The Evolution of Care for Acute Myeloid Leukemia and the Challenges of Defining Value

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ABSTRACT: Adult acute myeloid leukemia (AML) is a cancer of the blood and bone marrow. Distinct subtypes of AML have been defined based on morphology, immunophenotyping, and molecular genetics. AML usually progresses very quickly if it is not treated. Managed care needs to appreciate the expanded indications of existing drugs and how this changes the standard of care, whether that be more frequent or longer use. Like many other cancers there are indirect costs and burdens, some of which can be considerable. Managed care organizations can contribute to improved outcomes and reduced costs by increasing the understanding of current therapies, recognizing the potential benefit of future therapies, and developing an understanding of targeted drugs and their place in therapy.

KEY WORDS: acute myeloid leukemia, managed care, oncology medical home

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Adult acute myeloid leukemia (AML) is a cancer of the blood and bone marrow. It is the second most common type of acute leukemia in adults. AML is also sometimes referred to as acute myelogenous leukemia, acute myeloblastic leukemia, acute granulocytic leukemia, and acute nonlymphocytic leukemia.

In AML, there is a block in differentiation and uncontrolled proliferation of myeloid precursor cells, and the myeloid stem cells usually become a type of immature white blood cell called myeloblasts (or myeloid blasts).1 The myeloblasts in AML are abnormal and do not become healthy white blood cells. Leukemia cells, or leukemic stem cells (LSCs), can build up in the bone marrow and blood, so there is less room for healthy white blood cells, red blood cells, and platelets. When this happens, infection, anemia, or hemorrhaging may occur. The leukemia cells can spread outside the blood to other parts of the body.

AML is the most common type of acute leukemia in adults, and its incidence increases with age.2 This year, an estimated 21,380 people of all ages (11,960 men and 9420 women) in the United States will be diagnosed with AML.3 It is primarily a disease of older adults; the disease rarely occurs before age 45, and the median age of onset is around 67 years. With post-remission therapy, 5-year survival rates of <5% to 20% and >40% may be achieved for patients older and younger than 60 years, respectively.3

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AML CLASSIFICATION

Genetic mutations, often in genes coding for signaling proteins or transcription factors, are required to promote the transformation to LSCs and, consequently, overt AML. Genetic alterations in the tumor cell have been recognized as a cause of leukemia, initially described as karyotypic abnormalities (eg, deletions, translocations) that are detectable by cytogenetic analysis in approximately 50% of patients.

In addition to age and performance status, cytogenetic and molecular aberrations are the most important tools to predict outcome in AML. The cytogenetic profile (karyotype or chromosomal aberrations) serves as a prognostic indicator in AML by which patients are stratified into favorable, intermediate, and adverse risk groups (Table 1). Chromosome alterations and complex karyotype (described as > 3 chromosomal abnormalities) are associated with poor response to therapy and reduced survival. The presence of other cytogenetic abnormalities, such as inv(16) or t(8;21) in core-binding factor AML indicate longer disease remission and survival. Approximately 40% to 50% of all AML cases are cytogenetically normal AML (CN-AML). CN-AMLs have an intermediate risk for relapse. With respect to clinical outcomes, substantial heterogeneity is observed in this group.

AML is characterized by multiple somatically acquired mutations that affect genes of different functional categories (Box 1). Mutations in genes encoding epigenetic modifiers, such as DNMT3A, ASXL1, TET2, IDH1, and IDH2, are commonly acquired early and are present in the founding clone. By contrast, mutations involving NPM1 or signaling molecules (FLT3, TP53, RAS gene family) are typically secondary events that occur later during leukemogenesis. With a frequency of approximately 30%, AML with NPM1 mutation represents the largest class of AML. About 75% of patients also carry mutations in DNA methylation or hydroxymethylation genes (DNMT3A, IDH1, IDH2<sup>19, 20</sup>, TET2); 40% have concurrent FLT3 internal tandem duplication (FLT3-ITD) mutations; 20% have NRAS mutations; and approximately 20% exhibit mutations in cohesion complex genes (RAD21, SMC1A, SMC3). These mutations have been found to affect clinical outcomes such as remission rates, disease-free survival, event-free survival, and overall survival (OS).

