Learning objectives

Following review of these slides the reader should understand:

- How ASCT fits into the treatment landscape for the management of CML
- The purpose of ASCT and appropriate patient selection
- An overview of key safety and efficacy outcomes in patients with CML undergoing ASCT
- An overall summary of the clinical implications and strategies of ASCT in CML
How ASCT fits into the current treatment landscape for CML

- Long-term follow-up analyses indicate that by 5 years an estimated 40% of patients with CML require a change of therapy due to resistance, intolerance, or other indications. Developing resistance to TKIs and requiring multiple changes of therapies has been shown to decrease survival probability.

- ASCT was developed to replace the blood-forming cells that are destroyed during high-dose chemotherapy. Originally considered as frontline treatment in the early 90s, ASCT shifted to third- or fourth-line due to the introduction of TKIs in the 2000s.

- Candidates for ASCT in 2022 need to meet certain criteria: patients in CP with resistance or intolerance to multiple TKIs or patients with advanced disease after conventional therapy such as TKIs with or without chemotherapy. Patients should ideally have a low EBMT score.

ASCT, allogeneic stem cell transplantation; CML, chronic myeloid leukemia; CP, chronic phase; DMR, deep molecular response; EBMT, European Society for Bone and Marrow Transplantation; OS, overall survival; TFR, treatment-free remission; TKI, tyrosine kinase inhibitors.

## TKI response according to guidelines

- Failure to respond to TKIs is defined by specific guidelines (ELN, NCCN, and ESMO)\(^1-4\)

<table>
<thead>
<tr>
<th>Time</th>
<th>ELN (2020)(^2)</th>
<th>NCCN (2021)(^3)</th>
<th>ESMO (2017)(^4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 months</td>
<td>(\text{BCR-ABL1} &gt;10% \text{in PCR test if confirmed within 1–3 months})</td>
<td>(\text{BCR-ABL1} &gt;10%)</td>
<td>No CHR (\text{Ph} &gt;95%)</td>
</tr>
<tr>
<td>6 months</td>
<td>(\text{BCR-ABL1} &gt;10% \text{in PCR test})</td>
<td>(\text{BCR-ABL1} &gt;10%)</td>
<td>(\text{Ph} &gt;35%) (\text{BCR-ABL} &gt;10%)</td>
</tr>
<tr>
<td>12 months</td>
<td>(\text{BCR-ABL1} &gt;1% \text{in PCR test})</td>
<td>(\text{BCR-ABL1} &gt;1%)</td>
<td>(\text{Ph} &gt;1%) (\text{BCR-ABL} &gt;1%)</td>
</tr>
<tr>
<td>Then and at any time during treatment</td>
<td>(\text{BCR-ABL1} &gt;1% \text{in PCR test}) High-risk ACAs in cells with the Philadelphia chromosome</td>
<td>-</td>
<td>Relapse, loss of MMR ((\text{BCR-ABL1} &gt;0.1%))</td>
</tr>
</tbody>
</table>

AC, additional chromosomal aberration; CHR, complete hematologic response; ELN, EuropeanLeukemia.net; ESMO, European Society for Medical Oncology; MMR, major molecular remission; NCCN, National Comprehensive Cancer Network; PCR, polymerase chain reaction; Ph, Philadelphia; TKI, tyrosine kinase inhibitor.

# Recommendations for ASCT by disease phase

<table>
<thead>
<tr>
<th>Disease phase</th>
<th>Disease characteristics warranting the need to consider ASCT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic phase</td>
<td>Poor response to &gt;2 TKIs&lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>Advanced phase&lt;sup&gt;1&lt;/sup&gt;</td>
<td>Patients with appearance of high-risk ACAs as a sign of progression, presence of <em>T315I</em> mutation&lt;sup&gt;1,2&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Resistance to a second-generation TKI in first-line or second-line&lt;sup&gt;2&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Failure with ponatinib&lt;sup&gt;1,2&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Accelerated phase at diagnosis&lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>Blast phase</td>
<td>ASCT as soon as possible: patients returning to a second CP (CP2) before ASCT have improved transplantation outcomes&lt;sup&gt;1,3&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

ACA, additional chromosomal aberrations; ASCT, allogeneic stem cell transplantation; CP, chronic phase; TKI, tyrosine kinase inhibitor.

