Extracorporeal Immunoadsorption (Prosorba Column)

Number: 0355

Policy

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

Aetna considers extracorporeal immunoadsorption (ECI) medically necessary for any of the following indications:

1. Hemolytic uremic syndrome, with clinical evidence of serious bleeding with platelet count below 50,000 or the potential for serious bleeding with platelet count below 20,000; or

2. Idiopathic thrombocytopenic purpura, with clinical evidence of serious bleeding with platelet count below 50,000 or the potential for serious bleeding with platelet count below 20,000; or

3. Last resort treatment of life-threatening systemic lupus erythematosus when conventional therapy has failed to prevent clinical deterioration; or

4. Moderate-to-severe rheumatoid arthritis, for reduction of signs and symptoms in members who have failed other treatments (e.g., non-steroidal anti-inflammatory drugs, methotrexate); or

Policy History

Last Review
05/28/2019
Effective: 11/08/1999
Next Review: 03/27/2020

Definitions

Additional Information

Clinical Policy Bulletin
Notes
5. Myasthenic crisis when conventional therapy (e.g., intravenous immunoglobulin or plasmas exchange) has failed; or
6. Pemphigus vulgaris that is resistant to standard therapy, including dapsone, corticosteroids, and immunosuppressants (e.g., azathioprine or cyclosporine).

Aetna considers ECI experimental and investigational for all other indications because its effectiveness for indications other than the ones listed above has not been established (not an all-inclusive list):

- Allergic asthma
- Atopic dermatitis
- Anti-phospholipid syndrome
- Dermatomyositis
- Elevated lipoprotein(a)
- Epidermolysis bullosa acquisita
- Heart failure
- Hepatitis B.

See
  CPB 0206 - Parenteral Immunoglobulins,
also (../200_299/0206.html)
CPB 0285 - Plasmapheresis/Plasma Exchange/Therapeutic Apheresis (../200_299/0285.html)
, CPB 0315 - Enbrel (Etanercept) (0315.html)
, CPB 0341 - Infliximab (0341.html),
and CPB 0595 - Anakinra (Kineret) (../500_599/0595.html).

**Background**

Extra-corporeal immunoadsorption (ECI), also referred to as protein immunoadsorption therapy or by the trade name Prosorba Column (Cypress Bioscience), consists of a highly purified protein A (isolated from staphylococcus aureus) that is
bonded to a silica matrix. Plasma is collected from the patient in a pheresis procedure and then passed over the column. Circulating immune complexes and IgG bind to the protein A and are thus selectively removed from plasma. The plasma can then be returned to the patient, thus eliminating the need for a plasma exchange.

Idiopathic thrombocytopenic purpura (ITP) is characterized by rapid platelet destruction and typically appears in young women and also in male patients who are sero-positive for HIV infection. It is usually a relatively benign disorder in its chronic form and there is no indication to treat when the platelet count is above 50,000. In cases involving more serious bleeding or with platelet counts less than 20,000, ECI has successfully reversed the immune thrombocytopenia by removal and modulation of platelet-specific IgG and circulating immune complexes.

Extra-corporeal immunoadsorption has also been used in the treatment of hemolytic uremic syndrome (HUS), which is characterized by thrombocytopenia, microangiopathic hemolytic anemia, and progressive renal failure. It may occur in patients with malignancies treated with mitomycin C and cisplatin. This syndrome is known to be associated with circulating immune complexes that may play a role in its pathogenesis. A number of patients treated with protein A columns have achieved a definite increase in platelet count, decrease of hemolysis, and stabilization of renal function.

More recently, the Food and Drug Administration (FDA) has approved extra-corporeal immunoadsorption for the treatment of rheumatoid arthritis (RA). It is indicated for use in therapeutic reduction of signs and symptoms of moderate-to-severe RA in adult patients with long-standing disease who have failed or are intolerant of disease-modifying anti-rheumatic drugs. The FDA stressed that the machine was for use in a small proportion of patients, those with moderate-to-
severe symptoms who have failed other treatments; it is not first-line therapy. The machine offers a 30% chance of improving the swelling and pain that cripples a patient's joints.

