## 16<sup>th</sup> Annual INDY HEMATOLOGY REVIEW 2019

## State of the Art 2019: Emerging Therapies in Hematologic Malignancies

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## **DISCLOSURES**

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## **Aggressive Lymphoma**

### DIFFUSE LARGE B-CELL LYMPHOMA

- <u>FLYER</u>:
- Phase III Trial of R-CHOP x 4 Followed by Rituximab x 2 vs R-CHOP x 6 in younger patients with favorable-prognosis DLBCL: Non inferior with 1/3 less toxicities.
- R-CHOP x 4  $\rightarrow$  R x 2 noninferior PFS, EFS, and OS vs standard R-CHOP x 6
- 36-mo PFS rate: 96% with R-CHOP x 4  $\rightarrow$  R x 2 vs 94% with R-CHOP x 6
- <u>Non–Germinal Center B-Cell Subtypes of DLBCL IBRUTINIB + CHOP:</u>
- Phase III Trial of First-line Ibrutinib + R-CHOP vs Placebo + R-CHOP; did not significantly improve EFS in the ITT population or in those with ABC subtype.
- Exploratory Analysis: Age< 60 yrs, ibrutinib + R-CHOP improved EFS, PFS, and OS,
- Among those aged  $\geq$  60 yrs, ibrutinib + R-CHOP showed higher rates of serious Aes

### **PRIMARY MEDIASTINAL LARGE CELL LYPHOMA**

- <u>KEYNOTE-170/KEYNOTE-013: Pembrolizumab in R/R PMBCL;</u>
- Phase II Studies, in a combined analysis, Median DoR not yet reached ,12-mo OS > 50%; durable CR in both studies
- <u>CHECKMATE 436: Nivolumab plus Brentuximab Vedotin in R PMBCL;</u>
- Phase 1/2 trial, ORR 70%, CR 27%.
- AEs consistent with the safety profiles of nivolumab and brentuximab vedotin alone.

## **Aggressive Lymphoma**

### **T-CELL LYMPHOMA**

- <u>ECHELON-2: Phase III Trial of Brentuximab Vedotin + CHP vs CHOP in Previously</u> <u>Untreated CD30+ Peripheral T-Cell Lymphomas</u>
- BV+ CHP superior to standard CHOP: Risk of PFS reduced by 29% (HR: 0.71, P = .011)
  - Risk of death reduced by 34% (HR: 0.66, P = .0244)
- Duvelisib plus Romidepsin:
- Highly active in relapsed/refractory PTCL: ORR 50% (18/35) with tolerable side effects.
- Duvelisib can be safely combined with romidepsin with lower rate of Gr 3-4 transaminitis than single-agent duvelisib at the same dose.

### MANTLE CELL LYMPHOMA

• <u>MCL0208</u>: Phase III, Lenalidomide maintenance in patients age <60, following RCHOP/BEAM, improves survival after ASCT, 3 yr PFS 80% vs 60%, HR 0.52 (P = .015) and a stratified HR of 0.51 (P = .013).

## **INDOLENT LYMPHOMA**

### • FOLLICULAR LYPHOMA

- <u>AUGMENT:</u> Phase III Study of Rituximab/Lenalidomide (R<sup>2</sup>) vs Rituximab/Placebo for R/R Indolent NHL (Grade 1-3a FL and MZL): R<sup>2</sup> results in significantly greater efficacy than R plus placebo
- Median IRC-PFS: 39.4 vs 14.1 mos (HR: 0.46; 95% CI: 0.34-0.62; P < .0001)
- ORR, median DoR improved with R2, OS improved with R<sup>2</sup> in patients with FL
- <u>CHRONOS 1:</u> Copanalisib active, deepening of the responses and with no evidence of worsening of AEs ORR was 61%. CR: 20.2%,mDOR:14.1 mos, mPFS:12.5 mos and the median OS:42.6 mos

### • HAIRY CELL LYMPHOMA

- <u>ROAR</u>: Phase II Study of Dabrafenib + Trametinib for Relapsed/Refractory BRAF V600E–Mutated HCL: ORR: 78%, Estimated 12-mo PFS and OS: both 97.6%
- <u>Mutation in BCL2 Protein Confers Resistance to Venetoclax:</u>
- 75% of resistance is due to a late Gly101Val mutation which renders cells 30- to 50-fold less sensitive to venetoclax than cells expressing wild-type *BCL2*.
- *BCL2* Gly101Val was found to result in impaires venetoclax binding, with a reduction in the capacity of venetoclax to bind to the Gly101Val mutation by approximately 180-fold