Advances in ASCT

- Advances within ASCT are evident and include an increase in the number of registered volunteer donors, the creation of large cord blood banks, and advances in immunosuppression and supportive care.¹
- However, since the advent of TKIs, the number of ASCTs registered with CIBMTR has decreased.² In 2020, less than 200 of over 10,000 allotransplants reported to the CIBMTR were for CML³

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¹Data incomplete.
ALL, acute lymphoblastic leukemia; AML, acute myelogenous leukemia; AA, aplastic anemia; ASCT, allogeneic stem cell transplantation; CIBMTR, Center for International Blood and Marrow Transplant Research; CLL, chronic lymphocytic leukemia; CML, chronic myeloid leukemia; LYM, lymphoma; MM, multiple myeloma; TKI, tyrosine kinase inhibitor.


## Considerations for ASCT by EBMT score

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Score points</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age of patient (years)</strong></td>
<td></td>
</tr>
<tr>
<td>&lt;20</td>
<td>0</td>
</tr>
<tr>
<td>20-40</td>
<td>1</td>
</tr>
<tr>
<td>&gt;40</td>
<td>2</td>
</tr>
<tr>
<td><strong>Disease stage</strong></td>
<td></td>
</tr>
<tr>
<td>Early</td>
<td>0</td>
</tr>
<tr>
<td>Intermediate</td>
<td>1</td>
</tr>
<tr>
<td>Late</td>
<td>2</td>
</tr>
<tr>
<td><strong>Time interval from diagnosis to transplant (months)</strong></td>
<td></td>
</tr>
<tr>
<td>&lt;12</td>
<td>0</td>
</tr>
<tr>
<td>&gt;12</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Score points</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Donor type</strong></td>
<td></td>
</tr>
<tr>
<td>HLA-identical sibling donor</td>
<td>0</td>
</tr>
<tr>
<td>Unrelated donor, other</td>
<td>1</td>
</tr>
<tr>
<td><strong>Donor recipient sex combination</strong></td>
<td></td>
</tr>
<tr>
<td>All other</td>
<td>0</td>
</tr>
<tr>
<td>Female donor, male recipient</td>
<td>1</td>
</tr>
</tbody>
</table>

ASCT, allogeneic stem cell transplantation; EBMT, European Group for Marrow and Blood Transplantation; HLA, human leukocyte antigen

Considerations for ASCT by EBMT score

- The EBMT Risk Score calculates the chances and risks of ASCT for a patient pre-transplant by assessing five factors and generating a score from 0 as best and 7 as worst in an additive way\(^1\).

- A high EBMT score was found to be associated with higher NRM\(^1\).

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Stem cell sources in ASCT

- ASCT uses stem cells from a donor, either bone marrow, peripheral blood or cord blood, with these graft sources varying according to the demographics of the recipient.¹

Number of allogeneic HCTs in the US in recipients aged <18 years by graft source

Number of allogeneic HCTs in the US in recipients aged ≥18 years by graft source

ASCT, allogeneic stem cell transplantation; BM, bone marrow; CB, cord blood; HCT, hematopoietic cell transplant; PB, peripheral blood.

ASCT protocol


ASCT, allogeneic stem cell transplantation.

- Matched sibling, matched unrelated donor or haploidentical (half-matched) family member
- Mismatched unrelated donor

**Administration of stem cells** to patient pre-treated with a conditioning regimen

- Bone marrow
- Peripheral blood
- Cord blood cells

**Administration of post-transplant regimen**
Outcomes of ASCT: Efficacy

- Many factors can affect efficacy of treatment at all stages of the procedure (pre- to post-transplant)\(^1\)–\(^4\)

\[ \text{Pre-transplant} \quad \text{Transplant} \quad \text{Post-transplant} \]

\[ \text{factors affecting} \quad \text{Efficacy} \]

measured in terms of\(^1\)

- CR
- MR
- NRM

Survival (overall, disease-free, progression-free)

ASCT, allogeneic stem cell transplant; CR, complete remission; MR, molecular response; NRM, non-relapse mortality.

