Hematopoietic Cell Transplantation for Myelofibrosis

Policy

*Please see amendment for Pennsylvania Medicaid at the end of this CPB.

Aetna considers allogeneic (ablative and non-myeloablative) hematopoietic cell transplantation (HCT) medically necessary for individuals with myelofibrosis (MF) when any of the following criteria is met:

- The individual is dependent on transfusions of red blood cells; or
- The individual is dependent on transfusions of platelets or has frequent infarctions; or
- The individual has an absolute neutrophil count less than 1000/mm³; or
- The individual is resistant to conservative therapy; or
- The individual has intermediate or high risk MF

Aetna considers a repeat allogeneic (ablative or non-myeloablative) HCT medically necessary for individuals with myelofibrosis and primary graft failure or who have relapsed.

Aetna considers mutational profiling (use of genetic
biomarkers) for assessing prognosis following HCT for myelofibrosis experimental and investigational due to insufficient evidence in the peer-reviewed literature.

Aetna considers pre-HCT ruxolitinib for myelofibrosis experimental and investigational due to insufficient evidence in the peer-reviewed literature.

Aetna considers splenic irradiation before HCT for myelofibrosis experimental and investigational due to insufficient evidence in the peer-reviewed literature.

Aetna considers autologous hematopoietic cell transplantation experimental and investigational for myelofibrosis due to insufficient evidence in the peer-reviewed literature.


Background

Primary MF is considered a chronic myeloproliferative disorder and is characterized by variable degrees of cytopenia, cytosis, bone marrow fibrosis, a leukoerythroblastic blood picture, and
extramedullary hematopoiesis, which can result in hepatosplenomegaly (Cervantes et al, 2009). MF is a heterogeneous disease in that MF is an indolent disease in some patients, who may survive for decades, to an aggressive disease in others, with disabling symptoms, lowered quality of life and in some cases survival of less than a year (McLornan et al, 2012). MF can be either primary or secondary, and can develop in patients with polycythemia vera or essential thrombocythemia. The median age is in the seventh decade and approximately 70% of patients are positive for the Janus2 kinase mutation (Ballen, 2012).

There have been no available conventional drug therapies for MF which have been shown to prolong survival. Palliative agents include erythropoietin, androgens, immunomodulatory agents, interferons, cyto reductive therapies and non-pharmacologic approaches. The non-pharmacologic approaches include blood transfusion, splenic irradiation, and splenectomy. Allogeneic hematopoietic stem cell transplantation (SCT) is considered to be the only potentially curative therapy for MF (McLornan et al, 2012).

The American Society for Blood and Marrow Transplantation Guideline on the role of cytotoxic therapy with hematopoietic stem cell transplantation in the therapy of myelodysplastic syndrome states that “early SCT is recommended for patients with an International Prognostic Scoring System (IPSS) score of intermediate (INT) -2 (considered high risk) at diagnosis who have a suitable donor and meet the transplant center’s eligibility criteria, and for selected patients at low risk (IPSS score of INT-1) at diagnosis who have poor prognostic features not included in the IPSS (e.g. older age, refractory cytopenias) (Oliansky et al, 2009). The IPSS estimates survival from the time of diagnosis based on five risk factors: age > 65 years, hemoglobin < 100 g/l, leukocyte count > 25 x 10^9/l, circulating blasts ≥ 1%, and the presence of constitutional symptoms. Patients are then classified as low
risk (score = 0), intermediate risk-1 (score = 1), intermediate risk-2 (score = 2), and high risk (score ≥ 2) (McLornan et al, 2012).

Cervantes et al (2009) studied 1054 patients at 7 centers who were diagnosed with primary MF. The purpose of this retrospective study was to develop a highly discriminative prognostic system. Variables selected for prognostic assessment were those previously shown to be of prognostic value in primary MF along with variables considered to be either clinically meaningful or potential confounders. Analysis using Cox proportional hazards modeling revealed identified age greater than 65 years, presence of constitutional symptoms, hemoglobin level less than 10 g/dL, leukocyte count greater than 25 x 10⁹/L, and circulating blast cells 1% or greater as predictors of shortened survival. Overall median survival was 69 months (95% confidence interval: 61-71). Four risk groups with no overlap in their survival curves were identified, including 0 (low risk), 1 (intermediate risk-1), 2 (intermediate risk-2), or greater than or equal to 3 (high risk), with respective median survivals of 135, 95, 48, and 27 months (p < 0.001). Additionally, in 409 patients with assessable metaphases, cytogenetic abnormalities were associated with shorter survival, but their independent contribution to prognosis was restricted to patients in the intermediate-risk groups. JAK2V617F did not cluster with a specific risk group or affect survival.

