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Leukemia insights

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Novel Therapeutic Options for Elderly AML patients

INTRODUCTION

Despite steady progress in the therapy of AML in younger patients, the outcomes in elderly AML have not improved significantly over the past four decades (Kantarjian et al, Cancer 2006; 106:1090-1098). A number of factors including an inherently resistant disease phenotype, increased incidence of drug resistance, frequent co-morbidities and poor tolerance to standard cytotoxic therapy contribute to the poor outcomes in elderly AML patients (Kantarjian HM, Cancer 2007;109:1007-1010). The 4-8 week mortality with intensive chemotherapy is 30%-50% in these patients, and the median survival is 4-7 months, indicating the need for alternate therapeutic strategies. In this Leukemia Insights, we update our current strategies for elderly patients with de-novo AML, those with therapy-related AML, AML arising from prior MDS, core-binding factor AML, and FLT3-, RAS-, and IDH- mutated AML.

In addition to traditional risk factors such as age, cytogenetics, and performance status, factors such as molecular mutations have emerged as potential modulaters of response to therapy. Recent data suggest that patients with mutations involving the epigenetic regulators of DNA-methylation (including DNMT3A, IDH1, IDH2, TET2) and/or mutations involving chromatin structure regulators (including EZH2, ASXL1) may have an increased propensity to respond to hypomethylating agents (decitabine and azacitidine). To this end, we routinely perform mutational analysis in AML and high-risk MDS prior to treatment, at the time of disease progression (MDS to AML) and at relapse. First-line testing is done with the Cancer Mutation Scan-28 Genes CLIA panel. Similarly, samples of patients with exceptional sensitivity or resistance to a particular regimen will be referred to the MD Anderson Institute for Personalized Cancer Therapy for whole-genome sequencing to

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identify novel lesions. These studies may lead to discovery of new mutations rendering AML cells sensitive to or resistant to individual therapies. Furthermore, identification and targeting of recurrent mutations may allow us to improve response rates. We currently have clinically active agents that are capable of targeting FLT3 (both ITD and D835), NRAS/KRAS and IDH1/IDH2 in patients with AML.

De-novo AML	Secondary AML including AML with pre-existing AHD	Core-binding factor AML	Lower-intensity regimens for unfit/ unable to tolerate standard regimens	FLT3-mutated AML	RAS-mutated AML	IDH-mutated AML
Vosaroxin +Decitabine	CPX351 vs. 3+7	FLAG-Ida	Hedgehog + LD Ara-C	AC220 +Azacitidine	Trametinib +AKT inhibitor	ABT-199
Cladribine + LD Ara-C alternating with Decitabine	Hedgehog + LD Ara-C		LD Ara-C +/-Volasertib	Crenolanib		AG-221
SGI-110	Lintuzumab + LD Ara-C		Decitabine 5 days vs. 10 days	ASP2215		
Decitabine 5 days vs. 10 days				Azacitidine +Sorafenib		
Azacitidine +Pracinostat						

De-novo AML

VOSAROXIN + DECITABINE

Vosaroxin is a first-in-class anti-cancer quinolone derivative (AQD), a class of compounds that has not been used previously for the treatment of cancer. The pivotal Phase III, Randomized, Controlled, Double-Blind, Multinational Clinical Study of the Efficacy and Safety of Vosaroxin and Cytarabine Versus Placebo and Cytarabine in Patients With First Relapsed or Refractory Acute Myeloid Leukemia (VALOR) (Ravandi et al, J Clin Oncol 30, 2012 suppl; abstr TPS6637) has completed accrual (n=712), and final results will be presented at American Society Hematology meeting in December. Vosaroxin induced durable remissions and was well tolerated in 3 dosing schedules. Vosaroxin and decitabine have non-overlapping primary molecular mechanisms of action. Our trial was the first of its kind to combine vosaroxin and decitabine, which appear to have synergism without additive toxicity. Initial results of the single arm, open-label study of the combination in previously untreated patients with AML/high-risk MDS who are 60 and older are encouraging and were presented last month at the American Association for Cancer Research meeting.