Outcomes of ASCT: Efficacy

- ASCT has been associated with improved long-term efficacy outcomes in early disease stages\(^1\),\(^2\)

**Survival after HCT for CML in the US, 2008-2018**

**Matched related donor**
- Hematologic CR (n=313)
- Chronic phase (n=367)
- Accelerated phase (n=93)
- Blast phase (n=56)

**Unrelated donor**
- Hematologic CR (n=476)
- Chronic phase (n=595)
- Accelerated phase (n=140)
- Blast phase (n=85)


# A summary of clinical trials assessing ASCT outcomes

<table>
<thead>
<tr>
<th>Study</th>
<th>Registry</th>
<th>Interval</th>
<th>N</th>
<th>Median age</th>
<th>Conditioning</th>
<th>Donor</th>
<th>1-year survival, %</th>
<th>5-year survival, %</th>
<th>10-year survival, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Millot et al.</td>
<td>SGFMTC</td>
<td>1982–1998</td>
<td>42</td>
<td>14</td>
<td>MA</td>
<td>REL</td>
<td>87</td>
<td>73</td>
<td>73</td>
</tr>
<tr>
<td>Cwynarski et al.</td>
<td>EBMT</td>
<td>1985–2001</td>
<td>156</td>
<td>14</td>
<td>NR</td>
<td>REL</td>
<td>78</td>
<td>72</td>
<td>70</td>
</tr>
<tr>
<td>Arora et al.</td>
<td>CIBMTR</td>
<td>1988–2003</td>
<td>3514</td>
<td>36</td>
<td>MA</td>
<td>REL</td>
<td>74</td>
<td>63</td>
<td>60</td>
</tr>
<tr>
<td>Arora et al.</td>
<td>CIBMTR</td>
<td>1988–2003</td>
<td>531</td>
<td>37</td>
<td>MA</td>
<td>UNR</td>
<td>70</td>
<td>55</td>
<td>50</td>
</tr>
<tr>
<td>Radich et al.</td>
<td>Seattle</td>
<td>1995–2000</td>
<td>131</td>
<td>43</td>
<td>MA</td>
<td>REL</td>
<td>91</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Gratwohl et al.</td>
<td>German Study III</td>
<td>1997–2004</td>
<td>151</td>
<td>38</td>
<td>MA</td>
<td>REL</td>
<td>90</td>
<td>78</td>
<td>76</td>
</tr>
<tr>
<td>Gratwohl et al.</td>
<td>German Study III</td>
<td>1997–2004</td>
<td>148</td>
<td>41</td>
<td>MA</td>
<td>UNR</td>
<td>97</td>
<td>76</td>
<td>76</td>
</tr>
<tr>
<td>Bacher et al.</td>
<td>German Registry</td>
<td>1998–2004</td>
<td>1084</td>
<td>40</td>
<td>MA 62%</td>
<td>REL</td>
<td>61%</td>
<td>67</td>
<td>64</td>
</tr>
<tr>
<td>Ohashi et al.</td>
<td>Japanese Registry</td>
<td>2000–2009</td>
<td>531</td>
<td>40</td>
<td>MA 89%</td>
<td>UNR</td>
<td>51%</td>
<td>87</td>
<td>85</td>
</tr>
<tr>
<td>Chaudury et al.</td>
<td>CIBMTR</td>
<td>2001–2010</td>
<td>224</td>
<td>24</td>
<td>MA</td>
<td>REL</td>
<td>90</td>
<td>83</td>
<td>NA</td>
</tr>
<tr>
<td>Chaudury et al.</td>
<td>CIBMTR</td>
<td>2001–2010</td>
<td>225</td>
<td>24</td>
<td>MA</td>
<td>UNR</td>
<td>80</td>
<td>68</td>
<td>NA</td>
</tr>
<tr>
<td>Lee et al.</td>
<td>Korean</td>
<td>2001–2012</td>
<td>47</td>
<td>32</td>
<td>MA 77%</td>
<td>UNR</td>
<td>43%</td>
<td>88</td>
<td>NA</td>
</tr>
<tr>
<td>Lee et al.</td>
<td>Korean</td>
<td>2001–2012</td>
<td>50</td>
<td>33</td>
<td>MA 48%</td>
<td>UNR</td>
<td>42%</td>
<td>90</td>
<td>NA</td>
</tr>
<tr>
<td>Koenenke et al.</td>
<td>EBMT</td>
<td>2002–2005</td>
<td>193</td>
<td>31</td>
<td>MA</td>
<td>REL</td>
<td>90</td>
<td>85</td>
<td>84</td>
</tr>
<tr>
<td>Saussele et al.</td>
<td>German Study IV</td>
<td>2003–2008</td>
<td>19</td>
<td>35</td>
<td>MA 79%</td>
<td>REL</td>
<td>53%</td>
<td>95</td>
<td>NA</td>
</tr>
<tr>
<td>Saussele et al.</td>
<td>German Study IV</td>
<td>2003–2008</td>
<td>37</td>
<td>38</td>
<td>MA 65%</td>
<td>UNR</td>
<td>70%</td>
<td>95</td>
<td>NA</td>
</tr>
</tbody>
</table>