Kroger and colleagues (2009) noted that from 2002 to 2007, a total of 103 patients with primary myelofibrosis or post-essential thrombocythemia and polycythemia vera myelofibrosis and a median age of 55 years (range of 32 to 68 years) were included in a prospective, multi-center phase-II clinical trial to examine efficacy of a busulfan (10 mg/kg)/fludarabine (180 mg/m²)-based reduced-intensity conditioning (RIC) regimen followed by allogeneic stem cell transplantation (ASCT) from related (n = 33) or unrelated donors (n = 70). All but 2 patients (2 %) showed leukocyte and
platelet engraftment after a median of 18 and 22 days, respectively. Acute graft-versus-host disease (aGVHD) grade 2 to 4 occurred in 27% and chronic GVHD (cGVHD) in 43% of the patients. Cumulative incidence of non-relapse mortality (NRM) at 1 year was 16% (95% confidence interval [CI]: 9% to 23%) and significantly lower for patients with a completely matched donor (12% versus 38%; p = 0.003). The cumulative incidence of relapse at 3 years was 22% (95% CI: 13% to 31%) and was influenced by Lille risk profile (low, 14%; intermediate, 22%; and high, 34%; p = 0.02). The estimated 5-year event-free survival (EFS) and overall survival (OS) was 51% and 67%, respectively. In a multi-variate analysis, age older than 55 years (hazard ratio [HR] = 2.70; p = 0.02) and human leukocyte antigen (HLA)-mismatched donor (HR = 3.04; p = 0.006) remained significant factors for survival.

Tefferi et al (2011a) noted that "current drug therapy in primary MF is neither curative or essential for survival. Similarly, it is not clear if the application of allogeneic SCT, with its attendant risk of death or chronic morbidity from graft-versus-host disease, has had a favorable or unfavorable net effect. Therefore, one must first determine whether a particular patient needs any form of therapy at all and, if so, carefully select the treatment strategy with the best chance of inducing disease control without compromising life expectancy." Tefferi et al (2011b) reported that the presence of fibrosis, JAK2/MPL mutation or +9/13q- cytogenic abnormality is supportive but not essential for diagnosis, and that diagnosis is based on bone marrow morphology. The authors state that observation alone is adequate for asymptomatic low/intermediate-1 risk disease; allogeneic SCT or experimental drug therapy is reasonable for symptomatic intermediate-1 risk disease.

Alchalby et al (2012) evaluated 150 homogeneously treated MF patients who underwent reduced-intensity allogeneic SCT and developed a risk score for overall survival. The authors' prognostic scoring system compared to the Lille scoring system and correlated significantly with overall survival.
but discriminated poorly between the intermediate and high-risk groups. The authors concluded that a simple model which includes age, JAK2 V617F-status, and constitutional symptoms can clearly separate distinct risk groups. The authors further noted that such a model can be used in addition to the Lille model to predict overall survival after reduced-intensity allogeneic SCT.

Scott et al (2012) conducted a study to evaluate a Dynamic International Prognostic Scoring System (DIPSS) risk categorization. They evaluated the DIPSS in 170 MF patients aged 12 to 78 years who received SCT from related (n = 86) or unrelated (n = 84) donors. The investigators determined that 21 patients had low-risk disease, 48 had intermediate-1, 50 had intermediate-2, and 51 had high-risk disease. Additionally, they reported five-year incidence of relapse, relapse-free survival, overall survival, and nonrelapse mortality for all patients were 10%, 57%, 57%, and 34%, respectively. They concluded that SCT was curative for a large proportion of patients with MF, and post-SCT success was dependent on pre-SCT DIPSS classification.

In a recent review of allogeneic stem cell transplantation for MF, McLornan et al (2012) concluded that transplant-eligible MF patients with intermediate-2 and high-risk disease should be considered for SCT. Additionally, patients with transfusion dependency or an unfavorable karyotype should also be considered for SCT. The authors suggested a myeloablative conditioned approach in those greater than 45 years of age, and acknowledged that some patients between 45 and 50 years of age with low HCT-CI scores may well also be suitable for a myeloablative conditioned SCT. They further suggested that a reduced-intensity conditioning regimen be considered for those over the age of 45 years and that patients older than 65 years should not be definitively excluded from potential SCT on age criteria alone, but rather that “a frank discussion
with the patient regarding the association of older age and, in
general, an adverse post-SCT outcome should occur in
addition to a detailed risk assessment”.