CLADRIBINE + LOW-DOSE CYTARABINE (LD ARA-C) ALTERNATING WITH DECITABINE

We have previously demonstrated that the combination of clofarabine and low-dose cytarabine achieves high response rates with low induction mortality in elderly patients with previously untreated AML (Faderl et al, Blood ASH Annual Meeting Abstracts 2009 114: Abstract 2058).

Cladribine is a purine analog that modulates deoxycytidine kinase and is thought to possess more specific activity against myeloid blasts than clofarabine. Cladribine has been shown to improve survival when combined with cytarabine. The combination of cladribine and LD Ara-C alternating with decitabine is well tolerated with no 4-week mortality and no treatment-related grade 3/4 non-hematologic adverse events. The CR rate is 58%, and the overall response rate (CR+CRp+PR) is 67%. Initial results demonstrate a 1-year overall survival rate of 51% (Kadia et al, Blood November 15, 2013 vol. 122 no. 21 5011). These results compare favorably to previously published data on outcomes of elderly AML patients, including those with unfavorable risk features. The results from this study will be updated at the American Society of Clinical Oncology meeting in June.

SGI-110

SGI-110 is a second-generation hypomethylating agent formulated as a dinucleotide of decitabine and deoxyguanosine. It is delivered as a small-volume SQ injection allowing longer half-life and more extended decitabine exposure than decitabine IV infusion. SGI-110 produced potent hypomethylation and clinical responses in MDS and AML patients previously treated with first-generation hypomethylators (Kantarjian et

al, Blood ASH Annual Meeting Abstracts 2013 120: Abstract 414). A phase II, open label, multi-center study to determine the biologically effective dose of SGI-110 included patients with relapsed/refractory AML or elderly treatment-naïve AML patients who were not suitable for induction chemotherapy. SGI-110 was well tolerated with complete remissions observed in 16% of the relapsed/refractory patients and 53% in the treatment-naïve patients (Kantarjian et al, Blood ASH Annual Meeting Abstracts 2013: Abstract 497). These data compare favorably with previous results reported for hypomethylating treatment and have resulted in the activation of a phase III pivotal study of SGI-110 in elderly AML patients. The study is accruing patients, and is an especially attractive option for patients older than 70 years or those who have poor performance status or reduced organ function.

DECITABINE 5 DAYS VERSUS 10 DAYS

The standard dose for decitabine in elderly AML patients is 20 mg/m2 daily x 5 days (Kantarjian et al, JCO 2012 Jul 20; 30:2670-7). A study conducted by Ohio State University demonstrated a significantly higher response rate with decitabine 20 mg/m2 daily x 10 days (Blum et al, Proc Natl Acad USA; 2010 Apr 20;107:7473-8). The objective of our current study is to randomize patients to receive the standard 5-day schedule versus the novel 10-day schedule to identify whether the extended schedule produces an improved response rate without significant additive toxicity. To better elucidate the mechanism of action of decitabine, we will examine the correlation of numerous biological markers with response to decitabine therapy, including global and gene-specific methylation, somatic gene mutations, micro-RNA, and immune effector function.

AZACITIDINE + PRACINOSTAT

Abnormal epigenetic silencing of important regulatory genes via deacetylation of their histone residues has been described in AML and MDS. Histone deacetylase inhibitors capable of reversing histone deacetylation have been extensively studied in hematologic malignancies. Pracinostat is a rationally designed, potent, oral, pan-histone deacetylase inhibitor (Wang et al, J Med Chem 2011; 54:4694-4720). In vitro studies have demonstrated that azacitidine in combination with pracinostat synergistically increases expression of silenced tumor suppressors and promotes cell death. Our study is designed to evaluate the efficacy and safety of the combination of azacitidine and pracinostat in elderly patients with AML.

