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Sample Information

Patient Name: 張淑晶
Gender: Female
ID No.: A223978111
History No.: 46783500
Age: 47

Ordering Doctor: DOC8919E 周逸峰
Ordering REQ.: 0BZXUCG
Signing in Date: 2022/09/22

Path No.: S111-97900
MP No.: MY22027
Assay: Oncomine Myeloid Assay
Sample Type: Bone Marrow
Bone Marrow Aspirating Date: 2022/09/13

Reporting Doctor: DOC5466K 葉奕成 (Phone: 8#5466)

Note:

Sample Cancer Type: Chronic Myelomonocytic Leukemia

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Relevant Chronic Myelomonocytic Leukemia Variants

Gene	Finding
ASXL1	***ASXL1 p.(Y591*) c.1772_1773insAAGGTCAGCCCACTTA***

Relevant Biomarkers

Tier	Genomic Alteration	Relevant Therapies (In this cancer type)	Relevant Therapies (In other cancer type)	Clinical Trials
IA	*ASXL1 p.(Y591*) c.1772_1773insAAGGTCAGCCCACTTA* ASXL transcriptional regulator 1 Allele Frequency: 23.84%	None	allogeneic stem cells azacitidine cytarabine cytarabine + daunorubicin cytarabine + daunorubicin + etoposide	0

Public data sources included in relevant therapies: FDA[1], NCCN, **EMA**[2], ESMO
Public data sources included in prognostic and diagnostic significance: NCCN, ESMO

Relevant Biomarkers (continued)

Tier	Genomic Alteration	Relevant Therapies (In this cancer type)	Relevant Therapies (In other cancer type)	Clinical Trials
			cytarabine + etoposide + idarubicin cytarabine + fludarabine + idarubicin + filgrastim cytarabine + idarubicin cytarabine + mitoxantrone decitabine gemtuzumab ozogamicin + chemotherapy venetoclax + chemotherapy	
	Prognostic significance: NCCN: Poor			
IIC	*SF3B1 p.(K666N) c.1998G>T* splicing factor 3b subunit 1 Allele Frequency: 39.52%	None	luspatercept	0

Public data sources included in relevant therapies: FDA[1], NCCN, **EMA**[2], ESMO
Public data sources included in prognostic and diagnostic significance: NCCN, ESMO

Prevalent cancer biomarkers without relevant evidence based on included data sources

*NRAS p.(Q61H) c.183A>T, PTPN11 p.(E76K) c.226G>A, IKZF1 p.(S308Cfs*4) c.922_923insGT, U2AF1 p.(Q157P) c.470A>C*

Variants (Exclude variant in Taiwan BioBank with >1% allele frequency)

DNA Sequence Variants

Gene	Amino Acid Change	Coding	Variant ID	Locus	Allele Frequency	Transcript	Variant Effect	Coverage
NRAS	p.(Q61H)	c.183A>T	COSM585	chr1:115256528	23.52%	NM_002524.5	missense	1998
SF3B1	p.(K666N)	c.1998G>T	COSM131557	chr2:198267359	39.52%	NM_012433.4	missense	1999
IKZF1	p.(S308Cfs*4)	c.922_923insGT	.	chr7:50467685	7.52%	NM_006060.6	frameshift Insertion	1996
PTPN11	p.(E76K)	c.226G>A	COSM13000	chr12:112888210	18.01%	NM_002834.5	missense	1999
ASXL1	p.(Y591*)	c.1772_1773insAAGGTCAGCCCACTTA	.	chr20:31022268	23.84%	NM_015338.6	nonsense	1204
U2AF1	p.(Q157P)	c.470A>C	COSM211534	chr21:44514777	39.40%	NM_006758.2	missense	2000
CSF3R	p.(Q776E)	c.2326C>G	.	chr1:36932224	42.40%	NM_156039.3	missense	2000
IKZF1	p.(H480Q)	c.1440C>G	.	chr7:50468205	18.10%	NM_006060.6	missense	2000
U2AF1L5	p.(Q84P)	c.251A>C	COSM211534	chr21:44514777	39.40%	NM_001320651.1	missense	2000
BCOR	p.(P483L)	c.1448C>T	.	chrX:39933151	48.52%	NM_001123385.2	missense	1999

Biomarker Descriptions

ASXL1 (ASXL transcriptional regulator 1)