An UpToDate review on “Prognosis and treatment of primary
myelofibrosis” (Tefferi, 2013) provides the following
recommendations:

▪ For younger patients (i.e., age less than 45 years) at
intermediate-2 or high risk according to the DIPSS
[Dynamic International Prognostic Scoring System] Plus
scoring system, we suggest that the patient be
considered for hematopoietic cell transplantation (HCT)
shortly after diagnosis (Grade 2B). We prefer
conventional intensity conditioning for those less than
45 years of age and reduced-intensity conditioning for
those 45 to 65 years of age.

▪ For DIPSS Plus low-risk patients, who might live 10 to 15
years with supportive treatment alone, but might have a
transplant-related mortality of at least 8 percent, the
answer is not yet clear. Until further information is
available, we suggest against the use of HCT for this
group of patients (Grade 2C).

In an update on the diagnosis, risk-stratification, and
management of primary myelofibrosis (PMF), Tefferi et al
(2014a) stated that PMF is a myeloproliferative neoplasm
characterized by stem cell-derived clonal myeloproliferation,
abnormal cytokine expression, bone marrow fibrosis, anemia,
splenomegaly, extra-medullary hematopoiesis (EMH),
constitutional symptoms, cachexia, leukemic progression, and
shortened survival. Diagnosis is based on bone marrow
morphology. The presence of JAK2, CALR, or MPL mutation
is supportive but not essential for diagnosis; approximately 90
% of patients carry 1 of these mutations and 10 % are "triple-
negative". None of these mutations is specific to PMF and is
also seen in essential thrombocytemia (ET). Pre-fibrotic PMF
mimics ET in its presentation and the distinction, enabled by careful bone marrow morphological examination, is prognostically relevant. Differential diagnosis also includes chronic myeloid leukemia, myelodysplastic syndromes, chronic myelomonocytic leukemia, and acute myeloid leukemia. The Dynamic International Prognostic Scoring System-plus (DIPSS-plus) uses 8 predictors of inferior survival: age greater than 65 years, hemoglobin less than 10 g/dL, leukocytes greater than \(25 \times 10^9/L\), circulating blasts greater than or equal to 1%, constitutional symptoms, red cell transfusion dependency, platelet count less than \(100 \times 10^9/L\), and unfavorable karyotype (i.e., complex karyotype or sole or 2 abnormalities that include +8, -7/7q-, i(17q), inv(3), -5/5q-, 12p-, or 11q23 rearrangement). The presence of 0, 1, "2 or 3", and greater than or equal to 4 adverse factors defines low, intermediate-1, intermediate-2, and high-risk disease with median survivals of approximately 15.4, 6.5, 2.9, and 1.3 years, respectively. High risk disease is also defined by CALR(-)/ASXL1(+) mutational status. Observation alone is adequate for asymptomatic low/intermediate-1 risk disease, especially with CALR(+) /ASXL1(-) mutational status. Stem cell transplant is considered for DIPSS-plus high-risk disease or any risk disease with CALR(-)/ASXL1(+) mutational status. Investigational drug therapy is reasonable for symptomatic intermediate-1 or intermediate-2 risk disease. Splenectomy is considered for drug-refractory splenomegaly. Involved field radiotherapy is most useful for post-splenectomy hepatoamegaly, non-hepatosplenic EMH, PMF-associated pulmonary hypertension, and extremity bone pain.