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Secondary AML including AML with pre-existing antecedent hematologic disorder (AHD)

Secondary AML is a term that has been used to cover a heterogeneous group of poor prognosis AML

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CLL Treatment Priorities

1. Untreated

- Fludarabine + Cytoxan + Rituximab (FCR) (2008-0431)
- Ofatumumab (2010-0241/2011-0520)
- Lenalidomide + Rituximab (2011-0509)
- CAL-101 + Rituximab (2011-0612)
- TRU-016 + Rituximab (2012-0626)
- PCI-32765 vs. Chlorambucil (2012-1007)

2. Prior Therapy

- FBR (2009-0546)
- Sapacitabine + Cytoxan + Rituximab (2010-0516)
- AVL-292 (2011-0513)
- ABT-199 (2011-0164)
- Lenalidomide + Rituximab (2011-0509)
- CD19 CAR (2011-1169)
- GS-1101 + Rituximab (2012-0171)
- GS-1101 (2012-0411)
- GS-9973 + Idelalisib (2013-0319)
- Ublituximab + TGR-1202 (2013-0566)
- Ibrutinib +/- Rituximab (2013-0703)
- ACP-196 (2013-0907)

3. Minimal Residual Disease

- Revlimid (2007-0213)

4. Hairy Cell

- 2CDA + Rituximab (2004-0223)
- Moxetumomab (2013-0302)
- PCI-32765 (2013-0299)

AML/MDS Treatment Priorities

1. Newly Diagnosed

A. Acute Promyelocytic Leukemia: cytogenetic feature: t(15;17):
   - ATRA + Arsenic +/- Gemtuzumab (2010-0981)

B. Cytogenetic feature: Inv16 or t(8:21): Fludarabine + Ara-C + Idarubicin (2007-0147)

C. Younger Patients:
   - CIA vs FAI (2010-0788)
   - 3 + 7 vs IA+Vorinostat (S1203)
   - PF-04449913 with 3 + 7 (2012-0062)

D. Older Patients:
   - Sapacitabine (2007-0727)
   - DAC +/- Clofarabine (2008-0092)
   - Vorinostat + Aza (2007-0685)
   - Plerixafor + Clofarabine (2009-0536)
   - SGI-110 (2010-0615/2013-0843)
   - Sapacitabine vs. DAC vs. Both (2010-0727)
   - Omacetaxine + LD Ara-C (2010-0736)
   - Cladribine + LD Ara-C/DAC (2011-0987)
   - PF-04449913 with LD Ara-C or DAC (2012-0062)
   - LD Ara-C + Lintuzumab (2012-0434)
   - CPX-351 vs. Ara-C + Dauno (2012-0980)
   - DAC 5 vs. 10day (2012-1017)
   - Vosaroxin + DAC (2013-0099)
   - LD Ara-C +/- Volasertib (2013-0416)
   - Aza + Sorafenib (2014-0076)

2. Salvage Programs

- Plerixafor + Sorafenib (2008-0501)
- IA + E7070 (2009-0570)
- Ara-C +/- Vosaroxin (2010-0692)
- MK-8242 (2011-0547)
- Erlotinib (2012-0060)
- Clofarabine + LD Ara-C (2011-0660)
- Crenolanib (2012-0569)
- BL-8040 (2012-1097)
- AC220 + Aza or Ara-C (2012-1047)
- Trametinib + GSK2141795 (2013-0001)
- Rigosertib + Aza (2013-0030)
- Birinapant + Aza (2013-0141)
- Eltrombopag (2013-0225)
- Pracinostat + Aza (2013-0321)
- WT 2725 (2013-0404)
- Vismodegib (2013-0433)
- Volasertib + DAC (2013-0583)
- ABT-199 (2013-0656)
- Brentuximab +/- Aza (2013-0706)
- IGN 523 (2013-0971)

