

performing drug use evaluations, and analyzing a clinician's workload statistics. The following reports were generated as part of the database application for the infectious diseases consult service:

- Antimicrobial usage, including itemization of costs and indications
- Summary of specific diagnoses, treatments used, and cost of treatments
- Types and number of specific diagnoses
- Diagnoses, with organisms grown and antimicrobial sensitivity patterns
- Summary of pharmacist interventions
- Summary of "active" patients within the consult service

The development and piloting of an electronic patient monitoring form for the infectious diseases consult service provided assurance of the benefits of incorporating a PDA into a specialized area of clinical practice. There are many untapped resources and applications that PDAs can offer to pharmacists. Exploring the contributions of this new technology will definitely facilitate the progress of clinical pharmacy services.

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Combined Olanzapine- and Risperidone-Induced Diabetic Ketoacidosis

Atypical antipsychotics have been reported to cause diabetic ketoacidosis and new-onset diabetes.¹ The risk of developing diabetes mellitus or impaired glucose tolerance (or both) is 2-fold greater with atypical agents than with typical agents.² A recent admission to our intensive care unit illustrates the need to be aware of the possibility of drug-induced diabetic ketoacidosis and new-onset diabetes.

A 22-year-old Caucasian man with a 1-year history of schizophrenia was transferred to our institution for management of diabetic ketoacidosis. The patient had presented with thirst, polyuria, polydipsia, nocturia, and mild epigastric pain of several weeks' duration. The results of laboratory investigations at the time of presentation were as follows: presence of serum ketones, blood glucose 27.6 mmol/L (normal range 3.3 to 6 mmol/L), serum pH 7.10, serum bicarbonate 5 mmol/L (normal range 22 to 26 mmol/L), and anion gap 20 (normal range 8 to 12). The patient had no history of diabetes mellitus, but there was a family history of the condition (both father and grandfather). The patient was obese, weighing 125 kg and having a body mass index of 40 kg/m².

Before admission, the patient had been receiving olanzapine 10 mg daily, risperidone 4 mg twice daily, clonazepam 0.25 mg twice daily, and benzotropine 2 mg as needed. A review of the patient's neuroleptic therapy revealed that he had been started on risperidone 0.5 mg twice daily approximately 1 year before the current admission; olanzapine had been added soon after to control decompensation. Medical records confirmed that 2 months before the current presentation, the patient had been taking olanzapine 10 mg once daily and risperidone 4 mg twice daily. The possible contribution of these 2 agents to the patient's metabolic problems went unrecognized until 3 days after presentation. At that time, both the olanzapine and the risperidone were discontinued, as it was believed that they were contributing to the diabetic ketoacidosis and new-onset diabetes. The patient was discharged on alternative antipsychotics and insulin therapy with no adverse sequelae.

Both olanzapine and risperidone have been reported to induce diabetic ketoacidosis and new-onset diabetes.^{1,3,4} The exact mechanism or mechanisms underlying this toxic effect are unknown. The lag period from initiation of atypical antipsychotics to development of hyperglycemia also varies. Several



factors increase the risk of diabetic ketoacidosis and diabetes induced by atypical antipsychotics, including a diagnosis of schizophrenia, overweight (body mass index greater than 30 kg/m²) before initiation of treatment, weight increase of more than 10% while receiving treatment, previous history of glucose dysregulation and hypertension, African-American or Hispanic background, and family history of diabetes mellitus.⁵ While absence of these risk factors does not preclude development of diabetic ketoacidosis or diabetes while a patient is receiving atypical antipsychotics, their presence should encourage clinicians to weigh the risks and benefits before initiating these drugs.⁵

After initiation of an atypical antipsychotic agent, baseline and 6-month monitoring of fasting plasma glucose levels, fasting cholesterol levels, and triglyceride levels is recommended.⁵ For patients with the risk factors described above, it has been recommended that glycosylated hemoglobin levels be measured every 3 months.⁵ Weight should be recorded at baseline and monitored regularly during treatment, and prevention of weight gain should be emphasized.^{5,6} If the patient is already obese, he or she should be encouraged to lose weight.⁶ Switching from one atypical antipsychotic to another may improve glucose control in some individuals.⁷

As pharmacists, we can discuss these possible adverse effects during patient counselling, explore alternative therapies for patients at high risk, and ensure that proper monitoring is undertaken when patients are started on atypical antipsychotics.

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