About 5 to 18% of patients with myelofibrosis with myeloid metaplasia (MMM) who undergo allogeneic myeloablative transplantation will relapse after 3 years (Tanvetyanon and Stiff, 2004). Relapse carries a high mortality: 50% of the patients will die within 1 year. Increased age, impaired performance status, and reduced organ reserve often hamper further attempt at myeloablative chemotherapy.
Klyuchnikov et al (2012) reported the results of a second hematopoietic stem cell transplantation (HSCT) as salvage therapy in myelofibrosis patients who have relapsed or experienced graft rejection. A total of 30 myelofibrosis patients (21 males, 9 females) with relapse (n = 27) or graft-rejection (n = 3) after dose-reduced allografting underwent a salvage strategy including donor lymphocyte infusions (DLIs) and/or second allogeneic hematopoietic stem cell transplantation (HSCT); 26 patients received a median number of 3 (range of 1 to 5) DLIs in a dose-escalated mode starting with a median dose of $1.2 \times 10^6$ (range of $0.003$ to $8 \times 10^6$) up to median dose of $40 \times 10^6$ T-cells/kg (range of 10 to $130 \times 10^6$); 10/26 patients (39%) achieved complete response (CR) to DLIs. Acute (grade II to IV) and chronic graft-versus-host (GVHD) disease occurred in 12% and 36% cases; 13 non-responders to DLI and 4 patients who did not receive DLI due to graft-rejection or acute transformation of the blast phase underwent a second allogeneic HSCT from alternative (n = 15) or the same (n = 2) donor. One patient (6%) experienced primary graft-failure and died. Acute (grade II to IV) and chronic GVHD were observed in 47% and 46% of patients. Overall responses after second HSCT were seen in 12/15 patients (80%: CR: n = 9, partial response: n = 3). The 1-year cumulative incidence of non-relapse mortality for recipients of a second allograft was 6%, and the cumulative incidence of relapse was 24%. After a median follow-up of 27 months, the 2-year overall survival and progression-free survival for all 30 patients was 70% and 67%, respectively.

The investigators concluded that their 2-step strategy, including DLI and second HSCT for non-responding or ineligible patients, is an effective and well-tolerated salvage approach for patients relapsing after reduced-intensity allograft after myelofibrosis.

Mutational Profiling (Use of Genetic Biomarkers) for Prognosis Following Hematopoietic Cell Transplantation
Kroger and colleagues (2017) stated that molecular genetics may influence outcome for patients with MF. These researchers examined the impact of molecular genetics on outcome following allogeneic stem cell transplantation (ASCT). They screened 169 patients with PMF (n = 110), post-essential thrombocytopenia/polycythemia vera MF (n = 46), and MFs in transformation (n = 13) for mutations in 16 frequently mutated genes. The most frequent mutation was JAK2V617F (n = 101), followed by ASXL1 (n = 49), calreticulin (n = 34), SRSF2 (n = 16), TET2 (n = 10), U2AF1 (n = 11), EZH2 (n = 7), MPL (n = 6), IDH2 (n = 5), IDH1 (n = 4), and CBL (n = 1).

The cumulative incidence of non-relapse mortality (NRM) at 1 year was 21 % and of relapse at 5 years 25 %. The 5-year progression-free survival (PFS) and overall survival (OS) were 48 % and 56 %, respectively. In a multivariate analysis, CALR mutation was an independent factor for lower NRM (hazard ratio [HR], 0.415; p = 0.05), improved PFS (HR, 0.393; p = 0.01), and OS (HR, 0.448; p = 0.03). ASXL1 and IDH2 mutations were independent risk factors for lower PFS (HR, 1.53 [p = 0.008], and HR, 5.451 [p = 0.002], respectively), whereas no impact was observed for "triple negative" patients. The authors concluded that molecular genetics, especially CALR, IDH2, and ASXL1 mutations, may thus be useful to predict outcome independently from known clinical risk factors following ASCT for myelofibrosis.

Salit and Deeg (2018) noted that the prognosis of myeloproliferative neoplasms, including PMF, polycythemia vera, and essential thrombocytopenia varies considerably, between these disorders as well as within each diagnosis. Molecular studies have identified "driver mutations" in JAK2, MPL1, and CALR and additional somatic DNA mutations, including ASXL1, EZH2, IDH1/2, and SRSF2, that affect prognosis differentially. Patients with mutations in CALR (type1) had a better outlook than patients with mutations in JAK2 or MPL, whereas patients without any of the driver mutations (triple-negative) had the shortest life expectancy. Mutations in ASXL1, EZH2, and SRSF2 may be associated
with shortened survival, and IDH mutations carried a higher risk of leukemic transformation. The combination and number of mutations were more important than a given single mutation. Mutations also appeared to impact the outcome of HCT, currently the only treatment with curative potential. Based on available data, the best post-HCT outcome was observed with CALR mutations. Triple negativity had a negative impact. The data on JAK2 are controversial.

Mutations in ASXL1 or IDH1/2 reduced the probability of PFS following HCT, although the impact of ASXL1 differed between patients with PMF and secondary MF. Although it is not clear to what extent HCT can overcome the risks associated with a given mutational pattern, at present, early HCT should be considered in triple-negative patients and patients with PMF who harbored mutations in ASXL1. Mutations in EZH2, SRSF2, or IDH, particularly if combined with other mutations, should also lead to consideration of HCT. The authors concluded that further studies are needed to validate the present observations and determine the impact of additional mutations that have been identified.