Two staging systems are commonly used for AML. The French-American-British (FAB) classification system<sup>21</sup> is based on morphology to define specific immunotypes. The World Health Organization (WHO) classification reviews chromosome translocations and evidence of dysplasia. While the FAB classification system is useful and is still commonly used to group AML into subtypes, it does not take into account many of the factors that are known to affect prognosis. The WHO system is a newer system that divides the diagnosis into specific groups (Table 2).

This classification of AML is more clinically useful and produces more meaningful prognostic information than the FAB criteria.<sup>10</sup>

The current WHO classification is based on lineage demonstrated by cell surface antigen expression. Distinct subtypes within each lineage are further defined based on morphology, immunophenotyping, and molecular genetics. New somatic/acquired gene mutations have refined the classification of myeloid neoplasms and have been in-

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**Table 1. Risk Status Based on Validated Cytogenetics and Molecular Abnormalities**

<table>
<thead>
<tr>
<th>Risk Status</th>
<th>Cytogenetics</th>
<th>Molecular Abnormalities</th>
</tr>
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<tbody>
<tr>
<td>Favorable-risk</td>
<td>Core binding factor: inv(16) or t(16;16) or t(8;21) or t(15;17)</td>
<td>Normal cytogenetics: NPM1 mutation in the absence of FLT3-ITD or presence of FLT3-ITD&lt;sup&gt;ITD&lt;/sup&gt; or isolated biallelic (double) CEBPA mutation</td>
</tr>
<tr>
<td>Intermediate-risk</td>
<td>Normal cytogenetics (without poor-risk genetic lesions)</td>
<td>Core binding factor with KIT mutation</td>
</tr>
<tr>
<td>Poor-risk</td>
<td>Complex (&gt; clonal chromosomal abnormalities)</td>
<td>Normal cytogenetics: With FLT3-ITD mutation</td>
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Other undefined
Box 1. Notable Gene Mutations in Acute Myeloid Leukemia

**ASXL1**
Additional sex comb–like 1 (ASXL1) mutations are loss–offunction mutations that tend to occur in exon 12 and are generally frameshift and nonsense mutations that result in protein truncation and loss of PRC2-mediated histone 3 lysine 27 trimethylation. Mutations in ASXL1 occur with a frequency of 5% to 17% in de novo AML, and at least 30% in AML arising from antecedent myeloid neoplasms. **ASXL1** mutations are 5 times more common in older (60 years or older) patients than in those younger than 60 years. In AML, **ASXL1** mutations usually do not occur in **NPM1** or **FLT3-ITD** mutated patients, but are frequently associated with **RUNX1**, **SRSF2**, and **IDH2** mutations. Among older patients, **ASXL1** mutations are associated with t(8;21), wild-type **NPM1**, the absence of **FLT3-ITD**, mutated **CEBP4**, and overall inferior CR rate and survival. **ASXL1** mutations are also associated with resistance to chemotherapy and independently associated with shorter OS.

**CEBP4**
The CCAAT enhancer binding protein alpha (CEBP4) is an intronless gene with a single exon that encodes for a transcription factor that controls gene expression during hematopoiesis. Mutations in **CEBP4** are observed in 6% to 10% of all AML and 15% to 19% of CN-AML cases and commonly in association with del(9q). Bi-allelic mutations occurred in 4% to 5% of AML cases and are associated with a normal karyotype. Germine CEBPA mutations are also observed in familial AML cases. **FLT3-ITD** co-occurs with CEBPA mutations in 22% to 33% of AML cases.

AML patients with bi-allelic CEBPA mutations have an increased CR rate, longer remission rates, favorable survival, and a relatively good prognosis. AML with a single CEBPA mutation is associated with survival rates similar to that of AML with wild-type CEBPA. Patients with CN-AML who have mutated CEBPA, in the absence of FLT3 mutations, can be offered intensive chemotherapy alone, sparing the potential complications of HSCT.

**DNMT3A**
Mutations in the DNA methyltransferase 3A gene (DNMT3A) have recently been identified as pre-leukemic mutations, arising early in AML evolution and persisting in times of remission. They occur in 18% to 22% of all AML cases and approximately 34% of CN-AML cases. They often cooccur with mutations in FLT3 (ITD) or tyrosine kinase domain [TKD], **NPM1**, and **IDH1**.