3. Low Risk MDS and CMML with <10% Blasts

- Vorinostat + Aza (2007-0685)
- Deferasirox (2010-0041)
- DAC vs. Aza (2012-0507)
- Horse ATG (2012-0334)
- Sotatercept (2012-0428)
- Bortezomib (2012-0562)
- Rigosertib (2012-0598)
- Oral Aza vs. Placebo (2012-0733)
- Ruxolitinib (2013-0012)
- MK-3475 (2013-0531)
- Pracinostat + DAC or Aza (2013-0873)

4. MDS/MPN

- Ruxolitinib + Aza (2012-0737)

5. Maintenance/MRD

- Oral Aza vs. Best Care (2012-0866)

ALL Treatment Priorities

1. Newly Diagnosed or Primary Refractory

(one non-hyper-CVAD induction)

A. Age <40:Augmented BFM (2006-0375)

B. Age >60: Marquibo (2011-1071)
   - Low dose Hyper CVD + CMC-544 (2010-0991)

C. Hyper CVAD + Ofatumumab (2010-0708)

D. Hyper CVAD + Liposomal Vincristine (2008-0598)

E. T cell: Hyper CVAD + Nelarabine (2006-0328)

2. Salvage Programs

- A-dmDT390-bis Fv (2008-0077)
- Low Dose Hyper CVAD + CMC-544 (2010-0991)
- Rituximab (2011-0844)
- BMS-906024 (2011-0382)
- Blinotumimab (2011-0784)
- Inotuzumab Ozogamicin (2012-0151/2013-0144)
- MOR 00208 (2012-0904)
- Brentuximab (2012-1106)
- Moxetumomab (2012-1143)
- Ibrutinib (2013-0459)

3. CNS Disease

- Intrathecal Rituximab (2011-0844)

CML Treatment Priorities

1. CML Chronic Phase

- Dasatinib (2005-0422)
- Nilotinib (2005-0048)

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2. TKI Failures, T315I Mutations or Advanced Phases

- Dasatinib + DAC (2011-0333)

3. Minimal Residual Disease

- Ruxolitinib (2012-0697)

Myeloproliferative Disorders

1. Myelofibrosis

- NS-018 (2011-0090)
- Sotatercept (2012-0534)
- Ruxolitinib + Aza (2012-0737)
- PRM-151 (2013-0051)
- Momelotinib vs. Ruxolitinib (2013-0741)

2. Systemic Mastocytosis

- Masatinib (2008-0275)
- Brentuximab (2012-0734)

Phase I/II Agents for Hematologic Malignancies

- L-Grb2 Antisense (2003-0578)
- Nelarabine (2009-0717)
- KB004 (2010-0509)
- BKM120 (2010-0874)
- CWP232291 (2011-0253)
- AMG900 (2011-0369)
- PRI-724 (2011-0527)
- AZD1208 (2011-0816)
- DFP-10917 (2012-0262)
- EPZ-5676 (2012-0374)
- DAC + CIA (2012-1064)
- GSK525762 (2013-0527)

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arising in a setting of prior treatment with cytotoxic agents or large field radiation therapy and/or AHD. Specifically included are patients with treatment-related AML, those with documented pre-existing myelodysplasia and/or CMML, and patients with de novo AML with specific chromosomal abnormalities linked to myelodysplasia per WHO criteria.

CPX-351 VERSUS 3+7

CPX-351 is a liposomal formulation of a fixed combination of the antineoplastic drugs cytarabine and daunorubicin. CPX-351 markedly prolongs plasma drug levels and maintains the 5:1 molar ratio for optimal leukemic cell killing. In the initial phase II study CPX-351 produced superior rates of leukemic-free marrow and response with similar duration of remission (Lancet et al, Blood; March 2014; Epub ahead of print). Observations of reduced early mortality and a significant survival advantage (HR=0.41, P=0.02) among secondary AML patients suggest that clinical benefit is likely for this patient group (Lancet et al, ASCO Annual Meeting Abstracts 2011, J Clin Oncol 29, abstract 6519). The ongoing phase III study will be a direct test of whether molar ratio controlled drug delivery can improve antitumor efficacy in secondary AML.

