STANFORD CANCER CENTER | UCDAVIS COMPREHENSIVE CANCER CENTER | UCSF Helen Diller Family Comprehensive Cancer Center | Association of Northern California Oncologists

Clinical Case Vignettes

Association of Northern California Oncologists (ANCO) Hematologic Malignancies Update

September 9, 2017

Brian Sworder MD, PhD, fellow (Stanford)
bsworder@stanford.edu

Disclosures

- Nothing to disclose
- I have attempted to highlight “off-label” use if applicable in cases

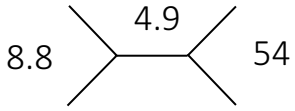
Outline of Clinical Case Vignettes

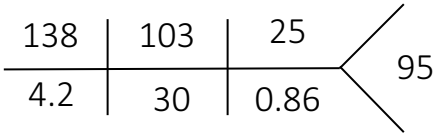
- 2 clinical cases with multiple scenarios highlighting common treatment and management issues in hematologic malignancies
- Take away “learning points” highlighted in each case
- Audience questions welcome at any time, expert opinion on each case
- Feel free to discuss your own variations of cases with our experts

Case 1 – 62 year old female

- HPI: Initially presents to her primary care physician complaining of progressive dyspnea; found to be severely anemic and thrombocytopenic. She is admitted for transfusion and hematology is consulted.
- Past Medical History: No significant past medical history
- Past Surgical History: Tonsillectomy
- Social History: Works actively as a school teacher. No smoking. No EtOH.
- Family History: Father and grandfather had head and neck cancer, both smokers. Sister with cervical cancer.
- Meds: None
- Exam – Fit woman in no acute distress. Essentially unremarkable. Excellent performance status.

Case 1 – Initial Labs





MCV - 96.5
ANC - 1400
Segs - 11%
Bands – 5%
Lymph – 17%
Mono – 3%
Blasts – 64%

AST – 21
ALT – 24
Alk Phos – 70
T. Bili – 0.8

LDH - 261

Smear: Numerous blasts, no hemolysis

Case 1 – Bone Marrow

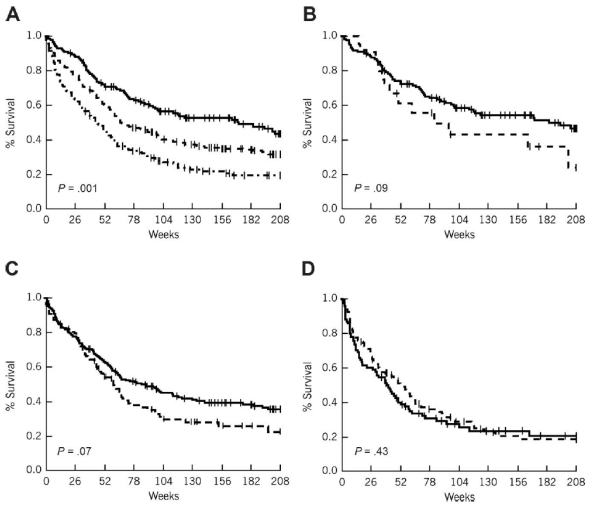
- Bone Marrow Aspirate and Biopsy
  - Acute myeloid leukemia (88% blasts)
  - Reduced trilineage hematopoiesis
  - Bone marrow, aspirate, flow cytometry
    - Abnormal CD34+ blast population, 85%, expressing dim CD45, partial dim cytoplasmic CD3, CD7, partial CD4, dim CD38, dim CD33, partial dim CD13, partial dim CD10, HLA-DR, partial CD117 and partial dim CD15 without MPO expression.
    - Heterogeneous B, T and NK cells without aberrant antigen expression or B cell monotypia.
  - **Cytogenetics and molecular studies sent and pending**

Case 1 – Initial Management

What should be the next step in management?

a) Cytoreduction with hyroxyurea
b) Immediately start induction chemotherapy with 7+3 (Cytarabine + Anthracycline)
c) Start treatment with a hypomethylating agent
d) Send rapid sequencing panel for *FLT3* and *NPM1* mutations; await start of treatment until this and cytogenetics return.
e) Next generation sequencing myeloid gene panel

Case 1 – Impact of delay prior to starting treatment



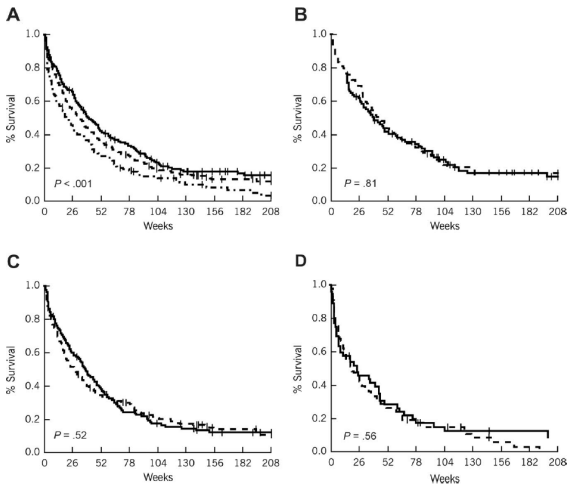
Survival decreased in patients < 60 years old for whom treatment is delayed by greater than or equal to five days after diagnosis.

Figure 2. Survival in patients less than 60 years of age by risk group and interval from diagnosis to treatment. (A) Survival by risk group. ------- indicates favorable (age ≤ 40 and de novo AML); - - - -, intermediate (age ≤ 40 and secondary AML, or age > 40 and de novo AML); and - - - - -, unfavorable (age > 40 and secondary AML). (B) Survival by treatment lag (favorable risk patients). (C) Survival by treatment lag (intermediate risk patients). (D) Survival by treatment lag (unfavorable risk patients). (B-D) ------- indicates less than or equal to 5-day treatment delay; and - - - -, more than 5-day treatment delay.