Patients with AML harboring DNMT3A mutations tend to have shorter OS. It was reported that patients with DNMT3A-mutated AML have an inferior survival when treated with standard-dose anthracycline induction therapy. High-dose daunorubicin compared with standard-dose daunorubicin improved the rate of survival among patients with **DNMT3A** or **NPM1** mutations or **KMT2A** translocations, but not among patients with wild-type **DNMT3A**, **NPM1**, and **KMT2A**.

**FLT3**
FLT3 encodes a class II family receptor tyrosine kinase that acts as a cytokine receptor for the FLT3 ligand and is responsible for the proliferation and differentiation of hematopoietic stem cells. In normal bone marrow, FLT3 is expressed only on immature hematopoietic stem cells. FLT3 mutations are among the most frequent mutations observed in AML, occurring at a frequency of 20% to 30%, and 2 types are distinguished: **FLT3-ITD** (75%–80%) and **FLT3-TKD** (20%–35%). Both types of mutations constitutively activate FLT3 signaling, promoting blast proliferation.

The **FLT3-ITD** mutation has a negative prognostic effect in AML. Normal-karyotype AML patients bearing an **FLT3-ITD** mutation have a poorer prognosis than AML patients with wild-type **FLT3**. TKD predict a particularly poor prognosis. **FLT3-ITD** mutations are associated with increased risk of relapse. In AML with an **FLT3-ITD** mutation, the high relapse rate and poor outcome depend mainly on the ITD allelic ratio. This effect on prognosis is modulated by the mutated to wild-type allele ratio, with inferior outcome in the presence of an increased load of ITDs in **FLT3**.

**IDH1/2**
Mutations of the isocitrate dehydrogenase (**IDH**) 1 and 2 genes are gain–of–function mutations. These mutations lead to novel enzymatic activity leading to the production of a putative oncometabolite, inhibiting TET2 function, increasing global DNA hypermethylation, and impairing hematopoietic cell differentiation. **IDH1** and **IDH2** mutations were discovered in 15% to 25% AML. They are found more frequently in older patients and also in non-hematological tumors. **IDH1** and **IDH2** mutations are frequently associated with normal karyotype AML (25%–30%) and **NPM1** mutation. **IDH** mutations are associated with reduced survival in CN-AML cases with **NPM1** mutations and wild-type **FLT3**. **IDH1/2** gene mutations have been considered a potential predictive marker with hypomethylating agents such as decitabine and azacytidine.

**KIT**
The KIT gene encodes a type III tyrosine kinase receptor glycoprotein that is responsible for the growth, proliferation, and...
Box 1. Notable Gene Mutations in Acute Myeloid Leukemia (continued)

differentiation of hematopoietic cells, melanocytes, and germ cells. This mutation is rare in AML (<5%) but present in 20% to 25% in AML with t(8;21) and 30% in AML with inv(16).

\textit{KIT} mutations are associated with an increased risk of relapse and negate the good prognosis of core-binding factor AML. \textit{KIT} mutations confer increased relapse risk and reduced survival.\textsuperscript{,}\textsuperscript{4.7}

\textbf{KMT2A}

The lysine methyltransferase 2A (\textit{KMT2A}) plays roles in hematopoiesis and cell differentiation and is rearranged commonly by translocations in acute lymphoblastic leukemia and AML. In adult CN-AML, the frequency of \textit{KMT2A} rearrangement is 11%.\textsuperscript{4} Translocations affecting the \textit{KMT2A} gene and \textit{NPM1} mutations are mutually exclusive. The \textit{KMT2A} partial tandem duplication (\textit{KMT2A}-PTD) mutation is seen in 3% to 7% of AML and is associated with a normal karyotype (5%-11%) and trisomy 11 (90%). \textit{KMT2A}-PTD has been associated with shorter OS and a poorer prognosis compared with CN-AML without the \textit{KMT2A}-PTD.\textsuperscript{6,7}

\textbf{NPM1}

\textit{NPM1} encodes a nucleolar protein implicated in multiple cellular functions. Mutations in \textit{NPM1} are usually small insertions (4-11 bp in size) that result in a frameshift during translation and aberrant cytoplasmic localization of the NPM1 protein.\textsuperscript{6} Mutations in the \textit{NPM1} are among the most common genetic changes in AML (occurring in 25%-35% of patients), especially in CN-AML (present in 45%-64%).\textsuperscript{4}

