ELSEVIER

Blood 142 (2023) 5828–5829

The 65th ASH Annual Meeting Abstracts

ONLINE PUBLICATION ONLY

613.ACUTE MYELOID LEUKEMIAS: CLINICAL AND EPIDEMIOLOGICAL

Association between Cytogenetic Risk and Obesity in Patients with Acute Myeloid Leukemia (AML): A Meta-Analysis

Raed Benkhadra, MD[1], *Iuliia Kovalenko, MD*[2], *Ekaterina Proskuriakova, MD*[3], *Tarek Nayfeh, MD*[4], *Michael Auster, DO*[1], *Leslie Hassett*[5], *Supreet Kaur, MD*[1]

[1] University of Texas Health San Antonio MD Anderson Cancer Center, San Antonio, TX
[2] University of Pittsburgh Medical Center (UPMC) Harrisburg Hospital, Harrisburg, PA
[3] Mount Sinai Hospital, Chicago, IL
[4] Mayo Clinic, Evidence-Based Practice Center, Rochester, MN
[5] Mayo clinic libraries, Rochester, MN

Background:
The global obesity epidemic has been well characterized over decades of research, linking increased body mass index (BMI) ≥ 25 to multiple medical conditions including increased cancer risk, morbidity, and mortality. The International Agency for Research on Cancer (IARC) group found a positive correlation between obesity to 13 cancers and multiple studies suggest leukemia should be added to this list. The causation behind this risk in leukemia however is not well understood and the prognostic factors have not been well characterized. Acute Myeloid Leukemia (AML) specifically defines risk based on cytogenetics between favorable, intermediate and unfavorable which significantly affects treatment decisions. The link between obesity and cytogenic risk factors in AML have not previously been thoroughly evaluated. To address this knowledge gap, we conducted a comprehensive meta-analysis aiming to evaluate the cytogenetic risk with obesity and prognostic significance in patients with AML.

Methods:
A systematic search was conducted across major databases until June 2023, using tailored search structures, subject headings, and keywords developed in collaboration with a medical research librarian. Two investigators independently screened the studies for inclusion. The inclusion criteria encompassed retrospective and prospective clinical studies that reported on the association between established cytogenetic classifications and standardized measures of excessive weight (BMI 25-30) and obesity (BMI ≥ 30). Meta-analysis was conducted using restricted maximum likelihood random-effects model.

Results:
From an initial pool of 1673 identified studies, eleven studies met the inclusion criteria, encompassing a total of 5887 patients. Among these patients, 1363 met the criteria for obesity (BMI ≥ 30) and 2176 met the criteria for excessive weight (BMI 25-30). The median patient's age was 54 (ranging from 15 to 89). The included studies originated from diverse geographic regions, including the USA, Japan, and France. Baseline patient characteristics are summarized in Table 1. Compared to overweight patients, our meta-analysis revealed a significant association between obesity and higher odds of unfavorable/intermediate cytogenetic risk (odds ratio [OR] 1.27, 95% CI [1.04; 1.54]), as seen in Figure 1. However, no meaningful statistical inference could be made regarding the association between obesity and specific gene mutations (such as FLT3, NPM1, CEBPa, TET2, IDH1, IDH2, RUNX1, and KRAS) due to the limited number of studies and small number of patients involved.

Conclusion:
Our meta-analysis demonstrates a significant association between obesity and unfavorable/ intermediate cytogenetic risk in patients with AML. Although the precise biological mechanisms remain elusive, several potential explanations have been proposed. These include the impact of obesity leading to chronic low-grade inflammation, altered adipokine secretion and insulin resistance, and metabolic changes on leukemic cell growth and survival. Despite the limitations of unknown cytogenetic risk in 590 patients and limited mutation data in some studies, our findings suggest that obesity may impact the prognosis of AML. Future research should address these limitations and provide a more comprehensive understanding of the intricate interplay between obesity, cytogenetics, and AML prognosis.

Disclosures No relevant conflicts of interest to declare.

First Author, last name., year.	Patients, n	Male %	Mean age		Patients, n	Favorable cytogenetics, n	Intermediate risk cytogenetics, n	Unfavorable risk cytogenetics, n	FLT3, n	CEBPa, n	NPM1 , n
Foran et al., 2022	1088	53.3	NA	Obese	358	37	150	92	59	14	50
				excessive weight	730	65	217	174	109	22	112
Ando et al., 2017	369	59.8	49	Obese							
				excessive weight	101	27	48	26			
Tavitian et al., 2016	619	57.9	59	Obese	93	7	66	18	12	4	22
				excessive weight	191	23	114	48	34	6	36
Voshtina et al., 2019	86	NA	NA	Obese	41	13	13	15			
				excessive weight	45	10	20	16			
Cahu et al., 2017	823	51.5	47.1	Obese	51	6	30	13	6		7
				excessive weight	91	15	48	21	8		16
Dakal et al., 2020	314	53.1	NA	Obese	121	10	58	38	14		11
				excessive weight	106	9	61	35	19		11
Dombrowski et al., 2019	57	47.3	NA	Obese	15	3	6	55	1		2
				excessive weight							
Gallagher et al., 2011	247	NA	55.6	Obese	85	13	36	23			
				excessive weight	81	15	31	24			
Lin et al., 2013	63	47.6	55	Obese	21	6	10	5	2		2
				excessive weight							
Medeiros et al., 2011	1974	NA	57	Obese	493	148	123	119	12		
				excessive weight	750	248	286				
Wenzell et al., 2013	247	NA	55.6	Obese	85	13	36	23		4	22
				excessive weight	81	15	31	24		6	

Table 1. Baseline patient characteristics.

N – number of patients

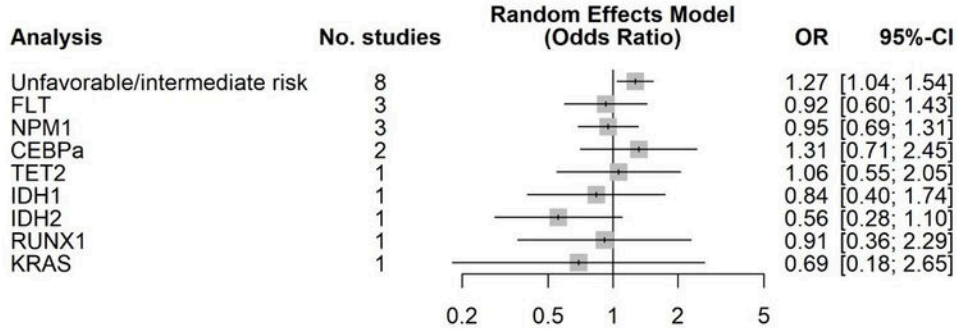

Figure 1. Association between obesity and AML cytogenetic profile

OR – odds ratio

Figure 1

https://doi.org/10.1182/blood-2023-178245