CIBMTR, Center for International Blood and Marrow Transplantation; EBMT, European Group for Marrow and Blood Transplantation; MA, myeloablative; NR, not reported; REL, related donor; SGFMTC, Société Française de Greffe de Moelle et de Thérapie Cellulaire; UNR, unrelated donor.

Clinical trials assessing ASCT outcomes

- Given the low number of ASCT for CML performed during the last two decades, recent analyses of large series are lacking.
- The following two studies highlight recent data from the Swedish CML registry and the German CML Study IV from populations having undergone ASCT, and on the role of ASCT in the era of imatinib\textsuperscript{1,2}

ASCT, allogeneic stem cell transplantation; AP, accelerated phase; CML, chronic myeloid leukemia.

Lübking et al. (2019) analyzed the outcomes of patients with CML who underwent ASCT between 2002 and 2017

OS was dependent on the disease stage, with patients in CP1 achieving a 5-year OS of 96.3%. Patients in advanced disease stages had a 5-year OS of 36.9%

- Relapse 34.3% within 5 years
- Deaths 7.1%

5-year survival
- CP1 (n=56): 96.3%
- CP>1 (n=48): 70.1%
- AP/BC (n=14): 36.9%

- Relapse 20% within 6 months
- Deaths 28.6%

- Relapse 45.7% within 5 years
- Deaths 16.7%

AP, accelerated phase; ASCT, allogeneic stem cell transplantation; BC, blast crisis; CML, chronic myeloid leukemia; CP, chronic phase; OS, overall survival.

## Population-based data from the Swedish cancer registry

<table>
<thead>
<tr>
<th>Phase at time of ASCT</th>
<th>CP1</th>
<th>CP&gt;1</th>
<th>AP/BC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>56 (47.5%)</td>
<td>48 (40.7%)</td>
<td>14 (11.9%)</td>
</tr>
<tr>
<td>Median age</td>
<td>43</td>
<td>45</td>
<td>54</td>
</tr>
<tr>
<td>Reason for ASCT</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AP/BC at diagnosis</td>
<td>0</td>
<td>30 (62.5%)</td>
<td>3 (21.4%)</td>
</tr>
<tr>
<td>Progression to AP/BC</td>
<td>0</td>
<td>18 (37.5)</td>
<td>9 (64.3%)</td>
</tr>
<tr>
<td>T315I mutation</td>
<td>7</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>TKI resistance</td>
<td>35 (62.5%)</td>
<td>0</td>
<td>2 (14.3%)</td>
</tr>
<tr>
<td>TKI intolerance</td>
<td>5 (8.9%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Other reason</td>
<td>9 (16.1%)</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

AP, accelerated phase; ASCT, allogeneic stem cell transplantation; BC, blast crisis; CP, chronic phase; TKI, tyrosine kinase inhibitor.

Population-based data from the Swedish cancer registry


AP, accelerated phase; ASCT, allogeneic stem cell transplantation; BC, blast crisis; CP, chronic phase; SCT, stem cell transplant; TKI, tyrosine kinase inhibitor.