Almost 40% of \textit{NPM1}-mutated AML patients have \textit{FLT3}-ITD mutations. AML patients with an \textit{NPM1} mutation and \textit{FLT3} wild-type have better complete remission, event-free survival, and OS compared with patients with \textit{NPM1} and \textit{FLT3} mutations. However, patients with mutated \textit{FLT3}-ITD and mutated \textit{NPM1} have better prognosis that patients with mutated \textit{FLT3}-ITD and wild-type \textit{NPM1}.\textsuperscript{7}

\textit{NPM1} mutations have been associated with chemosensitivity to intensive chemotherapy in both young and old patients, which may account for improved outcomes. The pattern of mutations largely shapes clinical outcomes. Patients who have mutated \textit{NPM1} without the \textit{FLT3}-ITD genotype have a comparatively better outcome than those with coexisting \textit{FLT3}-ITD mutations and hence are no longer recommended for transplantation during their first complete remission.\textsuperscript{2,4,5}

\textbf{RUNX1}

\textit{RUNX1} is a transcription factor regulating hematopoiesis and myeloid stem cell differentiation into mature cells. Somatic mutations of \textit{RUNX1} have been identified in 5% to 20% of AML. \textit{RUNX1} mutations are associated more with older patient age, normal karyotype AML, and \textit{KMT2A} and \textit{IDH} mutations. \textit{RUNX1} mutations are mutually exclusive with \textit{NPM1} and \textit{CEBP4} mutations but tend to co-occur with \textit{ASXL1}, \textit{SRSF2}, \textit{IDH2}, and \textit{KMT2A}-PTD. \textit{RUNX1} mutations are associated with high-risk disease and a poorer prognosis in patients with AML.\textsuperscript{7}

\textit{RUNX1} mutations in AML are associated with poor outcomes, which contrast with the favorable prognostic effect of gene fusions involving \textit{RUNX1}. \textit{RUNX1} mutations are associated with resistance to standard induction therapy with inferior survival for both younger and older patients. \textit{RUNX1} mutations were associated with inferior survival.\textsuperscript{4,9}

\textbf{TET2}

\textit{TET2} is a key regulator in hematopoietic stem cell renewal and differentiation. \textit{TET2} mutations are loss-of-function mutations and result in increased self-renewal of stem cells, myeloid hyperplasia, and impaired differentiation. \textit{TET2} mutations are seen in 7% to 25% of AML. \textit{TET2} mutations co-occur with mutations of \textit{EZH2} and \textit{IDH2} mutations and are commonly associated with a normal karyotype in AML.\textsuperscript{4,7}

The prognosis for AML patients is not yet clear. However, patients with \textit{TET2} mutations show a greater response to azacytidine compared with those with wild-type \textit{TET2}.\textsuperscript{4,7}

\textbf{TP53}

\textit{TP53} is a tumor suppressor gene that plays a critical role in cell cycle regulation, DNA repair, and apoptosis. P53 protein binds directly to DNA and determines whether the cell undergoes repair, senescence, or apoptosis in response to cellular stress or damage. Missense mutations are quite common (70%-80%), resulting in impaired function and facilitating the evolution of malignant neoplasms. \textit{TP53} mutations are present in 5% to 18% of de novo AML. In cases of AML with a complex karyotype, the frequency of \textit{TP53} mutation is 50%.\textsuperscript{4}

In general, \textit{TP53} mutations confer an adverse prognosis with documented chemoresistance. \textit{TP53} mutations may be mainly responsible for the very poor prognosis of complex karyotype AML.\textsuperscript{4} \textit{TP53} mutations are independently associated with shorter OS and resistance to chemotherapy, hypomethylating agents, and stem cell transplantation in patients with myelodysplastic and AML.\textsuperscript{6} Patients in the subgroup AML with \textit{TP53} mutations, chromosomal aneuploidy, or both, are significantly older and more frequently have secondary AML and poor outcomes.\textsuperscript{20}
Treatment of AML should be sufficiently aggressive to achieve a complete remission (CR), because partial remission offers no substantial survival benefit.