Sekeres et al *Blood* 2009

Case 1 – Impact of delay prior to starting treatment

Figure 3. Survival in patients more than 60 years of age by risk group and interval from diagnosis to treatment. (A) Survival by risk group. ------- indicates favorable (age < 70 and WBC ≤ 5.0 K/μL); - - - -, intermediate (age < 70 and WBC > 5.0 K/μL, or age ≥ 70 and WBC ≤ 5.0 K/μL); and - · - · -, unfavorable (age ≥ 70 and WBC > 5.0 K/μL). (B) Survival by treatment lag (favorable risk patients). (C) Survival by treatment lag (intermediate risk patients). (D) Survival by treatment lag (unfavorable risk patients). (B-D) ------- indicates less than or equal to 5-day treatment delay; and - - - -, more than 5-day treatment delay.



No difference in survival among patients > 60 years old for whom treatment is delayed by greater than or equal to five days after diagnosis.

Sekeres et al *Blood* 2009

Case 1 – Impact of delay prior to starting treatment

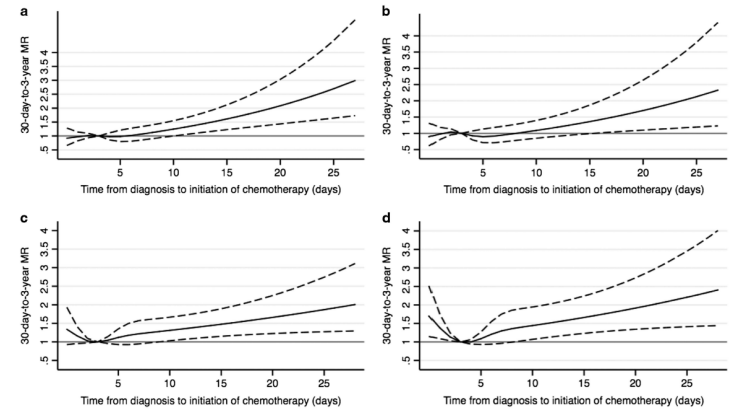


Figure 1. Estimated 30-day-to-3-year MRs according to time from diagnosis to treatment (TTT). (**a**) Crude and (**b**) adjusted MR for younger patients, and (**c**) crude and (**d**) adjusted MR for patients ⩾60 years. To obtain a graphical picture of the shape of the relation between TTT as a continuous covariate and overall survival, we used restricted cubic splines to smoothen the TTT mortality with linear restrictions imposed on both tails. It shows MRs (crude and adjusted 31-day-to-3-year MRs) stratified by age with 95% CI bands according to TTT as a continuous exposure. The four knots are placed at the 20th, 40th, 60th and 80th percentiles. The adjusted 30-day-to-3-year MRs reflect the results of Table X and show an almost U-shaped relation with the lowest MRs found in patients with TTT = 3–4 days in both age groups. In patients ⩾60 years, delaying chemotherapy had a significant impact on mortality for shorter TTTs than in younger patients (<60 years) (data not split at the day of initiated chemotherapy).

Mortality rate increased in both patient groups (both older and younger than 60 yo) if start of treatment delayed greater than 5 days.

Ostgard et al *Leukemia* 2014

Case 1 – Recommendations for New AML Diagnosis

- Recommended initial work-up for a patient with new or suspected diagnosis of AML
  - Bone marrow biopsy, send for:
    - Morphologic analysis
    - Flow cytometry
    - Cytogenetics +/- FISH **for rapid turnaround.** (STAT t15:17 if APL is suspected)
    - Next generation myeloid sequencing panel (Stanford, OHSU, GenPath). Typically includes 50 – 80 genes recurrent mutated in AML and other myeloid disorders.
  - "AML Prognosis panel" (FLT3 and NPM1 mutations) **for rapid turnaround**. Can be sent off the peripheral blood if there is adequate circulating disease (typically greater than 20% blasts)
- If transfer to another hospital pending, recommend close observation with labs (CBC, CMP) and possible transfusion every 2-3 days.

Case 1 – Scenario 1

- 62yo female with new diagnosis of de-novo AML
  - Cytogenetics return: 46,XX[20] – Normal Karyotype
  - "AML Prognosis Assay" returns:

Component Results

Component	Value	Ref Range & Units	Status
Clinical Indication Prognostic test for patients with Acute Myeloid Leukemia (AML)			Final
FLT3, Blood Positive for FLT3 internal tandem duplication ! Percent blast 88%.		Negative	Final
D835, Blood Negative for FLT3 D835 mutation		Negative	Final
NPM1, Blood Negative for insertion mutation in NPM1		Negative	Final

Case 1 – Scenario 1 - Risk Stratification

National Comprehensive Cancer Network®

NCCN Guidelines Version 3.2017
Acute Myeloid Leukemia

NCCN Guidelines Index
Table of Contents
Discussion

RISK STATUS BASED ON VALIDATED CYTOGENETICS AND MOLECULAR ABNORMALITIES[1]

RISK STATUS	CYTOGENETICS	MOLECULAR ABNORMALITIES
Favorable-risk	Core binding factor: inv(16)[2,3,4] or t(16;16)[2,3,4] or t(8;21)[2,4] or t(15;17)[4]	Normal cytogenetics: NPM1 mutation in the absence of FLT3-ITD or isolated biallelic (double) CEBPA mutation
Intermediate-risk	Normal cytogenetics +8 alone t(9;11) Other non-defined	Core binding factor with KIT mutation[2]
Poor-risk	Complex (≥3 clonal chromosomal abnormalities) Monosomal karyotype -5, 5q-, -7, 7q- 11q23 - non t(9;11) inv(3), t(3;3) t(6;9) t(9;22)[5]	Normal cytogenetics: with FLT3-ITD mutation[6] TP53 mutation

From NCCN Guidelines

Case 1 – Scenario 1 - Management

What should be the next step in management?

a) Cytoreduction with hyroxyurea

b) Induction with 7+3 (Cytarabine + Anthracycline)