## Waldenström's Macroglobulinemia

- **iNNOVATE Study Update**:
- Ibrutinib and Rituximab superior to Rituximab
- In treatment-naïve and previously treated pts with WM regardless of genotypic factors.
- Similarly, in heavily pretreated, RTX-refractory pts with follow-up >3 y, single-agent ibrutinib is also highly active and responses improved over time.
- Venetoclax in Waldenström's Macroglobulinemia:
- Phase II, Active and tolerable in relapsed/refractory, N=30, Overall (≥Minor) 26 (87%), Major (≥Partial) 22 (74%)
- Ibrutinib-Rituximab superior to Real-World (RW) Treatments for patients with Waldenström's Macroglobulinemia:
- IR versus RW PC therapy for all analyzed patients with WM unadjusted HRs: 0.32, for PFS and 0.38 for OS

## Chronic Lymphocytic Leukemia

- <u>A041202: Phase III Trial of Ibrutinib ± Rituximab vs Bendamustine + Rituximab in Older</u> <u>Patients With Previously Untreated CLL:</u> Ibrutinib ± R significantly prolonged PFS vs BR (both P < .001), but no PFS benefit with addition of R to ibrutinib</li>
  - Median PFS: ibrutinib, NR; ibrutinib + R, NR; BR, 43 mos, 2-yr PFS: ibrutinib, 87%; i + R, 88%; BR, 74%,
  - PFS benefits seen with Ibrutinib ± Rituximab vs BR in all cytogenetic factor–related subgroups, best with del(17p13.1). **Ibrutinib improved PFS by 63% over the standard of care.**
  - No significant interaction between IgVH mutation status and PFS benefit by regimen.
  - Increased PFS among patients with mutated vs unmutated IgVH disease (HR: 0.51; 95% CI: 0.32-0.81)
- Phase III E1912: First-line Ibrutinib + Rituximab vs Standard-of-Care FCR in Younger Patients (<70 years) With Previously Untreated CLL: First-line ibrutinib + R showed statistically significant improved PFS and OS vs standard-of-care FCR (all  $P \le .00003$ ) HR for PFS: 0.35, HR for OS 0.17 ITT
  - **Ibrutinib and rituximab improved PFS by 65% and overall survival by 83% compared with FCR.**
  - <u>Mutated IgVH status</u>, no statistically significant PFS improvement with ibrutinib + R
- <u>iLLUMINATE: Phase III Trial of Ibrutinib + Obinutuzumab vs Chlorambucil + Obinutuzumab</u> <u>in Previously Untreated CLL/SLL:</u>
- I+O significantly improved PFS vs C+O independent of high-risk features, HR (ITT): 0.23; P < .0001) including patients with bulky disease and high-risk patients with del(17p), TP53 mutation, del(11q), and/or unmutated IgVH
- Ibrutinib and obinutuzumab improved PFS by 77% for all patients and by 85% in High-Risk CLL compared with Chlorambucil + Obinutuzumab.
- <u>Phase II Trial of Ibrutinib + Venetoclax in Previously Untreated, High-Risk CLL:</u>
  - High response rates with 96% and 69% of patients achieving CR/CRi and BM MRD negativity, respectively, at 18 months.
- Acalabrutinib in CLL (ACE-CL-001): PhaseI/II: Initial therapy: ORR 97% (100% in high-risk CLL)

## Chronic Myelogenous Leukemia

### • TKI CHIOCE ?

- <u>SPIRIT 2</u>: 5-yr analysis of the UK National Cancer Research Institute UKNCRI randomized study of dasatinib vs imatinib as Firstline therapy for cCML
- PCR RR: MR3 and MR4 83.0% vs 63.0% and 77.5% vs57.2%, 5yr EFS: 91.0% vs 89.0%.

