CO181

Patient Characteristics, Clinical Management and Costs of Patients with Newly Diagnosed Acute Myeloid Leukaemia from Finland

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INTRODUCTION

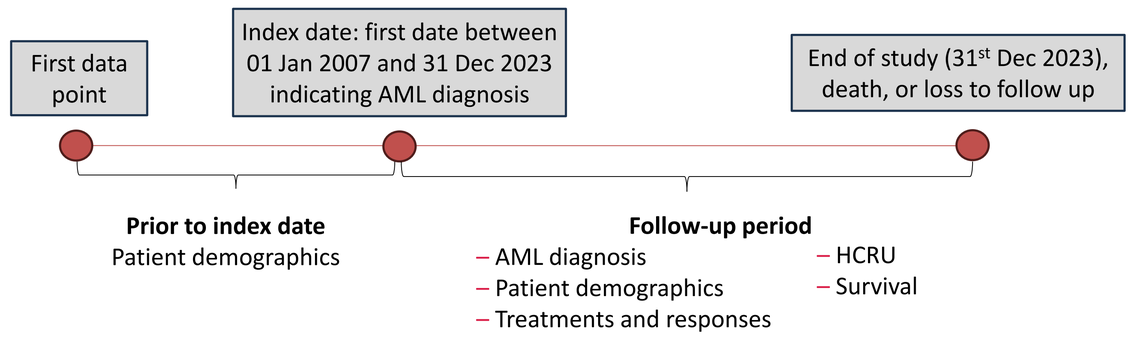
- Acute myeloid leukaemia (AML) arises from a range of genetic abnormalities, which may lead to variations in patient survival rates and need for individualised treatments[1]
- The FMS-like tyrosine kinase 3 (*FLT3*) gene has been identified as one of the most common genetic alterations in adult AML, occurring in ~30% of AML cases[2]
- *FLT3* mutations can present with high disease burden, poor prognosis and a negative impact on the management of patients with AML[2]
- Greater understanding of current clinical strategy, outcomes and resource utilisation may support development of appropriate treatment pathways for patients with AML

Objective

- To describe patient characteristics, treatment regimens, overall survival (OS) and healthcare resource utilisation (HCRU) of newly diagnosed patients with AML.

METHODS

- This non-interventional, retrospective registry-based study included pseudonymised clinical/medical data of newly diagnosed patients with AML (diagnosed 2007–2023) in the Hospital District of Helsinki and Uusimaa



- **Primary objective:** describe patient demographic and clinical characteristics of newly diagnosed AML patients
- **Secondary objectives included:**
  - *FLT3* mutation testing rate
  - Treatment regimens
  - OS
  - HCRU

RESULTS

Patient demographic and clinical characteristics

- Data were extracted for 866 newly diagnosed patients with AML (overall cohort)
- In the overall cohort, 591/866 (68.2%) patients underwent *FLT3* testing and 128/591 (21.7%) had a *FLT3* (TKD or ITD) alteration (*FLT3*+; '*FLT3* cohort')
  - Patients in the *FLT3* cohort were younger at diagnosis compared to the overall cohort (**Table 1**).
  - Baseline FLT3 inhibitor use was more frequent in the *FLT3* vs overall cohort (**Table 1**)

Treatment regimens

- Over the course of the study, FLT3 inhibitor use was more frequent in the *FLT3* vs overall cohort **(Table 2)**
- Compared with the overall cohort, a greater proportion of the *FLT3* cohort received high intensity chemotherapy (HIC) and allogeneic haematopoietic stem cell transplants (HSCT) **(Table 2)**

OS

- In patients treated with HIC, median OS (95% CI) was 5.1 (3.7–9.8) and 1.5 (1.3–8.5) years in patients with *FLT3* wildtype (FLT3wt) and *FLT3*+, respectively **(Fig. 1)**