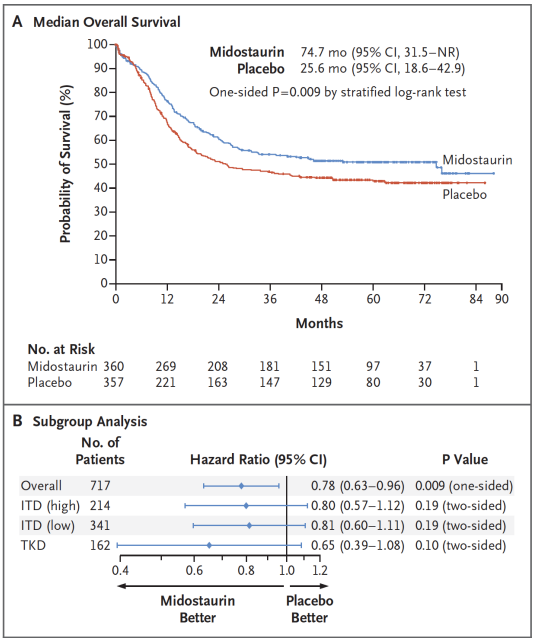
c) Start treatment with a hypomethylating agent

d) Induction with 7+3 (Cytarabine + Anthracycline) + Midostaurin

e) Induction with Alfa 0701 regimen (Cytarabine + Daunorubicin + fractionated Mylotarg)

f) Induction with FLAG-IDA

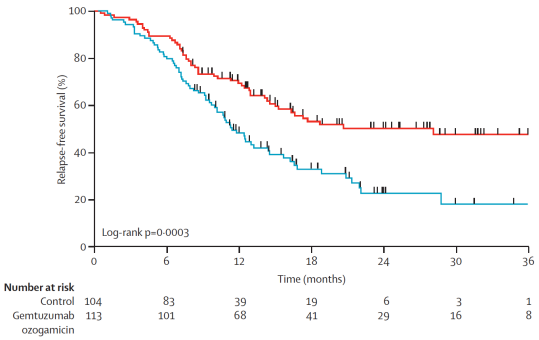
Case 1 – Scenario 1 – Midostaurin in addition to 7+3

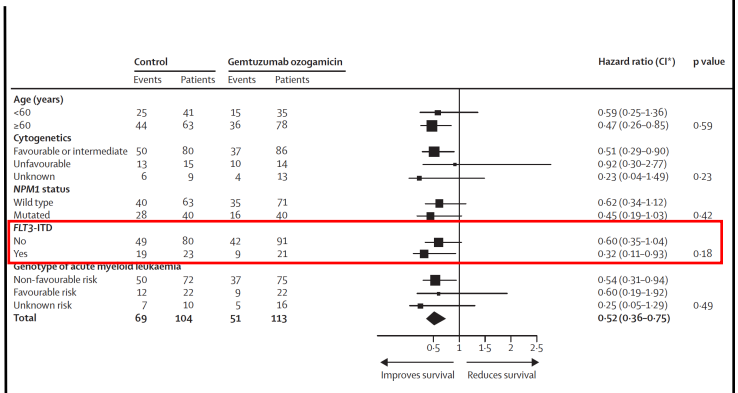


The addition of the multi-targeted kinase inhibitor midostaurin to standard chemotherapy significantly prolonged overall and event-free survival among patients with AML and a FLT3 mutation.

Stone et al *NEJM* 2017

Case 1 – Scenario 1 – Alfa 0701 Regimen - 7 + 3 + Mylotarg (gemtuzumab ozogamicin)





Statistically significant improvement in relapse free survival among patients who received gemtuzumab ozogamicin.

Benefits of gemtuzumab ozogamicin more pronounced among patients with FLT3-ITD.

Castaigne et al., Lancet 2012

Case 1 – Scenario 2

- 62yo female with new diagnosis of AML
  - Cytogenetics return: 47,XX,**+8**[6]/46,XX[14] – Trisomy 8
  - AML Prognosis Assay returns:

Component Results

Component	Value	Ref Range & Units	Status
Clinical Indication Prognostic test for patients with Acute Myeloid Leukemia (AML)			Final
FLT3, Blood Negative for FLT3 internal tandem duplication		Negative	Final
D835, Blood Negative for FLT3 D835 mutation		Negative	Final
NPM1, Blood Negative for insertion mutation in NPM1		Negative	Final

Case 1 – Scenario 2

- 76 gene next generation sequencing panel sent at time of diagnosis returns while patient is undergoing induction. It shows multiple mutations:

Gene: RUNX1
Mutation: p.H378fs*217 (2bp insertion frame shift mutation; Likely pathogenic)
Mutant Allele frequency: 36%

Gene: SF3B1
Mutation: p.G742D (Likely pathogenic)
Mutant Allele frequency: 29%
Variant ID: COSM145923

Gene: BCOR
Mutation: Splice site (3' end of intron 10 splice site mutation; Likely pathogenic)
Mutant Allele frequency: 32%

Case 1 – Scenario 2 – Patient summary

- 62 year old woman with AML
  - Excellent performance status
  - FLT3, NPM1, CEBPA mutations negative
  - Cytogenetics show Trisomy 8
  - Next generation sequencing panel shows BCOR, SF3B1 and Runx1 mutations
- What would be the most appropriate induction regimen, if any, for this patient?
- Should this patient be transplanted in first remission?

Case 1 – Scenario 2 – Runx1 Mutation

- Runx1 -
  - Transcriptional factor widely expressed in hematopoietic cells and important in hematopoiesis
  - Mutated in approx. 10% of cases of AML
  - Associated with decreased EFS, RFS and OS
  - Runx1 mutated AML is a new provisional WHO classification

Entire cohort *Clinical end point*	*RUNX1*^{mut} *(n = 245)*	*RUNX1*^{wt} *(n = 2194)*	P
CR rate, %	48.4	68.1	< 0.0001
RD rate, % Missing, n	40.6 1	23.4 34	0.03
EFS			< 0.0001
Median, mo (95% CI)	2.0 (1.6–2.8)	8.2 (7.3–8.9)	
5-year EFS (%)	9 (6–13)	24 (22–26)	
RFS			0.0007
Median, mo (95% CI)	12.1 (10.4–14.7)	16.0 (14.5–18.4)	
5-year RFS (%)	22 (16 to 30)	36 (34–39)	
OS			< 0.0001
Median, mo (95% CI)	13.3 (11.5–16.0)	21.1 (19.1–23.5)	
5-year OS (%)	22 (27–28)	37 (35–39)	