For HUS and ITP, treatment is typically given 6 times over the course of 2 to 3 weeks. For RA, patients are typically treated once-weekly for 12 weeks.

Ruocco et al (2005) stated that pemphigus vulgaris (PV) is a rare autoimmune bullous dermatosis with a high mortality rate if untreated. The disease results from autoimmunity to normal components of keratinocyte cell membrane (desmogleins 3 and 1) belonging to the cadherin super-gene family. Standard treatment for PV entails combination of glucocorticoids (high dosage) and immunosuppressants. In patients with severe, life-threatening, or refractory PV, stronger therapies should be considered (e.g., "pulse-therapy" with discontinuous intravenous infusion of mega doses of immunosuppressants over a short-time, plasmapheresis, and ECI of pathogenic autoantibodies using the extracellular domain of the PV main antigen (desmoglein 3) produced by baculovirus or, more recently, a tryptophan-linked polyvinyl alcohol adsorber.

Braun et al (2000) noted that reduction of pathological autoantibodies (Abs) as well as circulating immune complexes (IC) can be beneficial in the treatment of autoimmune disease. Plasmapheresis has been shown to reduce Abs in systemic lupus erythematosus (SLE), but its effect on health outcome was not better compared with conventional immunosuppression in the past. These investigators evaluated immunoabsorption (IAS) as rescue therapy in patients suffering from SLE. A total of 8 patients with severe, therapy-resistant SLE underwent immunoabsorption onto protein A sepharose without concomitant immunosuppressants. Remission of the disease was achieved in 7 patients. Therapy had to be stopped in 1 patient because of side-effects. The best results were obtained when IAS was performed daily, without supplementary intravenous
immunoglobulin therapy. Oral cyclophosphamide for 3 to 6 months during follow-up was used to suppress relapse. Circulating IC and Abs were effectively eliminated regardless of their IgG subclass. The authors concluded that IAS onto protein A might be used as an extra-corporeal treatment option in SLE when other therapies are ineffective.

Stummvoll et al (2009) stated that IAS with various methods is used as a rescue therapy in severely ill SLE patients who are refractory to conventional therapeutic procedures. The method aims at the rapid and extensive removal of pathogenic IC and Abs. Long-term observational studies suggested efficacy and have not seen an increase in the risk of infections (as were seen in other extracorporeal procedures). However, randomized controlled trials (RCT) are lacking. Recently, biologicals aiming at tumor necrosis factor-blockade or B-cell depletion have been used to treat severe SLE. They are easier to apply since they do not necessitate additional hardware or specially trained staff. While there is emerging evidence for efficacy from uncontrolled observations, no RCT could so far demonstrate benefit in SLE. Under these circumstances, IAS still has a role in treating severe SLE, when other therapies are ineffective or are contraindicated (as in pregnancy).

Biesenbach et al (2009) stated that pathogenic Abs are a hallmark of SLE and their rapid removal is beneficial in active SLE. Immunoadsorption is effective in removing serum levels of all classes of immunoglobulin (Ig), IC and anti-dsDNA Abs and appears superior to plasmapheresis with respect to side effects. Immunoadsorption can be performed with different columns, which use different ligands to bind their target. In particular, high affinity columns are in the focus of interest. Their ligands are either sheep IgG directed against human Ig (Ig-column, Ig-Therasorb((R))), or staphylococcal Protein A (ProtA-column, Immunosorba((R))), or the synthetic peptide Gam146 (GAM-column, Globaffin((R))). In the authors’ experience, Ig-columns have been effective in treating
active renal SLE. However, no analysis has so far been published on which column type should be preferred in treating SLE patients. Among the authors’ SLE patients maintained on prolonged IAS therapy, those with stable renal SLE and low-to-moderate disease activity who were successfully treated by using Ig-columns were identified. Six of these patients were switched to ProtA-columns, keeping the rest of the protocol and the medication constant. In addition, 2 patients were switched from Ig- to GAM-columns. All types of columns significantly lowered the serum levels of IgG, IgM, and anti-dsDNA Abs. Disease activity was constantly low before and after the switch, as were parameters of renal function. In addition, patients with highly active disease were effectively treated when ProtA- (n = 6) or GAM-columns (n = 1) were used as first-line extracorporeal treatment. The authors concluded that these findings demonstrated that all columns are adequately effective in controlling key parameters of SLE. Thus, it is not the type of the ligand, but only the outcome, i.e., the successful removal of Ig, IC, and auto-Abs that is needed for controlling SLE activity.

