



## Co-management Guide

Pediatric  
Endocrinology

Type 1 Diabetes

<b>Guidelines Referenced</b>	<a href="http://care.diabetesjournals.org/content/40/Supplement_1/S11.long">http://care.diabetesjournals.org/content/40/Supplement_1/S11.long</a> <a href="http://care.diabetesjournals.org/content/28/1/186.long">http://care.diabetesjournals.org/content/28/1/186.long</a>
<b>Background</b>	<p>Type 1 diabetes mellitus is caused by insulin deficiency following destruction of the insulin-producing pancreatic beta cells and most commonly presents in childhood. T1DM remains the most common form of diabetes in childhood, accounting for approximately two-thirds of new diagnoses of diabetes in patients ≤19 years of age in the United States.</p> <p>The age of presentation of T1DM has a bimodal distribution, with one peak at four to six years of age and a second in early puberty.</p> <p>In the United States, the incidence of T1DM in non-Hispanic white children and adolescents is 23.6 per 100,000 per year, and rates are substantially lower in other racial or ethnic groups [African American, Hispanic, Asian-Pacific Islanders, and American Indians (2.55, 1.62, 1.29, 0.6, and 0.35 cases per 1,000 children 0 to 19 years old, respectively) ].</p> <p>The lifetime risk of developing T1DM is significantly increased in close relatives of a patient with T1DM and varies depending on degree of relation.</p> <p>T1DM is always insulin requiring for the entirety of patient’s life (at least at this time).</p>
<b>Initial Evaluation</b>	<p><u>Classic presentation</u> (most common, usually ambulatory setting)</p> <ul style="list-style-type: none"> <li>• chronic polydipsia &amp; polyuria, (90%) (eg, nocturia and bedwetting, increased frequency and/or unusually wet diapers, and persistent thirst).</li> <li>• weight loss (50%)</li> <li>• hyperglycemia and +/- ketosis</li> <li>• vague complaints of lethargy.</li> <li>• perineal candidiasis (young children and girls)</li> <li>• Visual disturbances are also common.</li> </ul> <p><u>Diabetic ketoacidosis</u> (second most common form of presentation, more severe)</p> <ul style="list-style-type: none"> <li>• Classic presentation signs/symptoms PLUS ketoacidosis</li> <li>• fruity-smelling and/or heavy prolonged breath (Kussmaul)</li> <li>• nausea and/or vomiting</li> <li>• neurologic findings including drowsiness and lethargy.</li> </ul> <p>Note: DKA can be misinterpreted as an acute vomiting illness because classic pediatric symptoms of dehydration (decreased urination) are masked by the polyuria that is associated with glycosuria. DKA can also be thought to be an anxiety episode because Kussmaul breathing can replicate anxiety related hyperventilation.</p> <p><u>Silent</u> (asymptomatic, incidental discovery or by high index of suspicion)</p> <ul style="list-style-type: none"> <li>• Routine screening serum or urine: hyperglycemia or glucosuria</li> <li>• Patients with FHx of DMT1, pancreatic autoantibody screening (not standard of care)</li> </ul> <p><u>Special populations</u></p>



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	<p>Infants — A variety of disorders can cause hyperglycemia during infancy. Although autoimmune classic T1DM can occur in the first year of life, neonatal diabetes (&lt;6 months of age at diagnosis) is uncommonly – if ever – autoimmune in nature. Neonatal diabetes is a rare disorder caused by one of several genetic defects in pancreatic development or beta cell function.</p> <p>Young children — Younger children are more vulnerable to dehydration compared with older children because they are less able to compensate for pathologic processes by seeking fluids and increasing fluid intake. In addition, children younger than six years of age are more likely to present with DKA, because health care personnel and families less often suspect diabetes in this age group, especially if still in diapers as increasing urine volume is often missed. This can lead to a prolonged duration of illness and more severe metabolic decompensation before diagnosis.</p> <p>Obese children—It is important to note that children that are overweight or obese can still develop type 1 diabetes, even as a teenager. Likewise, even a child with type 2 diabetes with an A1c above 8.5% or a fasting glucose above 200 mg/dl would require insulin therapy initially. Children with type 2 diabetes can also develop DKA. Therefore a consult on an obese patient with new onset diabetes should be just as timely as if the child were lean or of normal body weight.</p>	
<p><b>Initial Management/ When to Refer</b></p>	<p>Children with DKA require hospitalization, rehydration, and insulin replacement therapy.</p> <p>Children without DKA but with new onset diabetes warrant immediate referral to prevent occurrence of DKA while awaiting initial appointment. We recommend contacting on call physician at the time diagnosis is suspected for instructions regarding admission versus same day or next day appointment and aid in confirming diagnosis if need be. No patient with new onset type 1 diabetes should wait more than 1 day for appointment. Sending a referral via typical channels may result in delay of care and poor outcome.</p>	
<p><b>Pre-Visit Work Up</b></p>	<p>Diagnostic criteria for diabetes — Diabetes mellitus is diagnosed based upon one of the following four signs of abnormal glucose metabolism:</p> <ul style="list-style-type: none"> <li>●Fasting (no caloric intake for 8 hours minimum) plasma glucose <math>\geq 126</math> mg/dL on more than one occasion.</li> <li>●Random venous plasma glucose <math>\geq 200</math> mg/dL in a patient with classic symptoms of hyperglycemia</li> <li>●Plasma glucose <math>\geq 200</math> mg/dL measured two hours after a glucose load of 1.75 g/kg (maximum dose of 75 g) in an oral glucose tolerance test (OGTT). Most children and adolescents are symptomatic and have plasma glucose concentrations well above <math>\geq 200</math> mg/dL; thus, OGTT is seldom necessary to diagnose T1DM.</li> <li>●Glycated hemoglobin (A1C) <math>\geq 6.5</math> percent. This criterion is more useful to diagnosis of type 2 diabetes mellitus (T2DM) in adults, and should be confirmed by hyperglycemia.</li> </ul>	
<p><b>Co-management Strategy (as appropriate)</b></p>	<p><b>Specialist scope of care</b></p> <ol style="list-style-type: none"> <li>1. Initiate and manage insulin therapy, including log reviews between visits</li> <li>2. Initiate initial and subsequent education</li> <li>3. Manage ketones and hyperglycemia during periods of insulin omission or illness</li> </ol>	<p><b>Primary care scope of care</b></p> <ol style="list-style-type: none"> <li>1. Manage all nondiabetes/nonendocrine related medical conditions</li> <li>2. Support patient by asking about diabetes, previous A1c and reviewing records from endocrine office in regards to issues of adherence and psychological distress</li> </ol>



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	<ol style="list-style-type: none"> <li>4. Prescribe all insulin and diabetes supplies</li> <li>5. Perform annual screening labs and referral as recommended for patients with type 1 diabetes</li> </ol>	<ol style="list-style-type: none"> <li>3. Contact endocrine office for needed support in diabetic patients with acute vomiting illness especially any including moderate to severe ketosis, illnesses that require steroid administration, or other special circumstances such as preoperative management</li> </ol>
<b>Return to Primary Care Endpoint</b>	<p>Patients with type 1 diabetes will follow with an endocrinologist for the entirety of their lives, even into adulthood. However, primary care physicians are critical in helping manage diabetes, by discussing issues of adherence and psychological coping. Referrals to therapy services and rarely, psychiatrists, are often needed in patients with diabetes at some during the duration of their childhood and adolescent years.</p>	