HCRU

- The *FLT3* cohort vs overall cohort had higher total healthcare cost accumulation excluding treatments per patient (€157,669 vs €119,703, respectively)
  - In particular, costs associated with inpatient care and procedures were elevated **(Fig. 2)**
- In both cohorts, the initial stages (first 12 months) of treatment incurred the highest costs, before costs subsequently stabilised **(Fig. 2)**

Table 1. Patient demographics and clinical characteristics at baseline

	Overall cohort n=866	*FLT3* cohort n=128
Male, n (%)	417 (48.2)	61 (47.7)
Age (years), mean (SD)	60.0 (21.0)	57.7 (17.6)
FLT3 inhibitor, n (%)	37 (4.3)	26 (20.3)
Allo-HSCT, n (%)	220 (25.4)	49 (38.3)

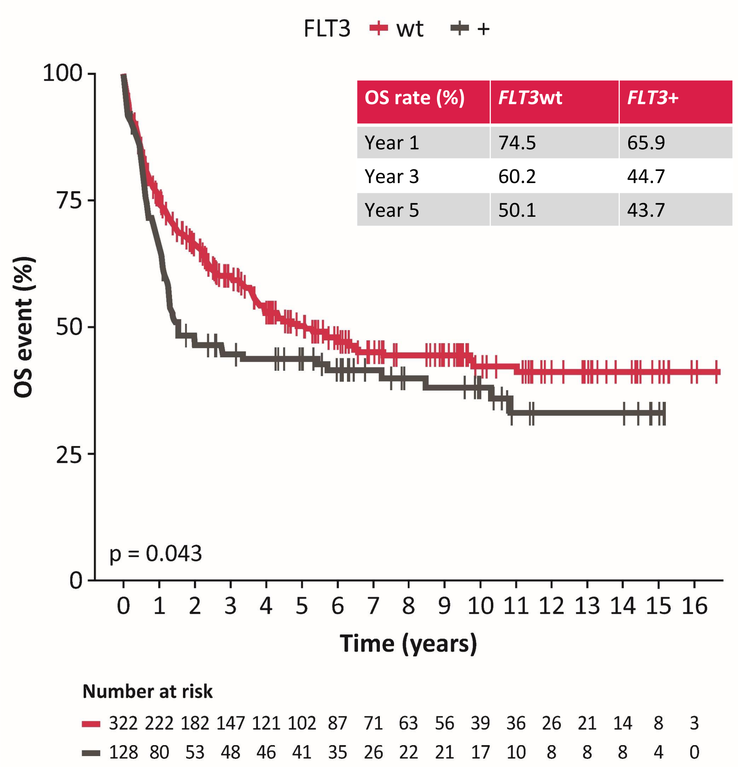
allo-HSCT, allogeneic haematopoietic stem cell transplants; *FLT3*, FMS-like tyrosine kinase 3.; SD, standard deviation

Table 2. Treatment regimens at any time of the disease course

		Overall cohort n=866	*FLT3* cohort n=128
Treatment regimens	HIC, n (%)	494 (57.0)	98 (76.6)
	HMA, n (%)	327 (37.8)	59 (46.1)
	HMA-VEN, n (%)	103 (11.9)	19 (14.8)
	ATRA-ATO, n (%)	32 (3.7)	5 (3.9)
	Other, n (%)	162 (18.7)	13 (10.2)
	Palliative, n (%)	342 (39.5)	44 (35.4)
FLT3 inhibitor	Midostaurin, n (%)	27 (3.1)	20 (15.6)
	Sorafenib, n (%)	20 (2.3)	16 (12.5)
	Gilteritinib, n (%)	14 (1.6)	10 (7.8)
Allo-HSCT	Allo-RIC, n (%)	20 (2.3)	4 (3.1)
	Allo-MAC, n (%)	223 (25.8)	47 (36.7)

