy were less likely than those with high numeracy to self-adjust insulin dosage and count carbohydrates provides a possible mechanism. This would suggest that not only does numeracy affect quality, it may also have important implications for patient safety. Hypoglycemia relating to insulin use is a leading cause of adverse drug events resulting in emergency department visits (2), and we have found that patients with diabetes who report having problems learning about their health because of reading difficulties have higher incidences of severe hypoglycemia episodes (3). Taken together, this research suggests that system-level interventions to mitigate the effects of limited literacy and low numeracy must involve some combination of adequate self-management support regarding insulin use, individualized glycemic targets, and proactive surveillance (4) for adverse drug events related to insulin.

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References

IN RESPONSE: We agree that interventions to reduce the frequency and severity of hypoglycemic episodes in patients are an integral component of diabetes care and patient safety. Patient literacy and numeracy may play an important role in adverse events related to blood sugar management. For a subgroup of patients in our study (n = 163 [41% of the primary sample]), we were able to download data from their blood glucose meters and obtain information about the frequency of hypoglycemic events (defined as blood glucose level <3.33 mmol/L [<60 mg/dL]) and the proportion of blood glucose measures less than 3.89 mmol/L (<70 mg/dL). Most of these patients (58%) did not have a measured hypoglycemia event; however, 42% recorded at least 1 event, and 10% of these patients had more than 5 events recorded. Although patients with type 1 diabetes (n = 42) were more likely to have hypoglycemic events than patients with type 2 diabetes (n = 121) (86% vs. 27%; P < 0.001), neither health literacy nor diabetes-related numeracy was associated with the number of hypoglycemic events as measured by self-monitoring of blood glucose levels in patients with either type of diabetes. Health literacy or diabetes-related numeracy was also not found to be statistically significantly associated with a proportion of blood glucose meter readings that were low (<3.89 mmol/L [<70 mg/dL]). Results were also similar if patients using only insulin (n = 121) were included in the analysis. This subgroup evaluation was limited by its small, selected sample of patients, and it was not adequately powered to evaluate this important question. Patients with lower numeracy may have had higher rates of hypoglycemic episodes but did not record these episodes with their glucose meter or did not bring their meter to clinic for download. Additional, larger studies are needed to define the role of literacy and numeracy in the prevention of serious adverse events and overall safety of patients with diabetes and other chronic diseases.

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Clinical Observations

Disseminated Histoplasmosis Associated with the Treatment of Rheumatoid Arthritis with Anticytokine Therapy

Background: Tumor necrosis factor (TNF)-α antagonists increase risk for opportunistic infection.

References
Objective: To describe a case of disseminated histoplasmosis associated with treatment with adalimumab.

Case Report: A 41-year-old white man with rheumatoid arthritis, treated with adalimumab for 9 months, presented for evaluation of 3 weeks of worsening malaise and intermittent fevers. He did not report any joint pain, myalgias, or rash. He subsequently developed progressive dyspnea without cough or chest pain.

On physical examination, his temperature was 39.4 °C, and he had tachycardia and tachypnea. His oxygen saturation was 94% on 3 L of oxygen by nasal cannula. He had scleral icterus but no lymphadenopathy. His lungs were clear to auscultation. He had no cardiac murmurs. His abdomen was distended, with marked, tender hepatosplenomegaly. Joint and skin examinations were normal. He had mild anemia and elevated serum transaminases. Ultrasonography confirmed marked hepatosplenomegaly without ductal dilation.

On admission, adalimumab treatment was stopped and broad-spectrum antibiotics initiated. Blood and urine cultures for routine bacteria and fungi were negative. We performed bronchoscopy with bronchoalveolar lavage, and bacterial and fungal stains and cultures as well as acid-fast staining were all initially negative. As the patient developed respiratory failure, we added amphotericin B. Further analysis of the bronchoalveolar lavage revealed histoplasmosis by DNA polymerase chain reaction. Urine subsequently tested positive for histoplasma antigen. Because the patient had developed pancytopenia, we performed bone marrow biopsy; it showed noncaseating granulomas consistent with histoplasmosis. The patient responded well to amphotericin B and, after 7 days, was transitioned to itraconazole therapy for 6 weeks. We then discontinued itraconazole treatment but planned to resume prophylactic itraconazole if the patient required further immunosuppressive rheumatoid arthritis treatment. We permanently discontinued adalimumab treatment.

Discussion: Therapy targeting inflammatory cytokines, such as TNF-α, represents novel treatment for such diseases as rheumatoid arthritis but comes with increased risk for opportunistic infections (1). Tumor necrosis factor-α induces collagenase production by synovial macrophages, resulting in joint degradation; blocking this through anti-TNF agents leads to clinical improvement. Tumor necrosis factor-α is also important in host defense: It is essential in the cascade of inflammatory cytokines that cause granuloma formation, a necessary response for effective defense against histoplasma (2).

Currently available anticytokine agents include etanercept, infliximab, and adalimumab. All have been associated with an increased risk for infection, particularly by pathogens cleared by a granulomatous response, but with important differences. Infliximab and adalimumab suppress interferon-γ production, also needed for granuloma formation; this may further increase the risk for infection. Postlicensure reporting to the U.S. Food and Drug Administration indicates that disseminated histoplasmosis has occurred with greater frequency with infliximab than with etanercept. Given its similarities to infliximab, adalimumab, a newer agent with fewer reported cases of infection, is likely to have a higher incidence of granulomatous infection (3).