Background: The ASXL1 gene encodes the ASXL transcriptional regulator 1 protein, a ligand-dependent co-activator and epigenetic scaffolding protein involved in transcriptional regulation[1,2]. ASXL1 belongs to the ASXL gene family, which also includes ASXL2 and ASXL3[2]. ASXL proteins contain a conserved c-terminal plant homeodomain (PHD) which facilitates interaction with DNA and histones[2,3]. ASXL1 influences chromatin remodeling and transcription through interaction with BAP1 and polycomb repressive complex

Biomarker Descriptions (continued)

(PRC) proteins, as well as other transcriptional activators and repressors[2,4]. In cancer, ASXL1 is the target of somatic mutations which often result in a truncated ASXL1 protein and loss of its PHD[5,6,7]. Such mutations can lead to impaired protein function and consequent upregulation of HOXA gene expression, supporting a tumor suppressor role for ASXL1[8].

Alterations and prevalence: Missense, nonsense, and frameshift mutations in ASXL1 are reported in 3-6% of de novo acute myeloid leukemia (AML), up to 36% of secondary AML, approximately 15% of myelodysplastic syndromes (MDS), up to 23% of myeloproliferative neoplasms (MPN), up to 30% of systemic mastocytosis (SM), and approximately 45% of chronic myelomonocytic leukemia (CMML)[4,9,10,11,12,13,14,15,16]. The ASXL1 G646Wfs*12 mutation accounts for over 50% of ASXL1 mutated cases in myeloid malignancies[6,11,17]. This mutation results from a single nucleotide expansion that occurs within an eight base pair guanine repeat that extends from c.1927 to c.1934. It is proposed that the high prevalence of the G646Wfs*12 variant is due to replication slippage which can occur in areas of repetitive sequence[18]. As a consequence, detection of G646Wfs*12 may result as an artifact of PCR and/or sequencing[19]. However, multiple studies observe an increase in the frequency of G646Wfs*12 in myeloid cancer relative to normal suggesting that G646Wfs*12 is a bona fide somatic mutation[9,18,20].

Potential relevance: The majority of frameshift and nonsense mutations in ASXL1 that result in protein truncation and removal of the PHD domain are considered pathogenic[21]. Mutations in ASXL1 confer poor/adverse risk in AML and are associated with poor outcomes and adverse risk[16]. Additionally, ASXL1 nonsense or frameshift mutations are independently associated with poor prognosis in MDS and CMML[22]. Moreover, ASXL1 mutations are independently associated with inferior overall survival (OS) in patients with MPN or SM[23,24].

IKZF1 (IKAROS family zinc finger 1)

Background: The IKZF1 gene encodes the IKAROS family zinc finger 1 transcription factor belonging to the family of zinc-finger DNA-binding proteins that influence gene expression through chromatin remodeling. IKZF1 was originally characterized as a lymphoid-restricted transcription factor for its critical role in lymphocyte differentiation[25]. However, it has since been observed to be involved in myeloid differentiation as well[26,27,28]. IKZF1 is a tumor suppressor and target of genetic aberrations including deletions and mutations that lead to loss of function and contribute to the development of lymphoid malignancies[29,30,31]. Specifically, IKZF1 deletion is an acquired alteration associated with the transformation of chronic myeloid leukemia (CML) to lymphoid blast crisis in BCR-ABL1 positive acute lymphoblastic leukemia (ALL)[32,33]. Similarly, IKZF1 deletion is associated with acute myeloid leukemia (AML) and transformation of myeloproliferative neoplasms (MPN) to AML[31,34,35].

Alterations and prevalence: IKZF1 aberrations, including deletions and mutations, occur in 15-20% of pediatric B-cell ALL and in greater than 75% of BCR-ABL1 positive ALL[32,36,37,38]. In adults, IKZF1 aberrations occur in about 25-35% of ALL and in approximately 65% of BCR-ABL1 positive ALL[36,39,40,41]. IKZF1 deletion has also been observed in 4% of diffuse large B-cell lymphoma (DLBCL) and 1% of pediatric AML[12,34]. IKZF1 mutations are observed in about 2% of AML and myelodysplastic syndromes (MDS)[42]. Mutations in IKZF1 usually result in expression of a protein that lacks DNA binding ability with dominant negative activity[37,38].

Potential relevance: IKZF1 mutations and deletions that result in loss of function are associated with poor prognosis and a greater chance of relapse in B-cell ALL[36,43].