### • **<u>STOPPING THERAPY</u>**

- **STEM 2:** Relapse increases with <75 mos of therapy (42.6% lower likelihood of maintaining remission after stopping imatinib) and ddPCR < 0.023% IS (45.4% less likely to relapse.
- Imatinib suspension and validation (ISAV) study: n=118, 52.3% relapses over a median 59.2 months of follow-up. 69.6% of relapses within 9 mos of discontinuation, 3 late relapses occurred, at 31-month, 36-month, and 56-month timepoints.
- "Therefore, patients who discontinue imatinib should be monitored for a long period of time".

### • **RESISTANT CML**

- **PF-114 mesylate**, **HQP1351**: Novel TKIs, active and safe.
- <u>Asciminib</u>:
- A specific allosteric BCR-ABL1 inhibitor, for T315I mutant CML: Phase 1 Trial:
- "Safe and tolerable",
- MMR 36.7% @ 12.2 wks, MR4: 19.4% and MR4.5 16.1%. Ponatinib-naïve patients with higher responses.

## Acute Myeloid Leukemia

- QuANTUM-R: Phase III Trial of Quizartinib in Patients With *FLT3*-ITD–Mutated Relapsed/Refractory AML: Quizartinib significantly prolonged OS vs. salvage CT (HR: 0.76; P = .0177)
- OS benefit observed across subgroups and consistent across sensitivity analyses
- <u>Gilteritinib Combined With 7+3 Chemotherapy for Newly Diagnosed Acute Myeloid</u> <u>Leukemia:</u> Phase I Study, active and tolerable, CRc 94%
- SORMAIN: Sorafenib vs Placebo Maintenance Therapy After Allogeneic SCT in <u>FLT3-ITD AML</u>: 2-yr RFS and OS significantly improved with sorafenib vs placebo (PFS HR: 0.39; P = .013), OS at 30 mo vs placebo (HR: 0.447; P = .03)
- <u>Ivosidenib or Enasidenib Plus Standard Induction and Consolidation Chemotherapy</u> in Newly Diagnosed, *IDH1/IDH2*-Mutated AML:
- High response rates (CR + CRi/CRp rate: ivosidenib + CT, 80%; enasidenib + CT, 72%), Mutation clearance (41% and 25%, respectively) and MRD negativity (88% and 45%). 1yr survival rates ≥ 75%
- Frontline Venetoclax + Decitabine or Azacitidine in Older Patients with untreated AML and ineligible for Intensive Chemotherapy: Phase 1b trial, CR/CRi: 71% vs 74%, Median OS: 16.9 vs 16.2 mos with venetoclax + azacitidine or decitabine, respectively
- IMGN779 in R/R CD33+ AML: Novel CD33-targeted ADC, Activity observed in 41% patients receiving ≥ 0.39 mg/kg, no cumulative toxicity, few CR/CRi at current doses, 1 DLT: fatal VOD with acute kidney injury

## Acute Lymphoblastic Leukemia

- Sequential HCVAD With Blinatumomab as Frontline Therapy for Adults With Ph-Negative B-Cell ALL: HCVAD x 4 → B x 4 → 3POMP/1B x 15 cycles
- Phase 2, N = 19, Age 14-59, CR: 100%; MRD negativity: 95% (median: 17 days)
- 1-yr RFS: 75%; 1-yr OS: 93%, Early OS data suggest favorable survival outcomes
- Inotuzumab Ozogamicin + Low-Intensity Chemo ± Blinatumomab in Newly Diagnosed Older Patients (> 60) With Ph- ALL: INO + miniHCVAD x 4 → POMP x 3 yrs vs INO + miniHCVAD x 4 → B x 4 → 3POMP/1B x 4 cycles (18 mo)
- Phase 2, INO + mini-HCVD with (n=11) or without (n=41) blinatumomab effective, ORR: 98% (95% MRD by flow cytometry), 3-yr OS: 54%, 3-yr CR duration: 74%
- VOD rate: 8%, Study amended to use lower doses of fractionated INO (2.7 mg/m<sup>2</sup>)
- Addition of blinatumomab increased 1-yr OS (74% vs 91%)
- SWOG 1318: First-line therapy with Blinatumomab followed by POMP Maintenance in Older Patients with Ph-Negative B-Cell ALL: B x 1-2 → B x 4 → POMP x 18
  - Estimated 1-yr OS rate: 67%, CR + CRi rate: 66%, MRD negativity achieved in 92% of responders. Regimen tolerated with no early deaths, 1 case each of grade 3 neurotoxicity and grade 3 CRS observed.
- <u>BLAST Long Term Results</u>: Phase II, Blinatumomab in Adults with Ph-Negative B-Cell P ALL who are MRD Positive after chemo: Median OS; 36.5 mos (median follow-up: 53.1 mos), OS plateaued > 50% at 48 mos in the overall patient population and in those in CR1 or who achieved complete MRD response after blinatumomab.