Gürcan and Ahmed (2011) noted that long-term remission in patients with epidermolysis bullosa acquisita (EBA) is difficult to achieve. Patients who are resistant or develop side effects to conventional immuno-suppressive therapy (CIST) have been treated with several other agents. These investigators focused on the clinical outcome in patients treated with a single drug or combination, and determined if long-term remission can be induced. Data on 71 patients were analyzed. There are no controlled trials. The regimens used included colchicine, cyclosporine, daclizumab, dapsone, intravenous immunoglobulin, mesalazine, mycophenolic acid, rituximab, extra-corporeal photochemotherapy, and plasmapheresis. The use of CIST, especially in widespread and recalcitrant patients, usually does not produce a prolonged clinical remission and can have hazardous side effects. The authors stated that intravenous immunoglobulin,
rituximab and immunoabsorption have been successfully used in some, but the benefits from their use may require additional studies.

Lagoumintzis and colleagues (2010) noted that current medications for myasthenia gravis (MG) are non-specific and include acetylcholinesterase inhibitors, immunosuppressants, plasma exchange (PE), intravenous immunoglobulin (IVIG) administration and thymectomy. Treatments that selectively target the anti-acetylcholine receptor (AChR) auto-Abs may prove to be more effective and free of side-effects. These investigators reviewed 2 approaches aimed at the development of antigen-specific therapies for MG. The first is specific apheresis of Abs from patients' sera using immobilized recombinant AChR domains as immunoabsorbents. These researchers had shown that the combined recombinant extracellular domains of all human AChR subunits are capable of specifically immunoabsorbing the majority of pathogenic auto-Abs from several MG sera. The second therapeutic approach is the development of non-pathogenic anti-AChR monoclonal Abs that could potentially be used as protective agents by blocking the binding of patients' auto-Abs to the AChR.

Blaha et al (2011) described their experience with PE and IAS in patients with MG. The group of 27 patients consists of 21 patients treated with PE and 6 patients who received IAS. Plasma exchange led to stabilization in 20 patients. In patients treated with IAS, therapy could be discontinued in 2 patients after 13 months of therapy, and the other 4 patients were stabilized without myasthenic crises after 6 to 9 years of therapy. Extra-corporeal elimination therapy through PE or IAS is effective and sometimes life-saving and is safe in the hands of an experienced team (6 % complication rate).
Kohler et al (2011) noted that myasthenic crisis is the most serious life-threatening event in patients with MG, affecting up to 27% within the first 2 years following onset of disease. Extracorporeal removal of circulating Abs against the nicotinic acetylcholine receptor (AChRAb) by methods of therapeutic apheresis, such as PE and IAS had been demonstrated as effective treatment especially in acute situations of myasthenic crisis. These investigators presented the results of a prospective, randomized controlled clinical trial, investigating 19 patients with myasthenic crisis, who were randomized to receive either PE (n = 10) or IAS (n = 9) in addition to combined drug treatment. Patients received 3 to 5 (mean of 3.5 for PE, and 3.4 for IAS) treatments over a period of 7 days with a pre-defined treatment volume of 1.5 L plasma (i.e., 20 to 25 ml/kg plasma representing 0.5 to 0.6 patients' plasma volumes). Clinical courses were monitored using disease specific clinical scores. After initiation of IAS or PE, the mean value of myasthenia scores decreased equally until day 14 of the post-treatment phase. Patients from both treatment groups improved to a stable clinical status of Oosterhuis Classes 1 and 2. Substantial reduction of AChRAb was documented after each session of PE or IAS. During the treatment period, 16 adverse effects (7 serious adverse events, SAE) in the PE and 10 (1 SAE) in the IAS group were observed. The authors concluded that IAS proved to be equally effective compared with PE treatment in patients with myasthenic crisis; 3 to 5 treatment sessions using low plasma volume dosage of 20 to 25 ml/kg were adequate to improve clinically relevant symptoms significantly in most patients.