NRAS (NRAS proto-oncogene, GTPase)

Background: The NRAS proto-oncogene encodes a GTPase that functions in signal transduction and is a member of the RAS superfamily which also includes KRAS and HRAS. RAS proteins mediate the transmission of growth signals from the cell surface to the nucleus via the PI3K/AKT/MTOR and RAS/RAF/MEK/ERK pathways, which regulate cell division, differentiation, and survival[44,45,46].

Alterations and prevalence: Recurrent mutations in RAS oncogenes cause constitutive activation and are found in 20-30% of cancers. NRAS mutations are particularly common in melanomas (up to 25%) and are observed at frequencies of 5-10% in acute myeloid leukemia, colorectal, and thyroid cancers[12,47]. The majority of NRAS mutations consist of point mutations at G12, G13, and Q61[12,48]. Mutations at A59, K117, and A146 have also been observed but are less frequent[49,50].

Potential relevance: Currently, no therapies are approved for NRAS aberrations. The EGFR antagonists, cetuximab[51] and panitumumab[52], are contraindicated for treatment of colorectal cancer patients with NRAS mutations in exon 2 (codons 12 and 13), exon 3 (codons 59 and 61), and exon 4 (codons 117 and 146)[50]. NRAS mutations are associated with poor prognosis in patients with low-risk myelodysplastic syndrome[22] as well as melanoma[53]. In a phase III clinical trial in patients with advanced NRAS-mutant melanoma, binimetinib improved progression free survival (PFS) relative to dacarbazine with median PFS of 2.8 and 1.5 months, respectively[54].

PTPN11 (protein tyrosine phosphatase non-receptor type 11)

Background: The PTPN11 gene encodes a tyrosine phosphatase non-receptor type 11 protein, and is also known as Src homology region 2 domain-containing phosphatase-2 (SHP-2)[55]. PTPN11 is a member of the protein tyrosine phosphatase (PTP) family that

Biomarker Descriptions (continued)

is ubiquitously expressed and regulates cellular growth, differentiation, mitotic cycle, and oncogenic transformation. PTPN11 contains two tandem N-terminal Src homology-2 domains (N-SH2 and C-SH2), a PTP catalytic domain, and uncharacterized C-terminal domain[56]. PTPN11 regulates various signaling processes including the RAS/RAF/MEK/ERK, PI3K/AKT/MTOR, and JAK/STAT pathways[57,58]. Germline mutations in PTPN11 are associated with LEOPARD syndrome and Noonan syndrome with a predisposition to juvenile myelomonocytic leukemia (JMML) or myeloproliferative neoplasms (MPN)[22,59]. Somatic mutations in PTPN11 are associated with JMML[60,61] and solid tumors such as lung, colon, and thyroid[56,62]

Alterations and prevalence: Somatic alterations in PTPN11 include mutations and amplification[59,63]. PTPN11 mutations occur in 6% of uterine carcinoma and 5% of acute myeloid leukemia (AML) cases[49]. Mutations including E76K and D61Y result in PTPN11 activation and are associated with 30% of JMML[58].

Potential relevance: Currently, no therapies are approved for PTPN11 aberrations. Somatic mutations in PTPN11 confer drug resistance to venetoclax and azacitidine in AML[64,65].

SF3B1 (splicing factor 3b subunit 1)

Background: The SF3B1 gene encodes the splicing factor 3b subunit 1 protein, a core component of the U2 small nuclear ribonucleoprotein (snRNP) complex of the spliceosome responsible for RNA splicing. SF3B1 is involved in recognition of the branch point sequence during selection of the 3' splice site. Recurrent somatic mutations in SF3B1 and other components of the splicing machinery including SRSF2, U2AF1, and ZRSR2, are common in myelodysplasia. These components experience mutations in a mutually exclusive manner suggesting a common impact on RNA splicing and the pathogenesis of myelodysplasia[66]. SF3B1 mutations are believed to contribute to aberrant post-translational inactivation of the regulatory complex PPP2R5A of protein phosphatase 2A (PP2A), leading to the activation and stabilization of MYC activation and impairing apoptosis[67].