## <u>Myeloproliferative Neoplasms and</u> <u>Myelodysplastic Syndromes</u>

### **Myelodysplastic Syndrome**

- MEDALIST: Phase III, Luspatercept in Very Low– to Intermediate-Risk MDS with RS: First-in-class erythroid maturation agent, targets TGF-β ligands to block aberrant Smad2/3 signaling and augment late-stage erythropoiesis
- Significantly reduced RBC transfusion burden compared with placebo, and proportion of
  patients achieving RBC transfusion independence for ≥ 8 wks in wks 1-24 and for ≥ 12 wks
  in wks 1-24 and in wks 1-48 and an increase in Hb of ≥ 1.5 g/dL
- Nivolumab or Ipilimumab With or Without Azacitidine in MDS: Phase II, Single-agent ipilimumab after HMA failure: ORR, 30%; 1-yr OS, 45%
- Nivolumab + AZA: CR, 40%; ORR, 70% Median OS not reached with ipilimumab + AZA
   MPNs
- <u>Ruxolitinib + Thalidomide in Myelofibrosis:</u> Produced objective responses, including clinical improvement (40%) and platelet responses (60%)
- <u>MYF2001</u>: Phase II Trial of Imetelstat (13 mer telomerase inhibitor) in Patients With Myelofibrosis Who are Refractory to or Relapsed on JAK Inhibition: : SVR ≥ 35% in 6 patients (10%), TSS ≥ 50% reduction in 19 patients (32%), Median OS: 29.9 mos
- **EXPLORER:** Avapritinib, KIT D816V inhibitor ative in Systemic Mastocytosis: ORR 83%, 41% mean reduction in symptoms (58% reduction of severe symptoms)

## **Multiple Myeloma: Initial Therapy**

- MAIA: Phase III, Daratumumab + Len/Dex vs Len/Dex in Transplantation-Ineligible NDMM: 30mos PFS: 56 vs 71%, HR 0.56 P = <.0001, Improved PFS by 44%</li>
- Improved depth of response: 2-fold higher sCR/CR rate and 3-fold improvement in MRD
- Rd Followed by R vs Continuous Rd in Intermediate-Fit Elderly Patients with NDMM, <u>Phase III study:</u> R maintenance after Rd x 9 cycles: Median EFS, Rd-R vs Rd: 9.3 vs 6.6 mos, respectively (HR 0.72; P = .044), comparable efficacy of Rd-R vs Rd, fewer toxicities
- **TOURMALINE-MM3**: Phase III, Ixazomib maintenance after ASCT in NDMM: Significantly improved PFS by 39% (26.5 vs 21.3 mos with placebo), low SPM rate
- Daratumumab + IRd in NDMM, Phase II Study: Rapid and deep responses, best response ≥ VGPR in 69% of patients, deepened over time, CR rate 29% in cycle 2, 39% in cycle 4
- <u>HOVON 143:</u> Phase II; Ixazomib, Daratumumab, and low-Dose Dex in unfit, frail patients with NDMM: ≥ VGPR in 30% unfit, 20% frail patients; ORR (100% in unfit, 80% in frail)
- **FORTE:** KRd Induction/Consolidation and ASCT vs 12 Cycles of KRd vs KCd in NDMM:
- Both KRd–ASCT–KRd and 12 cycles of KRd vs KCd, similarly significantly improved rates of premaintenance ≥ VGPR, sCR, and MRD negativity
- <u>CoMMpass Study:</u> Prospective observational study: KRd significantly improved 12-mo EFS versus VRd in patients with NDMM (HR, 0.28; p=0.0043) with or without ASCT
- **<u>GRIFFIN</u>**: Run in trial, n =16, Dara + VRd shows early efficacy, 100% VGPR, in NDMM