Gaidzik et al, Leukemia 2016

Case 1 – Scenario 2 – CPX-351

- CPX-351 (Vyxeos) –lipososomal formulation of a fixed-combination of daunorubicin and cytarabine
  - Recently FDA approved for adult patients with newly diagnosed therapy-related acute myeloid leukemia (t-AML) or AML with myelodysplasia-related changes (AML-MRC)
  - Recent phase III study comparing CPX-351 with traditional cytarabine and daunorubicin (7+3) for patients with newly diagnosed t-AML or AML-MRC.
    - Median OS was 9.56 months (95% CI, 6.60-11.86) with CPX-351 versus 5.95 months (95% CI, 4.99-7.75) with 7+3, representing a 31% reduction in the risk of death (HR, 0.69; *P* = .005).

Lancet JE, et al. J Clin Oncol. 34, 2016 (suppl; abstr 7000).

Case 1 – Scenario 2 – Genomic Classification and Prognosis of AML

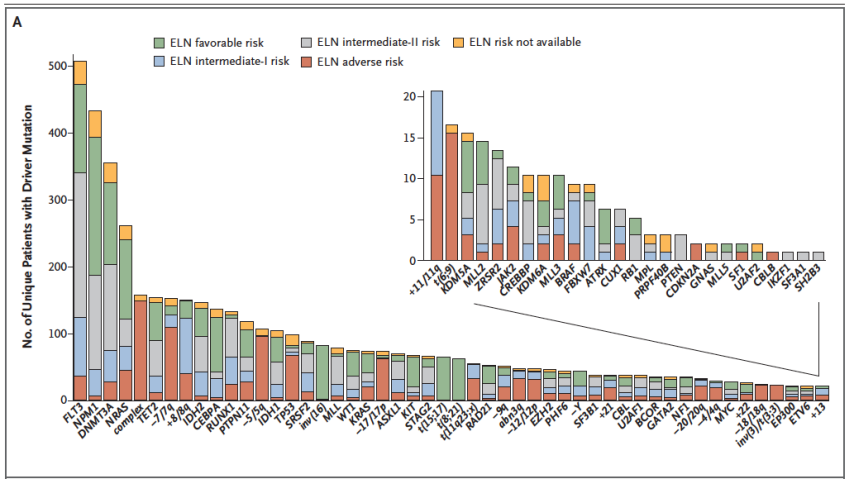


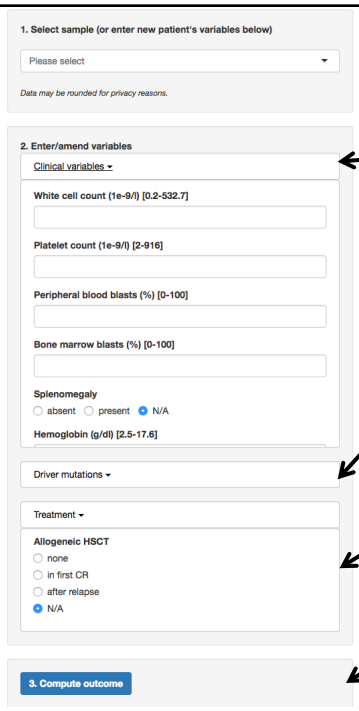
Figure shows driver events in 1540 AML patients. Each bar represents a distinct genetic lesion (mutations, chromosomal abnormalities). Colors represent molecular risk according to the classification.

Papaemmanuil et al., NEJM 2016

Case 1 – Scenario 2 – Genomic Classification and Prognosis of AML

- Personally tailored patient management based on statistical model that incorporates multiple patient characteristics, including clinical and genomic information.
  - http://cancer.sanger.ac.uk/aml-multistage/
- Developed on a cohort of 1,540 intensively treated patients with acute myeloid leukaemia, enrolled in three trials (AMLHD-98A, n=627; AMLHD-98B, n=173; and AMLSG-07/04, n=740).
  - The median age of patients was 50 (40-57 inter quartile range, min 18, max 84).
  - Median ECOG performance was 1 (0-2 inter quartile range).
  - A panel of 111 cancer genes was profiled in addition to cytogenetic data and diagnostic blood counts

Papaemmanuil et al., NEJM 2016
Gerstung et al., Nature Genetics 2017



http://cancer.sanger.ac.uk/aml-multistage/

Enter patient's clinical variables including: age, performance status, gender, presenting WBC, Plt, HGB, LDH, peripheral blast %, marrow %, AML type, presence/absence of splenomegaly

Enter status of 111 known driver lesions (including mutations and chromosomal abnormalities)

Choose at point patient would undergo allo stem cell transplant

Model will generate survival curves based on information provided

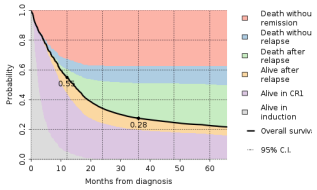
Papaemmanuil et al., NEJM 2016
Gerstung et al., Nature Genetics 2017

No allo transplant

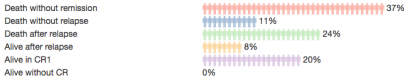
Patient summary

Patient: 62yr old female
Driver mutations: BCOR, RUNX1, SF3B1; +8/8q
Blood counts: 88 % Bone marrow blasts, 64 % Peripheral blood blasts, 8.8 1e-9/l White cell count, 261 units/l Lactic Acid Dehydrogenase, 4.9 g/dl Hemoglobin, 54 1e-9/l Platelet count
Treatment: No HSCT