Alterations and prevalence: SF3B1 mutations occur in the majority (70-80%) of myelodysplastic syndromes (MDS) with ring sideroblasts (RS) and at lower frequency in other myeloid neoplasms including MDS without RS (7%), chronic myelomonocytic leukemia (5-6%), therapy-related acute myeloid leukemia (AML) or AML with MDS features (5%), and de novo AML (3%)[66,68,69]. Recurrent somatic SF3B1 mutations are also common in certain solid cancers including uveal melanoma (20-30%) and breast cancer (2%) and at lower frequencies in diverse cancer types[12,49,70,71,72,73,74,75]. Cancer-associated recurrent missense mutations in SF3B1 occur within the HEAT repeat domains 5-9 at codon positions R625, K666, K700, G742, and D781[76]. The functional significance of recurrent SF3B1 mutations is to alter branch point selection thus inducing cryptic 3' splice site selection[76,77,78]

Potential relevance: Currently, no therapies are approved for SF3B1 aberrations. SF3B1 mutations are associated with aggressive disease and shorter survival in patients diagnosed with chronic lymphocytic leukemia (CLL)[79]. Investigational inhibitors of the spliceosome are in early clinical development[80,81].

U2AF1 (U2 small nuclear RNA auxiliary factor 1)

Background: The U2AF1 gene encodes the U2 small nuclear RNA auxiliary factor 1 protein that belongs to the splicing factor SR family of genes involved in RNA splicing[1,82]. U2AF1, also known as U2AF35, mediates the recruitment of the U2AF complex to the 3' end of that pre-mRNA that is being spliced[83]. U2AF1 is the smaller subunit of the U2 auxiliary factor and along with the larger subunit, U2AF65 regulates the removal of introns from pre-mRNAs to produce mature mRNAs for translation during protein synthesis[84]. Mutations in U2AF1 alter the differential splicing of genes that are involved in various biological pathways, including DNMT3B in DNA methylation, ATR along with FANCA in DNA damage response, and H2AFY in X-chromosome inactivation[85]. Spliceosomal genes such as U2AF1 are common targets of somatic mutations in myelodysplastic syndrome (MDS) and are associated with the progression of MDS to acute myeloid leukemia (AML)[85,86,87].

Alterations and prevalence: Recurrent mutations in U2AF1 occur at S34 and Q157 and are observed in 8-12% of MDS[22]. Somatic mutations in U2AF1 are also observed in 10% of uterine carcinoma, 4% of AML, as well as 2% of lung adenocarcinoma and stomach adenocarcinoma[49].

Potential relevance: U2AF1 mutations including S34 and Q157 are associated with poor prognosis in MDS[22]. U2AF1 mutations are associated with inferior overall survival in primary myelofibrosis (PMF)[23]. Specifically, the Q157 mutation is associated with a significantly shorter overall survival than U2AF1 S34 mutated and U2AF1 unmutated MPN[23].

Relevant Therapy Summary

● In this cancer type ○ In other cancer type ◑ In this cancer type and other cancer types ✖ No evidence

ASXL1 p.(Y591*) c.1772_1773insAAGGTCAGCCCACTTA

Relevant Therapy	FDA	NCCN	EMA	ESMO	Clinical Trials*
Allogeneic hematopoietic stem cell transplantation	✖	○	✖	✖	✖
azacitidine	✖	○	✖	✖	✖
cytarabine	✖	○	✖	✖	✖
cytarabine + daunorubicin	✖	○	✖	✖	✖
cytarabine + daunorubicin + etoposide	✖	○	✖	✖	✖
cytarabine + etoposide + idarubicin	✖	○	✖	✖	✖
cytarabine + fludarabine + idarubicin + filgrastim	✖	○	✖	✖	✖
cytarabine + idarubicin	✖	○	✖	✖	✖
cytarabine + mitoxantrone	✖	○	✖	✖	✖
decitabine	✖	○	✖	✖	✖
gemtuzumab ozogamicin + cytarabine + daunorubicin	✖	○	✖	✖	✖
venetoclax + azacitidine	✖	○	✖	✖	✖
venetoclax + cytarabine	✖	○	✖	✖	✖
venetoclax + decitabine	✖	○	✖	✖	✖

SF3B1 p.(K666N) c.1998G>T

Relevant Therapy	FDA	NCCN	EMA	ESMO	Clinical Trials*
luspatercept	✖	✖	✖	○	✖

Relevant Therapy Details

Current NCCN Information

● In this cancer type ○ In other cancer type ◑ In this cancer type and other cancer types

NCCN information is current as of 2022-08-01. For the most up-to-date information, search www.nccn.org.
For NCCN International Adaptations & Translations, search www.nccn.org/global/international_adaptations.aspx.