AP, accelerated phase; ASCT, allogeneic stem cell transplantation; BC, blast crisis; CP, chronic phase; SCT, stem cell transplant; TKI, tyrosine kinase inhibitor.

The role of ASCT as second-line therapy after imatinib failure

- Saussele et al. (2010) evaluated the role of ASCT as a second-line therapy after imatinib failure (according to ELN guidelines)\(^1\)
- Three-year survival probability post-ASCT in Groups 1, 2 and 3 were 91%, 94.1%, and 59%, respectively, and cumulative TRM was 8%\(^1\)

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AraC, cytarabine; ASCT, allogeneic stem cell transplantation; BC, blast crisis; CP, chronic phase; ELN, European LeukeimiaNet; IFN, interferon; TRM, transplantation-related mortality.

The role of ASCT as second-line therapy after imatinib failure

- Matched pair analyses showed that at 3 years, survival after diagnosis of 53 patients who underwent ASCT was not different from that of 106 matched patients who did not (91.9% [CI: 82.9–97.8%] vs 95.9% [CI: 91.1–98.9%], respectively).
- Complete molecular remission, however, was achieved in 52 transplanted patients (88%), highlighting the curative potential of this treatment approach\(^1\).
- Consistent with previous studies, the best long-term survival results in blast crisis are achieved by ASCT\(^1\).

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ASCT, allogeneic stem cell transplantation; CP, chronic phase; SCT, stem cell transplant.

Outcomes of ASCT: Safety

- The most commonly reported risks of ASCT include GvHD, relapse, and infection\(^1\)

- Complications as a result of ASCT may present at any stage post-transplant. These are classed as delayed (3 months to 2 years), late (2 years to 10 years) and very late (>10 years)\(^2\)

- Many of the long-term side effects can be attributed to the impact of GvHD, but may also depend on the intensity of the conditioning regimen and immunosuppression\(^2\)
Covariates affecting ASCT outcome: Safety & efficacy

Pre-transplant
- EBMT risk score\(^1\)\(^\text{-}^3\)
- Comorbidities (such as organ dysfunction)\(^2\)\(^,\)\(^4\)

Transplant
- Stem cell source\(^2\)
- Pre-transplant conditioning regimen (MAC vs. RIC)\(^3\)

Post-transplant
- GvHD\(^3\)
- Relapse\(^3\)
- Transplant toxicity/infection\(^3\)
- Post-transplant regime (cyclophosphamide)\(^3\)
- Optimisation of GVL effect\(^3\)

ASCT, allogeneic stem cell transplant; EBMT, European Society for Blood and Marrow Transplantation; GvHD, graft versus host disease; GVL, graft versus leukemia; MAC, myeloablative conditioning; RIC, reduced intensity conditioning.

Complications after ASCT

- According to the NIH, GvHD can be classified according to its time of presentation into acute or chronic using a 100 day cutoff period post-transplant. These categories can then be further subdivided according to clinical manifestations.\(^1,2\)

- **Acute classic GvHD**: presents within 100 days of the transplantation procedure with classical clinical features of acute GvHD.\(^1\)

- **Late acute GvHD**: presents after 100 days of the transplantation procedure with classical clinical features of acute GvHD. This can be further subdivided into **persistent** if it is a continuation of the classic acute GvHD, **Recurrent** if it has been resolved then recurs after the 100 day mark, **De novo** if the initial onset occurs after the 100 day mark with no prior acute GvHD.\(^1,2\)

- **Chronic GvHD**: presents after 100 days of the transplantation procedure with classical features of chronic GvHD. Furthermore, the diagnosis requires (a) at least one diagnostic manifestation or (b) one distinctive manifestation which is confirmed by biopsy or testing of the organ. These diagnostic manifestations can be found on the skin, in the mouth, genitalia, GI tract and lungs.\(^2\)

- Incidence of acute GvHD can be up to 50% in patients receiving the transplant from a matched sibling or even higher in unmatched donors.\(^1\) Incidence of chronic GvHD can range between 6 and 80% and overall mortality can approach 15%\(^1,3\)