Current treatment for AML involves 2 phases. The first, induction therapy, is an intensive chemotherapeutic regimen that attempts to eradicate the leukemia and normalize blood counts. Treatment with 7 days of cytarabine and 3 days of an anthracycline (“7 + 3” regimen) remains the current standard for remission-induction therapy. For patients with FLT3-mutated AML, midostaurin, an inhibitor of FLT3-ITD and FLT3-TKD mutations, may be added to the chemotherapy regimen. Gemtuzumab ozogamicin (GO) is a humanized anti-CD33 IgG3 antibody conjugated to the cytotoxin calicheamicin. CD33 is a transmembrane glycoprotein frequently expressed on adult and childhood AML blasts (85%-90% of patients presenting with AML). GO binds to the surface CD33, and the complex is internalized. Patients shown to have high CD33 expression may be given GO as part of the induction regimen. The presence of greater than 5% of leukemic blasts necessitates induction therapy.

The goals of treatment are to eradicate the disease as quickly as possible and induce complete remission, often at the cost of other aspects of the patient’s health. The traditional chemotherapeutic regimen is associated with a number of side effects that range from unpleasant to life threatening, including alopecia (hair loss), mucositis (sores in the mouth and intestines), organ damage, and myelosuppression, which may lead to deadly infections.

Advances in the treatment of AML have resulted in substantially improved CR rates. Approximately 60% to 70% of adults with AML can be expected to attain CR status following appropriate induction therapy, depending on patient age and the presence or absence of specific somatically acquired genetic alterations. Remission rates in adult AML are inversely related to age, with an expected remission rate of more than 65% for those younger than 60 years.

The second phase of treatment, post-remission therapy or consolidation, involves chemotherapy, possibly allogeneic HSCT (alloHSCT), or both. Midostaurin or GO may be added to the chemotherapy regimen. For patients with FLT3-mutated AML, the current standard for remission-induction therapy.

Table 2. WHO Classification of AML and Related Neoplasms

<table>
<thead>
<tr>
<th>WHO Classification of AML and Related Neoplasms</th>
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<tbody>
<tr>
<td>AML with recurrent genetic abnormalities</td>
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<tr>
<td>AML with t(8;21)(q22;q22.1); RUNX1-RUNX1T1</td>
</tr>
<tr>
<td>AML with inv(16)(p13.1q22) or t(16;16)(p13.1;q22); CBFB-MYH11</td>
</tr>
<tr>
<td>APL with PML-RARA</td>
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<tr>
<td>AML with t(9;11)(p21.3;q23.3); MLLT3-KMT2A</td>
</tr>
<tr>
<td>AML with t(6;9)(p23;q34.1); DEK-NUP214</td>
</tr>
<tr>
<td>AML with inv(3)(p21.3q26.2) or t(3;3)(p21.3q26.2); GATA2, MECOM</td>
</tr>
<tr>
<td>AML with mutated RUNX1</td>
</tr>
<tr>
<td>AML with biallelic mutations of CEBPA</td>
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<tr>
<td>Provisional entity: AML with mutated RUNX1</td>
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<tr>
<td>AML with myelodysplasia-related changes</td>
</tr>
<tr>
<td>Therapy-related myeloid neoplasms</td>
</tr>
<tr>
<td>AML, not otherwise specified</td>
</tr>
<tr>
<td>AML with minimal differentiation</td>
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<tr>
<td>AML without maturation</td>
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<tr>
<td>AML with maturation</td>
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<tr>
<td>Acute myelomonocytic leukemia</td>
</tr>
<tr>
<td>Acute monoblastic/monocytic leukemia</td>
</tr>
<tr>
<td>Pure erythroid leukemia</td>
</tr>
<tr>
<td>Acute megakaryoblastic leukemia</td>
</tr>
<tr>
<td>Acute basophilic leukemia</td>
</tr>
<tr>
<td>Acute panmyelosis with myelofibrosis</td>
</tr>
<tr>
<td>Myeloid sarcoma</td>
</tr>
<tr>
<td>Myeloid proliferations related to Down syndrome</td>
</tr>
<tr>
<td>Transient abnormal myelopoiesis (TAM)</td>
</tr>
<tr>
<td>Myeloid leukemia associated with Down syndrome</td>
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</table>

Abbreviations: AML, acute myeloid leukemia; WHO, World Health Organization.
than 25% of adults with AML can be expected to survive 3 or more years. The 5-year survival rate for people with AML is approximately 27%.  

Improvements in therapeutic regimens and supportive care (including infection control and transfusion support) have led to improved survival for AML. However, relapse, and the associated resistance to currently available therapies, represents one of the central problems in the treatment of AML. 