HEDGEHOG INHIBITOR (PF-04449913) + LOW-DOSE CYTARABINE (LD ARA-C)

Hedgehog and Gli signaling (Hh-

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Gli) are critical pathways that have been implicated in both hematapoietic and solid tumor malignancies by diverse mechanisms, including direct cell cycle and anti-angiogenic effects (Thomas BJ, Dev Cell, 2005; Straface G, et al, J Cell Mol Med, 2008). Aberrant Hh signaling has been described in a variety of human leukemia and leukemia stem cells (Bai et al, Leukemia, 2008). PF-04449913 is a novel small molecule inhibitor of the Sonic Hedgehog Pathway being developed for the treatment of hematologic malignancies and solid tumors. This is a Phase IB/II, open label, international, multi-center, safety and efficacy study of PF-04449913 in combination with intensive chemotherapy (cytarabine and daunorubicin), LD Ara-C, or decitabine in previously untreated patients with AML or high-risk MDS. Newly diagnosed and previously untreated patients are eligible, as well as patients with AHD. Elderly patients will be randomized to receive PF-04449913 in combination with LD Ara-C or decitabine.

LINTUZUMAB + LD ARA-C

Lintuzumab is an immunoconjugate consisting of the alpha particle-emitting isotope 225Actinium (225Ac) and the humanized anti-CD33 monoclonal antibody HuM195 (Jurcic J et al, Proc Am Soc Clin Oncol, 2000; Jurcic J et al, Cancer Res, 1995). Radioimmunotherapy with alpha-particle-emitting anti-CD33 monoclonal antibody HuM195 has demonstrated significant anti-leukemic effects with potentially reduced extramedullary toxicity as compared to conventional systemic AML therapy (Caron PC et al, Blood, 1994; Feldman E et al, Leukemia, 2003; Jurcic J et al, Clin Cancer Res, 2000). The current protocol is designed to assess the safety and efficacy of LD Ara-C followed by a fractionated dose of Lintuzumab-Ac225 in elderly patients with AML who are deemed unfit for intensive chemotherapy including patients with AHD.

Core-binding factor (CBF) AML

The combination of anthracycline and high-dose cytarabine in older (age ≥ 60) patients with CBF-AML produced complete remission rates > 85% but was associated with approximately 10% early treatment-related mortality (Prebet, Boissel et al. JCO, 2009). Our previous study showed that fludarabine and cytarabine based induction regimens were highly effective in CBF-AML in patients of all ages (Borthakur, Kantarjian et al. Cancer, 2008). In a recent update presented at ASH 2013 we noted that induction therapy with fludarabine and cytarabine based combination regimen continues to be highly effective (ORR 98%) in patients ≥ 60 years, provides durable remissions and is associated with low treatment-related mortality (3% in 4 weeks, 6% in 8 weeks) (Borthakur et al, ASH Annual Meeting Abstracts 2013). The protocol continues to accrue well and is an excellent option for patient's ≥ 60 years with CBF-leukemia.

Lower-intensity regimens for unfit/unable to tolerate standard regimens

HEDGEHOG INHIBITOR (PF-04449913) + LD ARA-C

The phase II portion of the previously described Hh inhibitor + LD Ara-C protocol includes an arm in fit patients as well as a randomized component in unfit patients, defined as patients who have one or more of the following: age ≥ 65 years, ECOG of 2, creatinine ≥ 1.3 or LVEF < 45%. The goal of the phase II unfit arm is to compare the overall survival of the Hh inhibitor with LD Ara-C versus LD Ara-C alone in previously untreated AML or high-risk MDS.