Felix and colleagues (2015) stated that dilated cardiomyopathy is a common myocardial disease characterized by ventricular chamber enlargement and systolic dysfunction that result in heart failure. In addition to genetic predisposition, viral infection and myocardial inflammation play a causal role in the disease process of dilated cardiomyopathy. Experimental and clinical studies suggested that activation of the humoral immune system, with production of circulating cardiac
autoantibodies, plays an important functional role in the development and progression of cardiac dysfunction in patients with dilated cardiomyopathy. Small open-controlled studies showed that removal of circulating antibodies by immunoadsorption resulted in improvement of cardiac function and decrease in myocardial inflammation. The authors concluded that currently immunoadsorption is an experimental treatment option for improvement of cardiac function -- therapy that calls for confirmation by a placebo-controlled multi-center study.

Also, an UpToDate review on “Possibly effective emerging therapies for heart failure” (Colucci, 2015) states that “Immunoadsorption -- Antibodies against cardiac cell proteins, including mitochondrial proteins, contractile proteins, and beta-receptors, have been identified in dilated cardiomyopathy. If cardiac autoantibodies contribute to myocardial dysfunction, their removal with immunoadsorption might improve left ventricular hemodynamics. Several studies have demonstrated the potential benefit of this approach …. Various types of immunotherapy [including immunoadsorption] have been investigated in patients with HF but such therapy does not have an established clinical benefit except for specific indications in patients with myocarditis”.

**Atopic Dermatitis**

Zink et al (2016) stated that patients with atopic dermatitis (AD) tend to have greatly elevated levels of serum immunoglobulin E (IgE). However, the role of IgE in the pathogenesis of AD is debated. In an open-label, pilot study, these researchers evaluated an anti-IgE-treatment approach by combining extracorporeal IAS and anti-IgE antibody omalizumab in 10 patients with severe, therapy-refractory AD. IgE levels decreased after IAS and decreased continuously in all patients during anti-IgE therapy. The reverse trend was observed during 6 months follow-up without treatment. In parallel with these observations, an improvement in AD was
observed during the treatment period, with aggravation during follow-up. The authors concluded that further research is needed, based on the principle of reducing IgE levels in order to improve clinical symptoms, using a combination anti-IgE treatment approach, adjusted according to IgE levels. The main drawback of this study was the combinational use of IAS and omalizumab. The findings of this pilot study need to be validated by well-designed studies.

Anti-Phospholipid Syndrome

Kronbichler et al (2016) stated that extracorporeal treatments have been used since the 1970s in the management of SLE. A RCT comparing the effectiveness of standard of care (SOC) combined with PE against SOC alone in patients with lupus nephritis revealed no difference in terms of renal outcome. Subsequently, initial expectations have been dampened and further experience with PE is mainly limited to observational studies and single-case reports. Beneficial effects have been reported in patients with refractory disease course or in pregnancy with prior complications due to SLE and anti-phospholipid syndrome (APS). A more specific form of extracorporeal treatment, IAS, has emerged as a valuable option in the treatment of SLE. In line with the PE experience, IAS appeared to have beneficial effects in patients with refractory disease, contraindications to standard immunosuppression or during pregnancy. The mechanism IAS relates to autoantibody removal but for PE removal of activated complement components, coagulation factors, cytokines and micro-particles may also be relevant. Both treatments have good safety profiles although reactions to blood product replacement in PE and procedure-related complications (e.g., bleeding or catheter-related infections) have occurred. The authors concluded that there is a need to more clearly define the clinical utility of PE and IAS in refractory lupus and APS subgroups.
Elevated Lipoprotein(a)