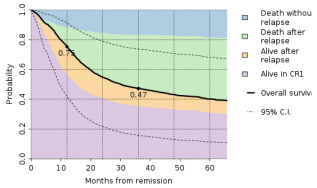
Outcome after diagnosis



Outcome 3 years after diagnosis



Outcome after first complete remission



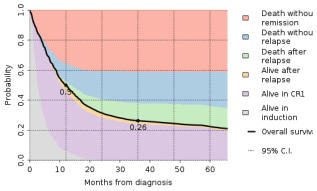
Outcome 3 years after remission

Transplant in first remission

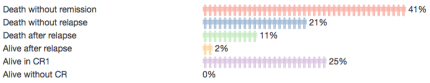
Patient summary

Patient: 62yr old female
Driver mutations: BCOR, RUNX1, SF3B1; +8/8q
Blood counts: 88 % Bone marrow blasts, 64 % Peripheral blood blasts, 8.8 1e-9/l White cell count, 261 units/l Lactic Acid Dehydrogenase, 4.9 g/dl Hemoglobin, 54 1e-9/l Platelet count
Treatment: HSCT in CR1

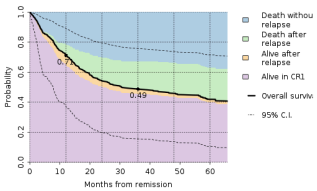
Outcome after diagnosis



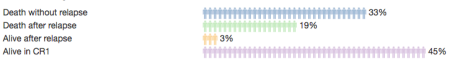
Outcome 3 years after diagnosis



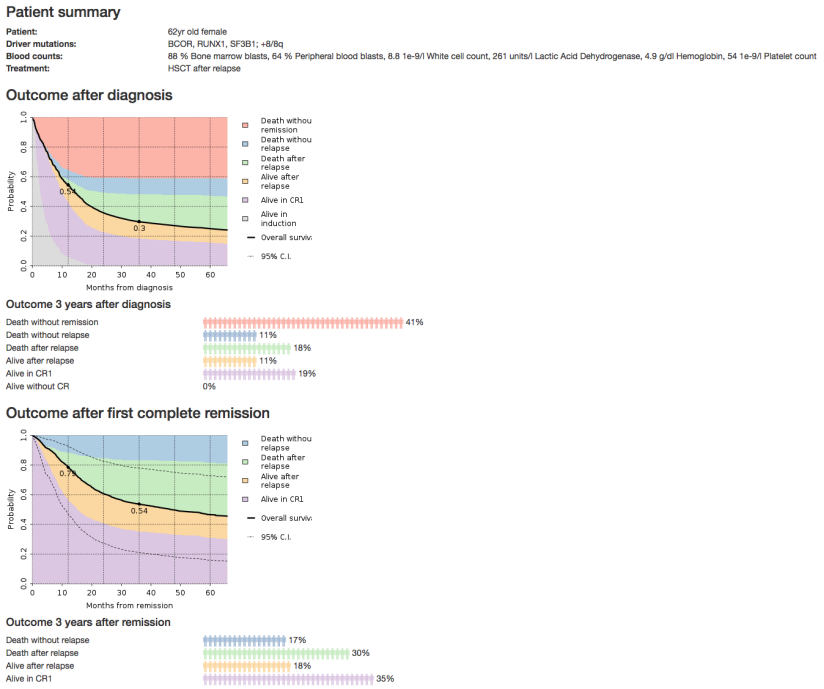
Outcome after first complete remission



Outcome 3 years after remission



Transplant only in first relapse



Patient summary

Patient:	62yr old female
Driver mutations:	BCOR, RUNX1, SF3B1; +8/8q
Blood counts:	88 % Bone marrow blasts, 64 % Peripheral blood blasts, 8.8 1e-9/l White cell count, 261 units/l Lactic Acid Dehydrogenase, 4.9 g/dl Hemoglobin, 54 1e-9/l Platelet count
Treatment:	HSCT after relapse

Outcome after diagnosis

Outcome 3 years after diagnosis

Death without remission	41%
Death without relapse	11%
Death after relapse	18%
Alive after relapse	11%
Alive in CR1	19%
Alive without CR	0%

Outcome after first complete remission

Outcome 3 years after remission

Death without relapse	17%
Death after relapse	30%
Alive after relapse	18%
Alive in CR1	35%

Case 1 – Scenario 2

- If this patient achieves a CR after induction therapy, should this patient go to transplant in first remission?

Case 1 – Take Away Points

- The addition of the multi-targeted kinase inhibitor midostaurin to standard chemotherapy significantly prolonged overall and event-free survival among patients with AML and a FLT3 mutation.
- Gemtuzumab ozogamicin, in addition to standard induction chemotherapy has been shown to cause a statistically significant improvement in relapse free survival in patients with AML, and in subset analysis has shown further benefit in patients with FLT3 mutations.
- Molecular diagnostics should be sent at diagnosis of all patients with AML, as the results can inform both front line and relapse therapy, and strongly influence the decision to whether to proceed to allo transplant.
- Runx1 mutated AML is a new provisional WHO classification
- Models incorporating clinical, molecular and pathologic data are being developed toward the goal of precision tailored patient specific treatment strategies in AML.

Case 2 – 66 year old man with fatigue and lymphadenopathy

- HPI:
  - Patient initially develops 3-4 months of progressive dyspnea and experiences a 10 pound weight loss over 2 months.
  - Develops diffuse lymphadenopathy. CT scan performed by PCP reveals extensive LAD, right sided pleural effusion and splenomegaly.
- PMHx: BPH
- PSHx: None
- SocHx: Retired, active. Denies EtOH or TOB
- FamHx: No family history of malignancy
- Meds: None
- Physical Exam: Multiple enlarged lymph nodes in bilateral cervical, supraclavicular, axillary and inguinal chains. Spleen palpable finger breadths below costal margin

Case 2 – Initial Labs

14.0	12.7 / 38.4	146

MCV – 92.4
ANC – 3,080
Segs - 21%
Bands – 1%
Lymph – 10%
Mono – 4%
Variant Lymphs– 63%