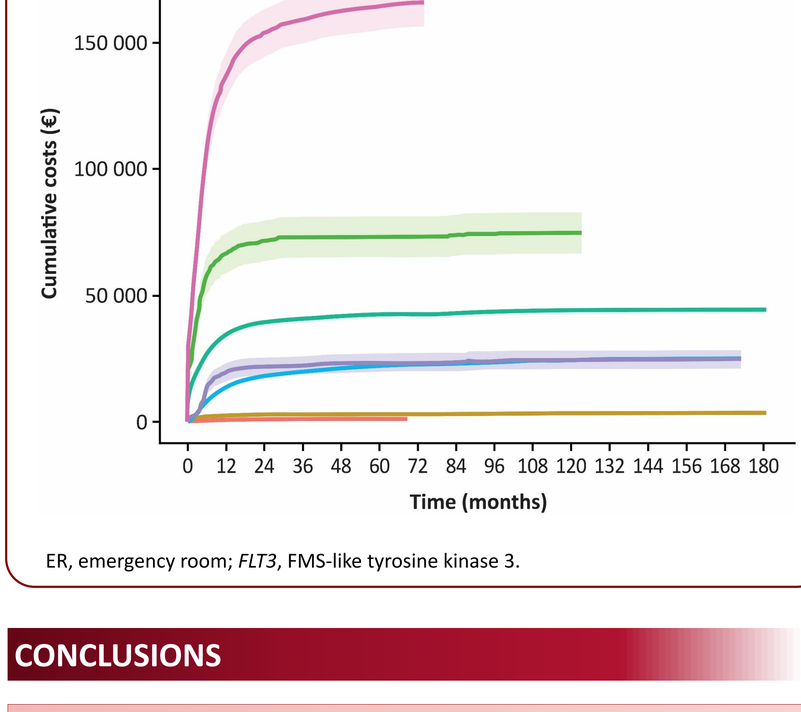
allo-HSCT, allogeneic haematopoietic stem cell transplants; allo-MAC, myeloablative conditioning prior to allogeneic hematopoietic stem cell transplant; allo-RIC, reduced intensity conditioning prior to allogeneic hematopoietic stem cell transplant; ATRA-ATO, all-trans retinoic acid and arsenic trioxide; *FLT3*, FMS-like tyrosine kinase 3; HIC, high intensity chemotherapy; HMA, hypomethylating agent; HMA-VEN, combination of hypomethylating agent and venetoclax.

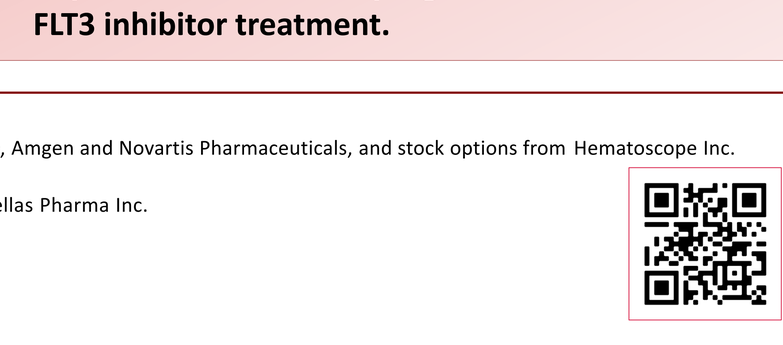
Figure 1. Overall survival of patients with AML treated with high intensity chemotherapy, according to their *FLT3* mutation status



Kaplan–Meier curve and p-value were calculated using the log-rank test and overall survival. +, mutation positive; AML, acute myeloid leukaemia; *FLT3*, FMS-like tyrosine kinase 3; OS, overall survival; wt, wild type.

Figure 2. Temporal accumulation of healthcare costs for A) overall cohort and B) *FLT3* cohort





ER, emergency room; *FLT3*, FMS-like tyrosine kinase 3.

CONCLUSIONS

- **Patients with *FLT3*+ AML were generally younger and treated more frequently with HIC and HSCT, which contributed to higher HCRU**
- **Patients with *FLT3*+ AML had shorter OS than patients with *FLT3*wt AML**
- **Further work is required to compare the cost profiles of patients with similar prognoses with or without FLT3 inhibitor treatment.**

Author disclosures
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