Cases of histoplasmosis generally occur in the Mississippi River Valley (the patient lived in Branson, Missouri), and initial infection often remains latent until an immunocompromised state arises, at which time patients may present with vague symptoms, including fever, malaise, cough, and dyspnea. Diagnosis can be confirmed through fungal stains or culture of bronchoalveolar lavage or direct lung biopsy specimens, or by urine histoplasma antigen testing.

Because few cases of histoplasmosis in patients treated with TNF-α antagonists have been reported, prophylactic treatment or routine screening for histoplasmosis is not recommended. Providers caring for patients with rheumatoid arthritis should weigh the potential benefits of anticytokine therapy against the risk for infection for each patient (4).

Conclusion: Treatment with adalimumab may increase risk for disseminated histoplasmosis in endemic areas.

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Potential Financial Conflicts of Interest: None disclosed.

References

Clomipramine-Induced Diabetes

Background: Clomipramine is a tricyclic agent with both antidepressant and antiobsessional properties. Like other tricyclics, clomipramine inhibits uptake of norepinephrine, dopamine, and serotonin. Clomipramine has anticholinergic side effects: retention of urine, reduced intestinal motility, increased eye pressure, orthostatic hypotension, and heart rhythm disturbance. Other side effects reported include weight gain, vomiting, diarrhea, and sedation.

Objective: To describe an 84-year-old woman who developed diabetes during treatment with clomipramine for depression.

Case Report: The patient was admitted to our hospital in December 2006 because of polyuria, dehydration, and obtunded sensorium 5 months after starting treatment with clomipramine, 25 mg/d, to control depression episodes. Her medical history was notable for well-controlled hypertension and atrial fibrillation; her medications were aspirin and irbesartan.

On admission, physical inspection showed dehydration, and neurologic examination revealed obtunded consciousness without other abnormalities. Laboratory data showed severe hyperglycemia (25.50 mmol/L), ketonemia, and a hemoglobin A1c level of 0.12. The serum sodium level was 158 mmol/L, and serum creatinine level was 137.25 μmol/L [1.8 mg/dL]. Arterial blood gas analysis showed acidosis (pH, 7.105; bicarbonate level, 3.5 mmol/L). Urinalysis revealed glycosuria and ketonuria. Additional data, including complete blood count, serum lipase level, serum amylase level, chest radiography, and computed tomography of the head and abdomen, were unremarkable.

Potential Financial Conflicts of Interest: None disclosed.

References
The patient had no family evidence or history of diabetes or glucose intolerance. Her body mass index was 23 kg/m², and we noticed no weight gain during treatment.

After discontinuation of clomipramine and metabolic compensation with intravenous insulin and fluids, she required regular insulin, 30 U/d. Her blood glucose levels rapidly normalized, and she was discharged 10 days later without insulin therapy and with a diabetic diet and psychological treatment.

The patient remained metabolically stable (hemoglobin A₁c, level, 0.05). After 3 months and after she provided written informed consent, we reintroduced clomipramine under medical surveillance. One week later, the patient’s fasting glucose level increased to 13.88 mmol/L (250 mg/dL) with glycosuria and ketonuria. Again, we discontinued clomipramine treatment, and the glycemia normalized in 2 days. She has been euglycemic without therapy for the past 6 months.

Discussion: It is well recognized that certain antipsychotic medications can cause clinically significant elevations in glucose concentration and an increased risk for obesity and the metabolic syndrome (1).

Of recent interest are the increasing numbers of reported cases of new-onset diabetes in patients receiving treatment with atypical antipsychotic agents, such as clozapine (2). In most cases, the mechanisms by which hyperglycemia occurs are not fully understood, although several possible theories have been proposed for this drug class (3).

We considered our patient’s hyperglycemia to be induced by clomipramine because of the temporal relationship, the recurrence of glucose elevation with reintroduction of the drug, and the normalization of the glucose level after discontinuation of therapy with the drug. To our knowledge, there have been no previous reports of hyperglycemia associated with the use of clomipramine. Although clomipramine has not been reported to cause diabetes in humans, it can cause diabetes in mice: Sugimoto and colleagues (4) recently demonstrated that clomipramine induced hyperglycemia in mice by blocking the 5-hydroxytryptamine-2B or -2C receptor, which resulted in facilitation of adrenaline release.

Conclusion: Given these consequences of poor glycemic control, physicians should be aware of medications that may affect glucose metabolism. However, further studies with this drug are necessary before any conclusions can be made, and more data are needed to prove an association between clomipramine and diabetes.

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References

Corrections

Correction: Sjögren-Type Syndrome after Allogeneic Bone-Marrow Transplantation

There is a typographical error in the name of the first author of an article on bone marrow transplantation (1). The author’s name is Alois A. Gratwohl.

Reference

Correction: In the Clinic: Allergic Rhinitis

The Treatment section of the In the Clinic on allergic rhinitis (1) mentions a discontinued antihistamine that is mistakenly identified as terbinafine, an oral antifungal agent that is still on the market. The correct drug is terfenadine, which was marketed in the United States as Seldane.

Reference

Correction: Reported Methodologic Quality and Discrepancies between Large and Small Randomized Trials in Meta-analyses

A recently published letter (1) lists the authors incorrectly. The corrected list of authors is as follows:

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