142	104	19	105
4.5	26	1.18	

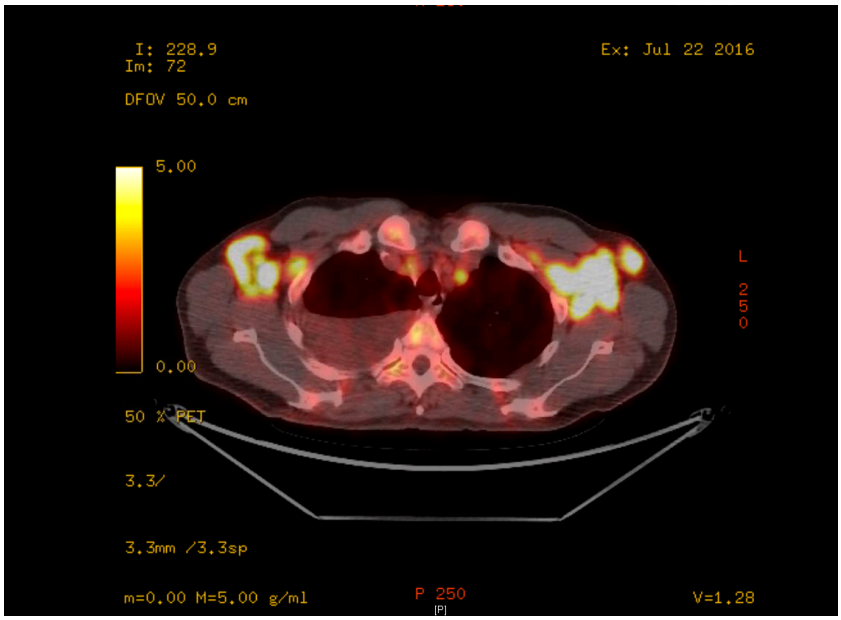
AST – 38
ALT – 20
Alk Phos – 88
T. Bili – 0.9

LDH - 672

Smear: Numerous variant lymphocytes

Case 2 – PET/CT

- Extensive hypermetabolic lymphadenopathy involving the neck, chest, abdomen, and pelvis. (SUVs up to 7.0)
- Right sided pleural effusion
- Enlarged spleen



Case 2 – Work-up

Inguinal Lymph Node Excisional Biopsy:

- Follicular lymphoma, predominately grade 1-2 with focal 3A morphology and high proliferation index.

Thoracentesis:

- Cytologic Diagnosis: Large cell lymphoma
- Ki67 – 60%
- CD20+, CD30+, BCL2+, CD10 neg
- FISH: BCL2 positive, BCL6 and c-myc negative

Bone Marrow Biopsy

- Involved by low grade follicular lymphoma. No evidence of large cell transformation in the marrow

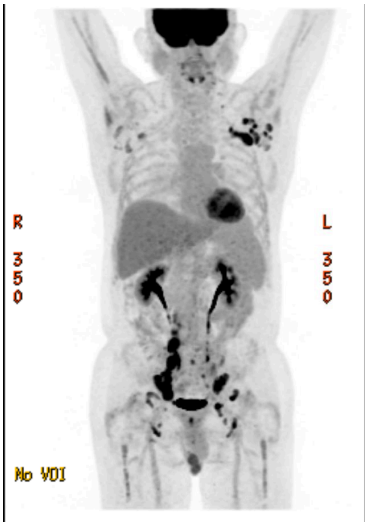
Diagnosis:

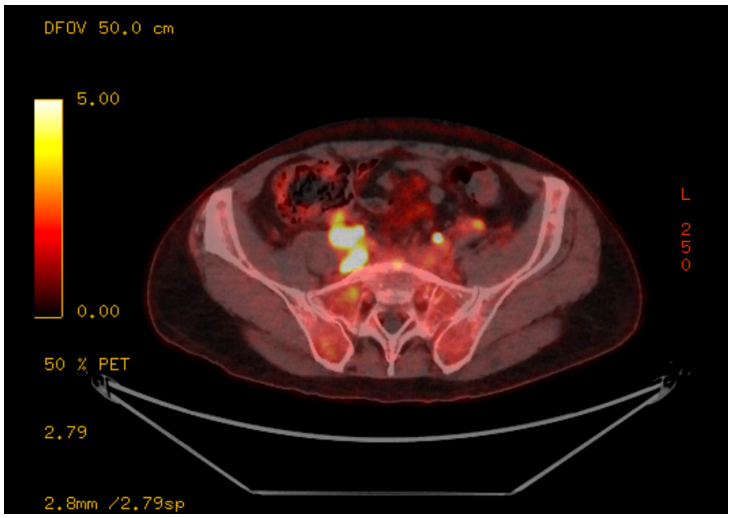
- Stage IVE diffuse large B-cell lymphoma. Transformed from follicular lymphoma. IPI score 4/5 (LDH, stage, age, extranodal sites)

Case 2 – Initial Course

- Given advanced stage, high IPI and good functional status, patient started on DA-EPOCH-R
- PET/CT after cycle 4 shows decrease in size and metabolic activity of lymphadenopathy
- PET/CT after 6 cycles shows partial response to therapy with persistent FDG avidity in axillary, inguinal, iliac lymph node chains. Small persistent pleural effusion.
- Repeat thoracentesis negative for lymphoma involvement
- Biopsy of axillary lymph node shows only grade 1-2 follicular lymphoma.
- Patient followed expectantly
- PET/CT performed 3 months later shows progressive disease involving axillary, pelvic, inguinal and cervical lymph node chains
- CT guided biopsy of deep pelvic lymph node reveals large cell lymphoma.
- Salvage chemotherapy with R-ICE started
- PET/CT performed after 2 cycles…

Case 2 –PET/CT after 2 cycles of R-ICE





Increase in size and FDG avidity of retroperitoneal lymphadenopathy. Mixed response elsewhere.