## Multiple Myeloma: Relapsed/Refractory

- STORM Trial Part 2: Selinexor + Low-Dose Dexamethasone in Penta-Refractory <u>MM</u>: Phase II, deep, durable responses with an ORR of 26.2% and DoR of 4.4 mos, 39.3% achieved ≥ MR; 78.7% achieved ≥ SD
- Median OS: 8.6 mos
- <u>Selinexor/Daratumumab/Dexamethasone</u>: Phase Ib, n=28,ORR: 74% (PI/IMiD refractory, dara naïve MM) ORR: 21% ORR (quad/dara refractory MM)
- **LEGEND-2**: Phase I Study of LCAR-B38M CAR T-Cell Therapy in R/R Multiple Myeloma (LCAR-B38M: CAR T-cell therapy targeting 2 BCMA domains): ORR 88%,
- MRD-negative CR: 68%, Survival improved in patients achieving MRD-negative CR
- Major AE, CRS: 90% mostly grade 1/2, 7% grade 3, Neurotoxicity in 1 patient (grade 1)
- First-in-Human Phase I Trial of AMG 420, a BiTE Construct Binding to BCMA and CD3, in Heavily Pretreated Patients With Multiple Myeloma. MTD 400 μg/day
  - ORR of 70% (7/10), including 4 patients achieving MRD-negative sCR
  - CR achieved by 3 patients at lower doses, including 1 MRD-negative sCR
- Serious infections observed in 12 patients (29%)
- HORIZON: Melflufen-dexamethasone active in multi-resistant MM: ORR 33%, clinical benefit rate (minimal response or better) 39%, and 84% of patients had stable disease or better. PFS 4mos.
- ELOQUENT-3: EHA2018; Elotuzumab + Pomalidomide, Dex (PD) improved PFS by 46% (HR = 0.54, p = .0078) versus PD, mPFS of 10.3 mo compared with 4.7 mo

## **Hematopoeitic Transplantation/CARTS**

- <u>Phase II REACH1: Ruxolitinib + Corticosteroids in Steroid-Refractory Acute Graft-</u> <u>vs-Host Disease:</u>
- ORR of 54.9% at day 28 varying by MAGIC grade of aGVHD at baseline, from > 80% with grade II to ~ 40% with grade 3 or 4. Best ORR at any time on treatment: 73.2%
- Sustained reductions in corticosteroid use with ruxolitinib
- 90% of pts on maintenance therapy achieved CR, with 60% MRD-negative rate
- **Defibrotide** in VOD/SOS following HSCT: Early defibrotide initiation improved day +100 survival regardless of MOD status.

### • CART THERAPY

- <u>UCART19</u> is safe, effective in ALL. UCART19 is an allogeneic, genetically modified, CAR T-cell product (anti-CD19 scFv- 41BB-CD3ζ) manufactured from healthy donor T cells, with CD52 Knock-out, and a CD20 mimotope. No GVHD. CR (overall): 67%, 82% (after alemtuzumab-based LD).
- <u>AMORED CART THERAPY</u>: <u>1928z-41BBL</u>: Designed to protect T-cells from tumor microenviroment: N=35, R/R CD19 hematologic malignancies: CR 78% in DLBCL
- Axicabtagene Ciloleucel (Axi-cel) CART Therapy in R/R DLBCL: Real World Data;
- Similar to ZUMA-1 trial findings: N =104, 95 evaluable.
- Best ORR: 71%, CR 44%, PR 26%

## **Benign and Not So Benign Hematology**

- <u>PAUCE</u>: DOAC interruption for an elective surgery, a simple and standardized management strategy yielded low rates of bleeding and thromboembolism , n= 3007
- **<u>BELIEVE</u>**: Phase III Study of Luspatercept vs Placebo in Beta-Thalassemia: Significantly reduced transfusion burden by ≥ 33% in Wks 13-24 compared with placebo
- <u>CASSINI:</u> Phase III, Rivaroxaban vs Placebo as VTE prophylaxis in ambulatory patients with cancer: Significantly reduced VTE with a low incidence of major bleeding, and with the use of the Khorana score  $\geq 2$  cutoff of identifying high risk m patients for VTEs.
- **ADAM:** Phase III: Apixaban safe as, and more effective than, dalteparin for patients with cancer-associated venous thromboembolism (VTE).
- <u>HAVEN 2:</u> Phase III, Emicizumab (bispecific mAb that bridges FIXa and FX to restore function of absent FVIIIa, with clinically relevant prevention or reduction in bleeds
- **Frontline Eltrombopag + Pulsed Dexamethasone in ITP**: Prolonged response rate significantly higher than historical rate for dexamethasone alone (< 36%; P = .036)
- **<u>Avatrombopag in Chronic ITP</u>**: long-term exposure is safe and effective.
- **Fostamatinib in ITP:** OLE study: Long term response  $\geq 24$  months: 56% response.
- <u>TELESTO:</u> Phase II, Deferasirox vs Placebo in Low-/Intermediate-Risk MDS and Iron Overload: 3-yr EFS rate 61.5% vs 47.3% with placebo, HR: 0.636, trend toward OS
- <u>APL-2 a cyclic peptide inhibitor of C3 (upstream of C5) in PNH</u>: Prevents intravascular and extravascular hemolysis with sustained increases in Hb in the absence of transfusions.