ECONOMIC IMPACT OF AML

There are few published articles that have examined the cost burden of AML specifically. One study published in 2010 set the cost of induction therapy at $63,000. In another study, health care costs and utilization during the first year after a diagnosis of AML for privately insured non-Medicare patients in the United States aged 50 to 64 years who were treated with either chemotherapy or chemotherapy and alloHCT were estimated based on MarketScan (Truven Health Analytics) adjudicated total payments for inpatient, outpatient, and prescription drug claims from 2007 to 2011. Adjusted mean 1-year costs were $280,788 for chemotherapy and $544,178 for alloHCT. 

In a more recent study, AML patients were identified in MarketScan claims databases between 1 January 2009 and 31 January 2015. Mean (SD) health care expenditures for patients from first-line induction to remission (n = 681) were $208,857 ($152,090). Of the patients who had a second remission (n = 70, expenditures from relapse to remission were $142,569 ($208,307). 

Given the need to hospitalize the patient upon diagnosis, the driver of costs would be related to hospital-based costs and physician payments. Once induction therapy is complete, the costs then shift to outpatient costs for drugs and laboratory as we see with many other cancers. Like many other cancers there are indirect costs and burdens; some of which can be considerable.

DEFINING VALUE IN AML CARE

In addition to the run up in costs, and the pressures of newly-approved drugs and others in the pipeline that are expected to come to market, the existing drugs will gain new indications and will be prescribed more frequently. Treatment cycles will extend beyond current recommendations. Many drugs will also be used in post-acute treatment maintenance. There is also the issue of off-label or outside-of-approval use of therapies. An important question to ask is: Where is the evidence of success? How do we evaluate and determine the value of extending treatment? This creates several legal and ethical questions around balancing the appropriate use of agents with the perception (often cited by advocates) of care.

Managed care needs to appreciate the expanded indications of existing drugs and how this changes the standard of care, whether that be more frequent or longer use. Our biggest source of concern is the off-label use of biologics, determining what level of evidence is acceptable in order to allow this use, and what issues, if any, are created by this use. As treatments evolve and new lines of therapy are added, we are seeing treatments designed to maintain the patient beyond the acute phase of treatment. This is another area where organizations will need to appreciate the value of these therapies.

I think we can all agree that the treatment of cancer is very complex and expensive. Managed care organizations struggle to find the right benefit design that allows the patient to access the care that they need at a price that is affordable to both the patient and society. The Institute of Medicine (now part of the National Academy of Medicine) has described quality medical care as patient-centered, safe, timely, equitable, effective, efficient, and sustainable. To me, “efficient and sustainable” means that cost is central to quality. Cost is also central to achieving an equitable distribution of health care with timely access. Organizations attempt to provide this by balancing and developing coverage and benefit language that enables access while controlling utilization and managing costs in order to ensure the organizations survival. Challenges are plentiful, and companies have well-defined mechanisms for decision-making and implementation. Organizations are always looking for new and more efficient models of care not only to control costs by also to improve quality.

In addition, patients should not be excluded from this discussion. What patients really want is to build a partnership with their treatment team. They want access to quality care with compassion and respect. While all of these domains are very important, what is most important is that patients and their families want hope. Partnering requires that clinicians actively interact with patients, provide them with the necessary medical information to make an informed decision and engage the patient in both the conversation and decision making. In short, a working partnership of patient and clinician provides the foundation for patient engagement and empowers the patient to be the steward of their own health care.

Is there a place here for a collaborative practice such as the medical home? While many of the services are similar to what oncology practices currently offer could an organized medical team with highly defined responsibilities and workflows provide efficient, patient-centered care that improves outcomes, lowers treatment costs, and provides an enhanced patient experience? Although the concept of the medical home has been around since the 1960s, it has in the past decade been adopted in many primary care settings. In oncology, the care would be directed by the patient’s oncologist who would lead the team depicted here, who collectively take responsibility for the care of the patient. Each team member
would apply his or her expertise toward improving the overall health status of the patient. The major challenge to this approach is in setting the correct reimbursement levels to support this type of practice.  

Managed care organizations can contribute to improved outcomes and reduced costs by increasing the understanding of current therapies, recognizing the potential benefit of future therapies, and developing an understanding of targeted drugs and their place in therapy.

References