Case 2

What should be the next course of therapy?

a) Refer to BMT for autologous bone marrow transplant
b) Change therapy to Ibrutinib
c) Switch to an alternative salvage chemotherapy such as R-DHAP
d) Refer to hospice
e) Enroll into chimeric antigen receptor (CAR) T-cell trial

Case 2 – CAR T-cell Therapy

- Patient referred to University of Washington for CAR T-cell trial
- Enrolled in a trial of CAR19 therapy + Durvalumab

JCAR014 and Durvalumab in Treating Patients With Relapsed or Refractory B-cell Non-Hodgkin Lymphoma

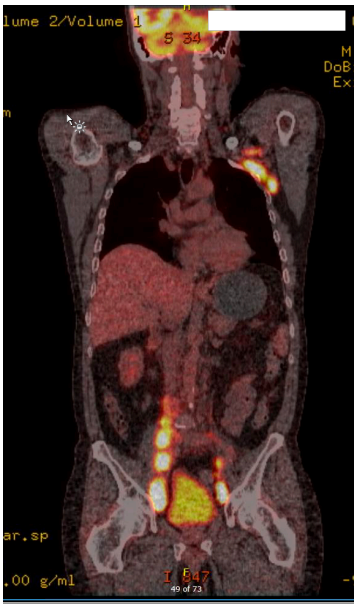
‹ Return to **search results**.

Complete title: A Phase 1b Study of JCAR014, Autologous T cells Engineered to Express a CD19-specific Chimeric Antigen Receptor, in Combination with Durvalumab (MEDI4736) for Relapsed/Refractory B-cell Non-Hodgkin Lymphoma

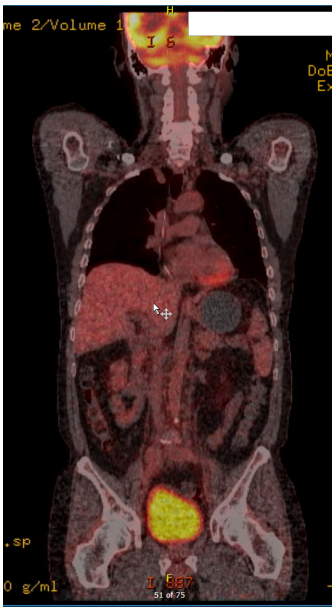
Case 2 – CAR T-cell Therapy

- Patient referred for CAR T-cell trial
- Enrolled in a trial of CAR19 therapy + Durvalumab
- Patients receive CAR19 cells on day 0 and Durvalumab IV on day 28, and then every 4 weeks for up to 10 doses in the absence of disease progression or unacceptable toxicity.
- Undergoes CAR T-cell therapy. Course complicated by fever related to cytokine release syndrome. Overall tolerates therapy well.

Case 2 – Patient Returns one month later with PET/CT



Prior to CAR T therapy



First post-treatment PET shows CR

Case 2 – Scenario 2

- Back to the initial presentation of this patient, who as you recall is is a 66 year old man who presented with progressive fatigue and lymphadenopathy, ultimately found to have Stage IVE DLBCL.
- **IF** a a next generation sequencing panel had been sent on his peripheral blood during his initial work-up and returned showing an ASXL1 mutation (frequently mutated gene found in myeloid disorders)
- **Should this influence the choice of first line therapy for his DLBCL? Would it be reasonable to consider CAR T-cell therapy in the first line?**

Case 2 – Clonal Hematopoiesis of Indeterminate Potential (CHIP)

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Age-Related Clonal Hematopoiesis Associated with Adverse Outcomes

Siddhartha Jaiswal, M.D., Ph.D., Pierre Fontanillas, Ph.D., Jason Flannick, Ph.D., Alisa Manning, Ph.D., Peter V. Grauman, B.A., Brenton G. Mar, M.D., Ph.D., R. Coleman Lindsley, M.D., Ph.D., Craig H. Mermel, M.D., Ph.D., Noel Burtt, B.S., Alejandro Chavez, M.D., Ph.D., John M. Higgins, M.D., Vladislav Moltchanov, Ph.D., Frank C. Kuo, M.D., Ph.D., Michael J. Kluk, M.D., Ph.D., Brian Henderson, M.D., Leena Kinnunen M.Sc., Heikki A. Koistinen, M.D., Ph.D., Claes Ladenvall, Ph.D., Gad Getz, Ph.D., Adolfo Correa, M.D., Ph.D., Benjamin F. Banahan, Ph.D., Stacey Gabriel, Ph.D., Sekar Kathiresan, M.D., Heather M. Stringham, Ph.D., Mark I. McCarthy, M.D.,* Michael Boehnke, Ph.D.,* Jaakko Tuomilehto, M.D., Ph.D., Christopher Haiman, Sc.D., Leif Groop, M.D., Ph.D., Gil Atzmon, Ph.D., James G. Wilson, M.D., Donna Neuberg, Sc.D., David Altshuler, M.D., Ph.D.,* and Benjamin L. Ebert, M.D., Ph.D.†

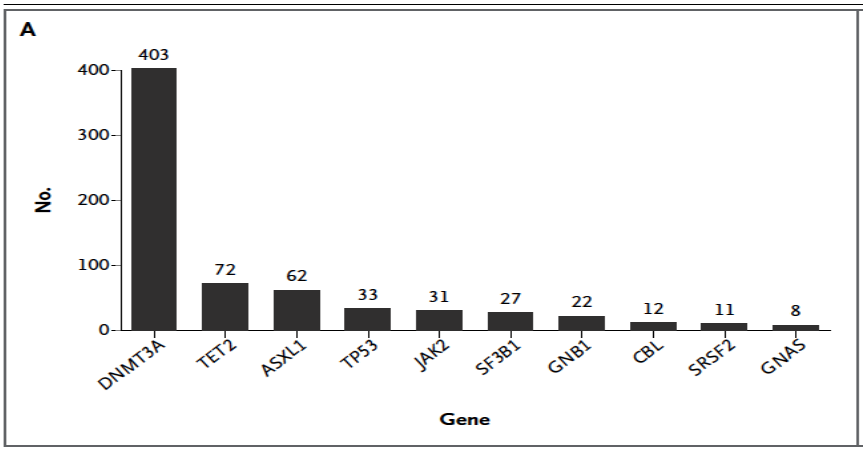
Jasiwal et al. N Engl J Med 2014

Case 2 – Clonal Hematopoiesis of Indeterminate Potential (CHIP)

- Jasiwal et al.
  - Analyzed whole-exome sequencing data from DNA in the peripheral blood cells of 17,182 persons who were selected without regard to hematologic characteristics
    - 15,801 were case patients and controls ascertained from 22 cohorts in type 2 diabetes association studies
    - 1381 were previously unsequenced participants in the Jackson Heart Study
- Looked for mutations in 160 recurrently mutated candidate genes in myeloid and lymphoid cancers