## What does it all mean ? My thoughts - 1

### • **PRACTICE changing:**

- Daratumumab + Chemo as first line therapy in Myeloma (MIAI)
- Brentuximab Vedotin + AVD as first line therapy in T cell lymphoma
- DOACs in the treatment and prevention cancer associated VTEs
- FIRST LINE therapy in CLL: The death of BR/FCR? Not yet?, but IR, is planning a funeral, and O+I is here
- THE JURY IS IN !!: YES, PAUCING DOACS for procedures
- **Potentially Practice changing:** 
  - TKI discontinuation in CML Continue to Monitor !!!!
  - Ixazomib maintenance in Myeloma
  - Lenalidomide maintenance in Mantle cell lymphoma, Lenalidomide + Rituximab in Follicular NHL
  - Luspatarcept therapy in low risk MDS/RA and anemias
  - IDH1/2 inhibition + chemo in first line therapy of IDH1/2 mutant AML
  - What is the best PI in Induction therapy for newly diagnosed Multiple Myeloma: Carfilzomib ?

### What does it all mean ? My thoughts - 2

- PRACTICE confirming:
  - PI-3 kinase inhibitors in in Follicular NHL and CLL
  - Antibody therapy in in Relapsed Myeloma
  - Blinatumumab in ALL: ?Induction and maintenance
  - CAR-T therapy in relapsed DLBCL NHL and ALL
- <u>Stay tuned</u>
  - CAR-T therapy in Myeloma
  - SELINIXOR in Penta Refractory Myeloma
  - "Off the shelf "CAR-Ts
  - Venectoclax and Ibrutinib in CLL
  - Avapritinib in Systemic Mastocytosis
  - Ruxolitinib in aGVHD

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## <u>Co-Chair Indy Hematology Review</u> Challenging Cases



### Michael C. Wiemann, MD, FACP President, Clinical St. John Providence Physician Network Detroit, Michigan

# Multiple Myeloma: Changing & Emerging Treatment Paradigms



### Kenneth Anderson, MD

PAST PRESIDENT AMERICAN SOCIETY OF HEMATOLOGY 2017

Kraft Family Professor,

Harvard Medical School, Myeloma Program Director and Chief, Division of Hematologic Neoplasia, Dana Faber Cancer Institute, Boston, MA

### Genomic Based Treatment of Waldenström's Macroglobulinemia



### Steven P. Treon, MD, PhD

Director, Bing Center for Waldenström's Macroglobulinemia Professor of Medicine, Harvard Medical School Boston, MA

## Indolent Lymphomas and Hodgkin's Lymphoma: Achieving Curability



### Bruce D. Cheson, MD

Professor of Medicine, Head of Hematology and Director of Hematology Research, Deputy Chief, Division of Hematology/Oncology, Lombardi Comprehensive Cancer Center. Georgetown University, Washington DC

### <u>Aggressive B and T Cell Lymphomas:</u> Chemotherapy, Antibodies, Immunotherapy



### John P. Leonard, M.D

Richard T. Silver Distinguished Professor of Hematology and Medical Oncology Weill Cornell Medical College Professor of Medicine Weill Cornell Medical College New York, New York

## Chronic Lymphocytic Leukemia: How to Treat in 2019



### Jennifer Woyach, MD

Associate Professor Of Medicine, Ohio State University College of Medicine, Columbus, OH,

## Chronic Myeloid Leukemia: Targeted Therapy in 2019



### **Richard A. Larson, MD**

Professor of Medicine, Director of the Hematologic Malignancies Clinical Research Program, University of Chicago, Chicago, Illinois