- This incidence is variable in function of a number of factors including degree of HLA mismatch, patient age, and intensity of the conditioning regimen.\(^4\)
## Complications after ASCT

<table>
<thead>
<tr>
<th>Organ/site</th>
<th>Acute GvHD</th>
<th>Chronic GvHD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Skin</strong></td>
<td>Erythema</td>
<td>Poikiloderma</td>
</tr>
<tr>
<td></td>
<td>Maculopapular rash</td>
<td>Lichen planus-like features</td>
</tr>
<tr>
<td></td>
<td>Pruritus</td>
<td>Sclerotic features</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Morphea-like features</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lichen sclerous-like features</td>
</tr>
<tr>
<td><strong>Mouth</strong></td>
<td>Gingivitis</td>
<td>Lichen-type features</td>
</tr>
<tr>
<td></td>
<td>Mucositis</td>
<td>Hyperkeratotic plaques</td>
</tr>
<tr>
<td></td>
<td>Erythema</td>
<td>Restriction of mouth opening from sclerosis</td>
</tr>
<tr>
<td></td>
<td>Pain</td>
<td></td>
</tr>
<tr>
<td><strong>Genitalia</strong></td>
<td></td>
<td>Lichen planus-like features</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vaginal scarring or stenosis</td>
</tr>
<tr>
<td><strong>GI tract</strong></td>
<td>Anorexia</td>
<td>Esophageal web</td>
</tr>
<tr>
<td></td>
<td>Nausea</td>
<td>Strictures or stenosis in the upper to mid third of the esophagus</td>
</tr>
<tr>
<td></td>
<td>Vomiting</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diarrhea</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Weight loss</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Failure to thrive (infants and children)</td>
<td></td>
</tr>
<tr>
<td><strong>Liver</strong></td>
<td>Total bilirubin, alkaline phosphatases $&gt;2 \times$ upper limit of normal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ALT or AST $&gt;2 \times$ upper limit of normal</td>
<td></td>
</tr>
<tr>
<td><strong>Kidney</strong></td>
<td>Chronic kidney disease</td>
<td></td>
</tr>
<tr>
<td><strong>Lung</strong></td>
<td>Bronchiolitis obliterans, bronchiolitis obliterans-organizing pneumonia, idiopathic pneumonia</td>
<td></td>
</tr>
<tr>
<td><strong>Muscle, joints</strong></td>
<td>Fascilitis</td>
<td>Joint stiffness or contractures secondary to sclerosis</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td>Infections due to abnormal immune reconstitution, higher incidence of diabetes and hypertension, secondary cancers, relapse</td>
<td></td>
</tr>
</tbody>
</table>

ALT, alanine aminotransferase; ASCT, allogeneic stem cell transplant; AST, aspartate aminotransferase; GI, gastrointestinal; GvHD, graft versus host disease.

Summary

- Whilst ASCT no longer represents the primary treatment option, it still remains a salvage therapy for some patients.
- The primary aims of ASCT are to improve survival rates in patients with CML.
- ASCT is a treatment option for patients who fail to respond to TKIs as stipulated by specific guidelines, have advanced disease or a low EBMT risk score.
- Efficacy of ASCT can be affected at all stages of the procedure by a variety of factors.
- Complications post-transplant are mainly down to manifestations of GvHD.
- Studies have shown ASCT to be an effective second-line strategy for prolonging OS, notably in patients with early disease who have failed first-line therapies.

ASCT, allogeneic stem cell transplantation; CML, chronic myeloid leukemia; EBMT, European group for blood and bone marrow transplantation; GvHD, graft-versus-host disease; OS, overall survival; TKI, tyrosine kinase inhibitor.
References (1/5)


References (3/5)


- Kumar, R. and Krause, D., 2021. Recent advances in understanding chronic myeloid leukemia: where do we stand?. Faculty Reviews, 10.


- NCCN, 2021. NCCN Guidelines for Patients Chronic Myeloid Leukemia. [online] Nccn.org. Available at: <https://www.nccn.org/patients/guidelines/content/PDF/cml-patient.pdf> [Accessed 28 April 2022].


References (5/5)