LD ARA-C +/- VOLASERTIB

Published preclinical and clinical data suggest that Polo-like kinase 1 (Plk1) is a potentially interesting therapeutic target for the treatment of AML (Mueller-Tidow C et al, ASH Annual Meeting Abstracts 2008; Renner et al, Blood 2009). Volasertib (BI 6727) is a first in class, highly selective and potent cell cycle kinase inhibitor targeting Plk1. In a phase I/II trial volasertib in combination with LD Ara-C had a higher remission rate (31%) than LD Ara-C monotherapy (11%) in patients with previously untreated

AML considered ineligible for intensive treatment. Furthermore a trend in event-free survival was seen in favor of volasertib + LD Ara-C. The primary objective of the present randomized, double-blind, phase III trial open at MDACC is to evaluate whether patients ≥ 65 years of age with previously untreated AML have higher response rates with volasertib and subcutaneous LD Ara-C as compared to placebo and LD Ara-C. This trial includes patients who are deemed ineligible for intensive induction chemotherapy or traditional clinical protocols due to poor performance status, concomitant diagnosis, or organ dysfunction.

FLT3 mutated AML

AC220 (QUIZARTINIB) + AZACITIDINE OR LD ARA-C

Activating gene mutations in FLT3 (ITD and D835) are present in approximately 30% of patients with AML. FMS-like tyrosine kinase 3 internal tandem duplications (FLT3-ITD) in acute myeloid leukemia are associated with early relapse after standard chemotherapy and poor survival. AC220 is a novel second-generation Class III RTK inhibitor with potent and highly efficacious FLT3 activity in vitro and in vivo (Cortes J et al, ASH Annual Meeting Abstracts 2010). Single agent therapy with AC220 produced an overall response rate (CR + PR) of approximately 50% in the FLT3 mutated patients with relapsed/refractory disease (Cortes J et al, ASH Annual Meeting Abstracts 2013). Similarly, AC220 has successfully been combined with intensive chemotherapy (3+7) in patients aged 18-60 years (Altman J et al, ASH Annual Meeting Abstracts 2013). The current phase II study is open to patients of all ages (including those ≥ 60 years of age) and will help determine the clinical activity of the combination of AC220 with either Azacitidine or LD Ara-C in patients with AML or MDS.

CRENOLANIB

Crenolanib besylate (CP-868,596-26) is an orally bioavailable, selective tyrosine kinase inhibitor (TKI) of both wild-type FLT3 as well as FLT3 with activating mutations including FLT3-ITD and FLT3-D835Y/H mutants (Ramachandran A et al, AACR abstracts 2012; Galanis A et al, AACR abstracts 2012). In a phase I study crenolanib was well tolerated as a single agent (Lewis N et al, JCO 2009). The current Phase II study is designed to evaluate the efficacy and tolerability of crenolanib in AML patients with FLT3 activation mutations including patients whose leukemia has recurred after prior chemotherapy not including a FLT3 inhibitor and patients whose leukemia has progressed after prior therapy with a FLT3 inhibitor.

ASP2215

ASP2215 hemifumarate is a new chemical entity with an inhibitory effect on tyrosine kinases, mainly FLT3, Axl and ALK. Axl overexpression in AML is associated with drug-resistance and adverse prognosis (Hong C et al, Cancer Lett 2008; Rochlitz C et al, Leukemia 1999). Axl inhibition suppresses the growth of human FLT3-positive AML in vivo (Park I et al, Blood 2013). ASP2215 demonstrated favorable efficacy in non-clinical AML models, including mouse xenografts and human AML cell lines. ASP2215 inhibits the growth of FLT3 wild-type, FLT3-ITD and FLT3-D835 cells. We are opening a phase I/II dose-escalation study of ASP2215 in patients with relapsed or refractory AML. Patients with FLT3 mutations (ITD and D835) who have failed prior FLT3-inhibitors would be eligible for this protocol.

RAS- and IDH-targeted therapy

We currently offer targeted therapy for patients with AML harboring specific molecular mutations including: RAS (K/N) mutations: Trametinib + AKT inhibitor, patients with IDH1/2 mutations: ABT-199 and patients with IDH2 mutations AG-221.

CONCLUSION

As we learn more about the risk factors and disease characteristics in the Elderly AML population, new treatment opportunities become available. For information about our program and therapeutic options for Elderly AML, or any Leukemia protocol, contact Navel Daver, Jorge Cortes, or any Leukemia physician.

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