## Not so Benign Hematology: Complementopathies and Aplastic Anemia



### **Robert Brodsky, MD**

Director, Division of Hematology Professor of Medicine and Oncology The Johns Hopkins Family Professor, Johns Hopkins University, Baltimore, MD

### Benign Hematology: Cytopenias, Clotting, and Bleeding



### **Craig M Kessler, MD**

Professor of Medicine and Pathology Director of Division of Coagulation, Department of Laboratory Medicine and Director of Therapeutic and Cellular Apheresis Unit Director of the Comprehensive Hemophilia and Thrombophilia Treatment Center, Georgetown University, Washington, DC

## **Myeloproliferative Neoplasms: Genetic Prognostication and Current Treatment**



### Ayalew Tefferi, MD, Ph.D

Professor of Medicine Mayo Clinic, Rochester, MN

## Practical Considerations in the Treatment of Polycythemia Vera and Myelofibrosis



### Angela Fleischman, MD PhD

Assistant Professor of Medicine University of California, Irvine Irvine, California

## **Acute Lymphoblastic Leukemia:** Emerging and Targeted Therapies



### Hagop Kantarjian, M.D.

Professor and Samsung Distinguished Leukemia Chair, Department of Leukemia The University of Texas MD Anderson Cancer Center, Houston, TX

## Acute Myeloid Leukemias: Treatment Options in 2019



### Martin S. Tallman

Professor of Medicine Chair of the Leukemia Committee of the Eastern Cooperative Oncology Group (ECOG) Weill Cornell Medical College Chief, Leukemia Service Memorial Sloan Kettering Cancer Center, New York

## Myelodysplastic Syndrome; How Much Further Do We Have to Travel?



### **Richard Stone**, MD

Professor of Medicine Chair Leukemia Committee ALLIANCE Chief of Staff and Director of Translational Research for the Adult Leukemia Program at Dana-Farber, and Harvard Medical School, Boston, MA

## Hematopoietic Stem Cell Transplantation: How I Treat and When I Transplant in 2019



### **Richard Childs, MD, RADM**

Clinical Director, National Heart, Lung, and Blood Institute (NHLBI), Section Chief and Senior Investigator, Laboratory of Transplantation Immunotherapy, Rear Admiral, United States Public Health Service, National Institutes of Health, Bethesda, MD

### Nursing and Allied Health Symposium

### Moderators: Donna M. Birhiray, OTR, MBA Thalia Hammond



Kristi Orbaugh, RN, MSN, RNP, AOCN Community Hospital Oncology Physicians Indianapolis, IN.



David Reeves, PharmD, BCOP Associate Professor, Butler University and Clinical Pharmacist at Franciscan Hospital, Indianapolis, IN.



Michelle Wright-Mast, NP-C Hematology Oncology of Indiana PC. St Vincent Hospital Indianapolis, IN

### **T. Howard Lee Keynote Lecture Emerging Targeted Therapies for Follicular Lymphomas: A Future without Chemotherapy**



**Pier Luigi Zinzani, MD, PhD** Professor Department of Experimental, Diagnostic and Specialty Medicine University of Bologna Bologna, Italy



T. HOWARD LEE, MD Founder and President Emeritus, Hematology Oncology of Indiana, PC Indianapolis, IN

## Inaugural Hematologic Malignancies Town Hall



Sonali Smith, MD Professor of Medicine , The University of Chicago, and Director of the Lymphoma Program. Chicago, IL



Harry Erba, MD, PhD Professor of Medicine Director, Leukemia Program Duke University Durham, NC



Morie Gertz, MD Professor of the Art of Medicine and Chair Emeritus Department of Medicine, Mayo Clinic, Rochester, MN.

## **SAVE THIS DATE !**

## 17<sup>th</sup> Annual Indy Hematology Review 2020 (http://www.indyhematologyteview.com)



March 21<sup>th</sup>, 2020 Westin Indianapolis, Indianapolis, Indiana, 46204



## Announcements and Acknowledgments

**16<sup>th</sup> Annual Indy Hematology Review** Saturday, March 9, 2019 | 7:00 am - 8:30 pm

JW Marriott Indianapolis 10 S. West Street Indianapolis, IN 46204



## And The Winner is ....