ASXL1 p.(Y591*) c.1772_1773insAAGGTCAGCCCACTTA

○ cytarabine + daunorubicin

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 1

Population segment (Line of therapy):
- (Induction therapy); Other recommended intervention

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

○ cytarabine + daunorubicin + etoposide

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 1

Population segment (Line of therapy):
- (Induction therapy); Other recommended intervention

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

○ cytarabine + etoposide + idarubicin

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 1

Population segment (Line of therapy):
- (Induction therapy); Other recommended intervention

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

○ cytarabine + idarubicin

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 1

Population segment (Line of therapy):
- (Induction therapy); Other recommended intervention

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

ASXL1 p.(Y591*) c.1772_1773insAAGGTCAGCCCACTTA (continued)

Allogeneic hematopoietic stem cell transplantation

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2A

Population segment (Line of therapy):
- (Consolidation therapy)

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

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cytarabine

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2A

Population segment (Line of therapy):
- (Consolidation therapy)

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

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cytarabine + daunorubicin

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2A

Population segment (Line of therapy):
- (Consolidation therapy)

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

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cytarabine + daunorubicin

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2A

Population segment (Line of therapy):
- (Induction therapy); Preferred intervention

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

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cytarabine + idarubicin

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2A

Population segment (Line of therapy):
- (Induction therapy); Preferred intervention

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

ASXL1 p.(Y591*) c.1772_1773insAAGGTCAGCCCACTTA (continued)

cytarabine + mitoxantrone

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2A

Population segment (Line of therapy):
- (Induction therapy); Preferred intervention

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

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decitabine

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2A

Population segment (Line of therapy):
- (Induction therapy)

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

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gemtuzumab ozogamicin + cytarabine + daunorubicin

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2A

Population segment (Line of therapy):
- (Induction therapy); Preferred intervention

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

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venetoclax + azacitidine

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2A

Population segment (Line of therapy):
- (Induction therapy)

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

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venetoclax + cytarabine

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2A

Population segment (Line of therapy):
- (Induction therapy)

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

ASXL1 p.(Y591*) c.1772_1773insAAGGTCAGCCCACTTA (continued)

venetoclax + decitabine

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2A

Population segment (Line of therapy):
- (Induction therapy)

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

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azacitidine

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2B

Population segment (Line of therapy):
- (Induction therapy)

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

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cytarabine + daunorubicin + etoposide

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2B

Population segment (Line of therapy):
- (Induction therapy); Other recommended intervention

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

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cytarabine + etoposide + idarubicin

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2B

Population segment (Line of therapy):
- (Induction therapy); Other recommended intervention

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

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cytarabine + fludarabine + idarubicin + filgrastim

Cancer type: Acute Myeloid Leukemia **Variant class:** ASXL1 mutation

NCCN Recommendation category: 2B

Population segment (Line of therapy):
- (Induction therapy); Other recommended intervention

Reference: NCCN Guidelines® - NCCN-Acute Myeloid Leukemia [Version 2.2022]

Current ESMO Information

● In this cancer type ○ In other cancer type ◑ In this cancer type and other cancer types

ESMO information is current as of 2022-08-01. For the most up-to-date information, search www.esmo.org.

SF3B1 p.(K666N) c.1998G>T

○ luspatercept

Cancer type: Myelodysplastic Syndrome **Variant class:** SF3B1 mutation

ESMO Level of Evidence/Grade of Recommendation: I / A

Population segment (Line of therapy):
- Refractory (Second-line therapy)

Reference: ESMO Clinical Practice Guidelines - ESMO-Myelodysplastic Syndromes [Annals of Oncology (2020), https://doi.org/10.1016/j.annonc.2020.11.002]

Prognostic Details

Current NCCN Information

NCCN information is current as of 2022-08-01. For the most up-to-date information, search www.nccn.org.
For NCCN International Adaptations & Translations, search www.nccn.org/global/international_adaptations.aspx.

ASXL1 p.(Y591*) c.1772_1773insAAGGTCAGCCCACTTA

Prognostic significance: NCCN: Poor

Cancer type: Chronic Myelomonocytic Leukemia **Variant class:** ASXL1 truncating mutation

NCCN Recommendation category: 2A

Summary:
- NCCN Guidelines® independently associate the biomarker with poor prognosis in MDS and CMML

Reference: NCCN Guidelines® - NCCN-Myelodysplastic Syndromes [Version 3.2022]

Signatures

Testing Personnel:

Laboratory Supervisor:

Pathologist:

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