Tamari and associates (2019) stated that mutational profiling has demonstrated utility in predicting the likelihood of disease progression in patients with MF. However, there are limited data regarding the prognostic utility of genetic profiling in MF patients undergoing ASCT. These researchers carried out high-throughput sequencing of 585 genes on pre-transplant samples from 101 patients with MF who underwent ASCT and evaluated the association of mutations and clinical variables with transplantation outcomes; OS at 5 years post-transplantation was 52%, and relapse-free survival (RFS) was 51.1% for this cohort; NRM accounted for most deaths. Patient's age, donor's age, donor type, and DIPSS score at diagnosis did not predict for outcomes. Mutations known to be associated with increased risk of disease progression, such as ASXL1, SRSF2, IDH1/2, EZH2, and TP53, did not impact OS or RFS. The presence of U2AF1 (p = 0.007) or DNMT3A (p = 0.034) mutations was associated with worse OS. A Mutation-
Enhanced International Prognostic Scoring System 70 score was available for 80 patients (79%), and there were no differences in outcomes between patients with high-risk scores and those with intermediate- and low-risk scores. Collectively, these data identified mutational predictors of outcome in MF patients undergoing ASCT. The authors concluded that these genetic biomarkers in conjunction with clinical variables may have important utility in guiding transplantation decision-making.

**Pre-Hematopoietic Cell Transplant Ruxolitinib**

Salit and associates (2020) stated that ruxolitinib (Rux), a Jak1/2 inhibitor, results in reduced spleen size and improvement in constitutional symptoms in the majority of patients with MF. Thus, Rux, when given prior to HCT in patients with MF was hypothesized to improve engraftment, decrease incidence and severity of GVHD, and lower NRM. In a phase-II clinical trial, these researchers examined the effects of pre-HCT Rux on post-HCT outcomes in patients with MF. The primary end-point was 2-year OS. To-date, a total of 28 patients (median age of 56 years) have been transplanted. The median time on Rux pre-HCT was 7 months; 23 patients received myeloablative and 5 reduced intensity conditioning (RIC). Donors included 14 HLA-matched siblings, 11 matched unrelated, 1 allele mismatched unrelated, and 3 umbilical cord blood. There have been no episodes of cytokine release syndrome (CRS) and all patients achieved sustained engraftment; 2 patients died from NRM and 2 patients relapsed. With a median follow-up of 13 months, OS was 93% (95% confidence interval [CI]: 0.73 to 0.98) at 1 year and 86% (95% CI: 0.61 to 0.96) at 2 years post-HCT. The authors concluded that the findings of this study demonstrated that pre-HCT Rux was well-tolerated and suggested that pre-HCT Rux may improve post-HCT outcome.
In a 2-stage Simon phase-II clinical trial, Gupta and colleagues (2019) examined the feasibility of Rux therapy followed by a RIC regimen for patients with MF undergoing transplantation. The objectives were to decrease the incidence of graft failure (GF) and NRM compared with data from the previous Myeloproliferative Disorders Research Consortium 101 Study. The plan was to enroll 11 patients each in related donor (RD) and unrelated donor (URD) arms, with trial termination if greater than or equal to 3 failures (GF or death by day +100 post-transplant) occurred in the RD arm or greater than or equal to 6 failures occurred in the URD. A total of 21 patients were enrolled, including 7 in the RD-arm and 14 in the URD-arm. The RD-arm did not meet the pre-determined criteria for proceeding to stage II. Although the URD-arm met the criteria for stage II, the study was terminated owing to poor accrual and a significant number of failures. In all 19 transplant recipients, Rux was tapered successfully without significant side effects, and 9 patients (47 %) had a significant decrease in symptom burden. The cumulative incidences of GF, NRM, acute GVHD, and chronic GVHD at 24 months were 16 %, 28 %, 64 %, and 76 %, respectively. On an intention-to-treat (ITT) basis, the 2-year OS was 61 % for the RD-arm and 70 % for the URD-arm. The authors concluded that Rux can be integrated as pre-transplantation treatment for patients with MF, and a tapering strategy before transplantation was safe, allowing patients to commence conditioning therapy with a reduced symptom burden. However, GF and NRM remain significant.

