

statistics were used for clinical and histological variables. Survival rates were estimated with Kaplan-Meier and variables compared with log-rank test.

Results: 498 pts with cHL from 7 PuI and PrI were analyzed. Median FU: 37.4 months (m). Pts characteristics: Table 1. Median time to FL initiation: 22 days, shorter in PrI ($p=0.0027$). 96.5% received ABVD as FL. 17.1% required dose modifications or delays. Complete remission (CR) rate: 83.4% (higher in PrI) and partial remission (PR): 6.3%. 85.4% had negative end of treatment (EOT) PET. 70% had an interim PET (i-PET) exam, 83.8% achieved metabolic CR but only 15.5% were treated with PET-adapted strategies (6.5% deescalated to AVD). Anemia, neutropenia and thrombocytopenia were found in 28.5%, 56.4% and 7.2%, respectively. Non-hematologic toxicities were observed in 28.6% (lung toxicity in 41 pts). 51 pts had primary refractory disease and 69 relapsed. 65 pts died, due to lymphoma progression (34) and toxicity (31). 2 and 5 year OS rate: 91% and 85%. There was no difference in OS between PrI and PuI ($p=0.27$). 5 year PFS rate: 76%. Every day of delay in initiating FL increased 0.89 (CI95% 0.6–1.8) the risk of PR or progressive disease after FL. On univariate analysis: women, age <60, non-bulky disease, normal ESR, stage I-III, favorable prognostic disease, Charlson score <3 and absence of extranodal were associated with better outcomes. On multivariate analysis Charlson score and EOT PET scan remained independent predictors of OS with HR of 1.2 (CI95% 1.1–1.7; $p=0.001$) and 2.3 (CI95% 1.7–3.2; $p<0.0001$), respectively.

Conclusions: This is one of the largest retrospective cohorts reported in cHL. ABVD is the FL regimen of choice in our country. It was well tolerated but not exempt from toxicity. Despite wide use of i-PET, only 15.5% received PET-guided treatment. The use of response-adapted strategies in our population should be strongly considered.

Variable	
Age	
Median (IQR) – yr.	34.5 (25–54)
Female gender – n°. (%)	239 (47.9)
Ann Arbor stage – %	
I	4.8
II	47.3
III	19.2
IV	28.6
Bulky disease – n°. (%)	164 (32.9)
B symptoms – n°. (%)	294 (59)
Extranodal involvement – n°. (%)	173 (34.7)
Charlson score, median (IQR)	2 (0–2)
Risk group – %	
Early favorable	15.8
Early unfavorable	36.3
Advanced favorable	15.6
Advanced unfavorable	32.3

Table 1: Characteristics of patients diagnosed with cHL treated in first line (n:498)

P014: PROGNOSTIC SIGNIFICANCE OF NUTRITIONAL INDEXES (CONUT AND PNI) IN CLASSICAL HODGKIN LYMPHOMA PATIENTS

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Background: Several recent studies have shown the clinical significance of poor nutritional status assessed by Prognostic Nutritional Index (PNI) and Controlling Nutritional status (CONUT) on patients (pts) with solid and hematological malignancies, but its impact on classical Hodgkin lymphoma (cHL) is not established.

Aim: To evaluate the prognostic value of baseline PNI and CONUT in cHL.

Methods: Retrospective analysis of adult pts with cHL, diagnosed between 1990 and 2017 and treated with curative intent with ABVD, escalated BEACOPP or ABVD hybrid regimen and with available data on all PNI variables. The cutoff point for PNI was evaluated using ROC statistics. The association between both scores and progression free survival (PFS) and overall survival (OS) was performed by Cox regression.

Results: 318pts were included, 46.2% male, with median age of 32 (18–80). Of these, 246 (77.3%) had nodular sclerosis and 213 (67%) had advanced stage cHL (GHSG classification). Information on BMI was available for 187pts (median 23.5kg/m²) and 3.2% had BMI<18.5kg/m².

Mean baseline PNI was 47.2 (SD±8.7) and median PFS of 364 months (m). After ROC statistics, the optimal cutoff for PNI to predict 5-year (y) PFS was 47 (Sensitivity 36%, Specificity 47%, AUC 0,377). PNI≥47 was significantly associated with an improved PFS (HR 0.42; $p<0.001$) in univariate analysis, with PFS 5y of 69% for PNI<47 and 82% for PNI≥47. In advanced stages, PNI≥47 retains its prognostic value, even after adjusting for IPS (HR 0.059; $p=0.034$).

After a median follow-up of 145m, median OS was not reached. PNI≥47 was significantly associated with an improved OS (HR 0.35; $p<0.001$) in univariate analysis but lost its significance in a multivariate model. CONUT was calculated in 191pts (median follow-up 163m), with a median score of 2 (0–9) and most pts with normal/mild risk (86.9%). Higher values of CONUT were associated with poorer PFS (HR 1.13; $p=0.040$) and OS (HR 1.26; $p=0.001$), despite its categorization not being a significant predictor of PFS (HR 1.30; $p=0.150$). The four categories of CONUT were predictors for OS (HR 2.04; $p=0.001$).

Conclusion: In our cohort, baseline poor nutritional status, calculated with PNI or CONUT, was associated with significantly worse prognosis in cHL. Categorization of CONUT was not a significant predictor of PFS most likely due to a shorter follow-up and fewer pts included. Further external validation of these scores in cHL is warranted.

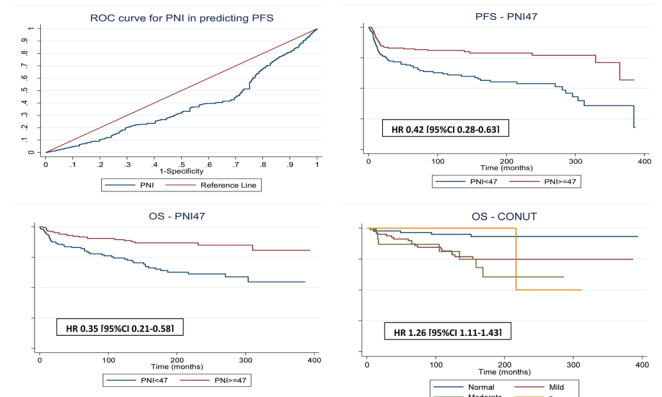


Figure 1: ROC curve for PNI in predicting progression-free survival and Overall and progression-free survival according to PNI and CONUT scores.

P015: REAL WORLD ESCALATED BEACOPP DELIVERS SIMILAR OUTCOMES TO ESCALATED BEACOPP WHILE POTENTIALLY REDUCING HAEMATOPOIETIC AND REPRODUCTIVE TOXICITY

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