Jasiwal et al. N Engl J Med 2014

Case 2 – Clonal Hematopoiesis of Indeterminate Potential (CHIP)



Jasiwal et al. N Engl J Med 2014

Case 2 – Clonal Hematopoiesis of Indeterminate Potential (CHIP)

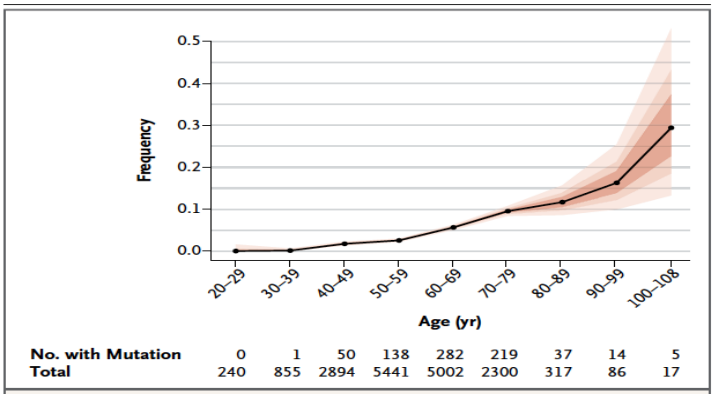
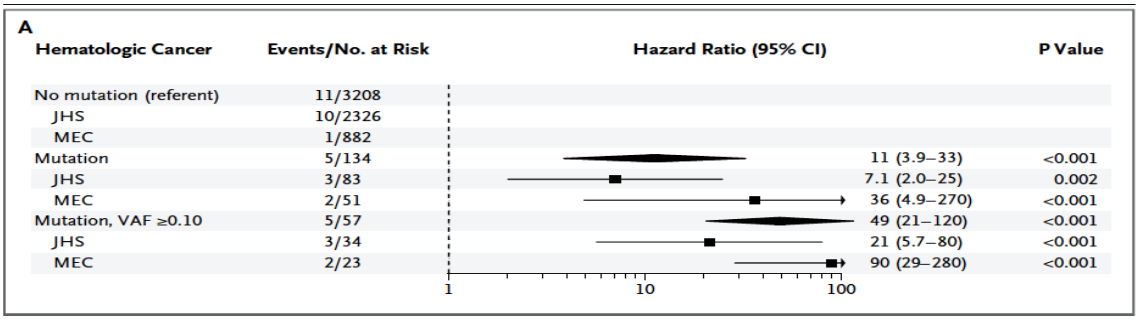


Figure 1. Prevalence of Somatic Mutations, According to Age.
Colored bands, in increasingly lighter shades, represent the 50th, 75th, and 95th percentiles.

Jasiwal et al. N Engl J Med 2014

Case 2 – Clonal Hematopoiesis of Indeterminate Potential (CHIP)



The presence of a somatic mutation was associated with an increase in the risk of hematologic cancer

Jasiwal et al. N Engl J Med 2014

Case 2 – Clonal Hematopoiesis of Indeterminate Potential (CHIP)

VOLUME 35 · NUMBER 14 · MAY 10, 2017

JOURNAL OF CLINICAL ONCOLOGY | BIOLOGY OF NEOPLASIA

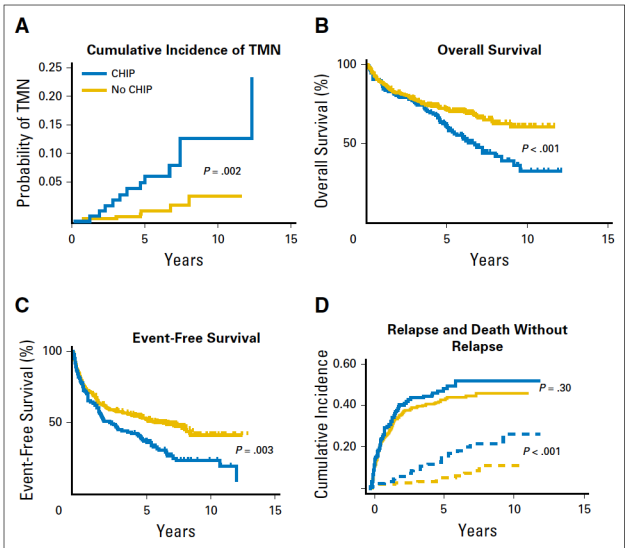
Clonal Hematopoiesis Associated With Adverse Outcomes After Autologous Stem-Cell Transplantation for Lymphoma

Christopher J. Gibson, R. Coleman Lindsley, Vatche Tchekmedyian, Brenton G. Mar, Jiantao Shi, Siddhartha Jaiswal, Alysia Bosworth, Liton Francisco, Jianbo He, Anita Bansal, Elizabeth A. Morgan, Ann S. Lacasce, Arnold S. Freedman, David C. Fisher, Eric Jacobsen, Philippe Armand, Edwin P. Alyea, John Koreth, Vincent Ho, Robert J. Soiffer, Joseph H. Antin, Jerome Ritz, Sarah Nikiforow, Stephen J. Forman, Franziska Michor, Donna Neuberg, Ravi Bhatia, Smita Bhatia, and Benjamin L. Ebert

Performed whole exome or targeted based sequencing on pre- and post-ASCT samples from who underwent autologous stem cell transplantation for Hodgkin lymphoma or non-Hodgkin lymphoma

Gibson et al. JCO 2017

Case 2 – Clonal Hematopoiesis of Indeterminate Potential (CHIP)



Gibson et al. JCO 2017

STANFORD CANCER CENTER | UCDAVIS COMPREHENSIVE CANCER CENTER | UCSF Helen Diller Family Comprehensive Cancer Center | ASSOCIATION OF NORTHERN CALIFORNIA ONCOLOGISTS

Thank you!

Questions/comments

Other cases?

Brian Sworder MD, PhD, fellow (Stanford)
bsworder@stanford.edu