**Splenic Irradiation Before Allogeneic Stem Cell Transplantation**

Helbig and colleagues (2019) noted that splenectomy before ASCT for patients with MF remains a matter of debate, and conflicting results have been reported to-date. The procedure appeared to fasten post-transplant hematological recovery, but it did not have an impact on survival. The role of pre-transplant splenic irradiation (SI) is much more difficult to
evaluate. In this study, a total of 44 patients (25 males and 19 females) with MF at median age of 49 years at diagnosis (range of 14 to 67) underwent ASCT. The post-transplant outcome was compared between irradiated and non-irradiated patients; 11 patients received irradiation before transplantation. Median dose of radiation was 1,000 cGy (range of 600 to 2,400). There was no difference in median time to engraftment between patients with and without previous radiotherapy. Acute and chronic GVHD occurred in 47% and 36% of patients, respectively. There was no difference in GVHD incidence between groups; 8 patients relapsed/progressed in irradiated group versus 17 in the non-irradiated group (70% versus 51%; p = 0.3). Transformation to acute myeloid leukemia (AML) was observed in 3 patients: 2 in irradiated and 1 in non-irradiated group. A total of 22 patients died with no statistical difference in death rate between irradiated and non-irradiated subjects. The probability of OS following ASCT for the entire cohort at 2 years was 54% (72% for irradiated and 48% for non-irradiated patients; p = 0.25). The authors concluded that splenic irradiation prior to ASCT for myelofibrosis had no beneficial effect on post-transplant outcome and should not be routinely recommended.

Allogeneic Hematopoietic Stem Cell Transplantation with Fludarabine, Busulfan, and Thiotepa Conditioning

Shouval and colleagues (2020) stated that ASCT is a curative therapy for myelofibrosis; however, the optimal conditioning regimen has not been well-defined. These researchers retrospectively compared transplantation outcomes in patients with myelofibrosis (n = 67) conditioned with myeloablative (MAC, 36%) and RIC (46%) regimens, and more recently with the combination of thiotepa, busulfan, and fludarabine (TBF, 18%). Patients were transplanted from HLA-matched sibling (n = 26) or unrelated donors (n = 41) between the years 2003 and 2018. The median follow-up was 2.9 years for all patients but shorter in the TBF group (1.1 years). The
probability of 3-year PFS was 43%. At 1 year, the rate of PFS was 80%, 54%, and 45% with TBF, MAC, and RIC, respectively (p = 0.031). In a multi-variable model, there was a greater risk for death with MAC (HR 12.26, p = 0.026) and lower PFS with both MAC (HR 7.78, p = 0.017) and RIC (HR 5.43, p = 0.027) compared with TBF. Relapse was higher with RIC (HR 8.20, p = 0.043) while NRM was increased with MAC (HR 9.63 p = 0.049). These investigators stated that these findings indicated that TBF is a promising preparative regimen in myelofibrosis patients transplanted from matched sibling or unrelated donors, and should be further examined.

### CPT Codes / HCPCS Codes / ICD-10 Codes

*Information in the [brackets] below has been added for clarification purposes. Codes requiring a 7th character are represented by "+":*

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<thead>
<tr>
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<tr>
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<td><strong>CPT codes covered if selection criteria are met:</strong></td>
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<tr>
<td>38204</td>
<td>Bone marrow or stem cell services/procedures-allogenic</td>
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**CPT codes not covered for indications listed in the CPB:**

*Mutational profiling (use of genetic biomarkers) for prognosis following hematopoietic cell transplantation (HCT) for myelofibrosis - no specific code:*

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<tr>
<td>38232</td>
<td>Bone marrow harvesting for transplantation; autologous</td>
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<tr>
<td>38241</td>
<td>Hematopoietic progenitor cell (HPC); autologous transplantation</td>
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</table>
The above policy is based on the following references:


31. Tefferi A. Primary myelofibrosis: 2012 update on
diagnosis, risk stratification, and management. Am J
32. Tefferi A. Primary myelofibrosis: 2014 update on
diagnosis, risk-stratification, and management. Am J
Hematol. 2014a;89(9):915-925.
33. Tefferi A. Prognosis and treatment of primary
myelofibrosis. UpToDate [serial online]. Waltham, MA:
UpToDate; reviewed September 2013; August 2014b.
2011a;117:3494-3504.
AETNA BETTER HEALTH® OF PENNSYLVANIA

Amendment to
Aetna Clinical Policy Bulletin Number: 0838 Hematopoietic
Cell Transplantation for Myelofibrosis

There are no amendments for Medicaid.

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