Waldmann and Parhofer (2016) noted that an elevated plasma concentration of lipoprotein(a) (Lp(a)) is an independent risk factor for cardiovascular disease. Lifestyle modification and currently available drugs either fail to effectively lower plasma Lp(a) levels or do not result in clinical benefit. However, lipoprotein apheresis is very efficient in decreasing Lp(a) concentrations. A single apheresis session can acutely decrease Lp(a) by approximately 60 to 75% and weekly or bi-weekly performed apheresis resulted in considerably decreased mean interval concentrations (approximately 25 to 40% reduction). While most apheresis systems (HELP, heparin induced extracorporeal LDL precipitation; DALI, direct adsorption of lipoproteins; lipoprotein apheresis with dextran-sulfate; lipid filtration; IAS) decrease LDL and Lp(a), Lipopac is specific and only decreases Lp(a). The authors stated that Lp(a) apheresis is an expensive and time-consuming process, but is associated with very few side effects. They stated that 2 RCTs gave conflicting results with respect to the effect on angiographic changes; retrospective analyses indicated that regular apheresis translates into clinical benefit in patients with elevated Lp(a); but adequate RCTs are lacking.

Allergic Asthma

In a randomized, controlled, open-label, pilot trial, Lupinek and colleagues (2017) evaluated the safety and efficacy of a single-use IgE immunoadsorber column (IgEnio) regarding the selective depletion of IgE in patients with allergic asthma; and examined if IgEnio can bind IgE-omalizumab immune complexes. A total of 15 subjects were enrolled and randomly assigned to either the treatment group (n = 10) or to the control group (n = 5). Immunoadsorption was done by veno-venous approach, processing the 2-fold calculated plasma volume during each treatment. A minimum average IgE-depletion of 50% after the last cycle in the intention-to-treat population was defined as primary end-point. Safety of the
treatment was studied as secondary end-point. In addition, possible changes in allergen-specific sensitivity were investigated, as well as clinical effects by peak flow measurement and symptom-recording. The depletion of IgE-omalizumab immune complexes was studied in-vitro. IgE immunoadsorption with IgEnio selectively depleted 86.2 % (± 5.1 % SD) of IgE until the end of the last cycle (p < 0.0001). Removal of pollen allergen-specific IgE was associated with a reduction of allergen-specific basophil-sensitivity and prevented increases of allergen-specific skin-sensitivity and clinical symptoms during pollen seasons. IgEnio also depleted IgE-omalizumab immune complexes in-vitro. The therapy under investigation was safe and well-tolerated. During a total of 81 aphereses, 2 SAE were recorded, one of which, an episode of acute dyspnea, possibly was related to the treatment and resolved after administration of anti-histamines and corticosteroids. The authors concluded that the findings of this pilot study indicated that IgE immunoadsorption with IgEnio may be used to treat patients with pollen-induced allergic asthma. Furthermore, the treatment could render allergic patients with highly elevated IgE-levels eligible for the administration of omalizumab and facilitate the desorption of IgE-omalizumab complexes. Moreover, they stated that additional clinical investigations with a higher number of participants are planned to confirm these findings and to further improve treatment efficacy.

These investigators also noted that a drawback of this study was that, due to the uneven distribution of allergic co-morbidities (i.e., rhinitis, dermatitis) in the study groups and due to the low number of study participants (n = 15), effects of the treatment on co-morbidities could not be investigated. The effects of treatment with IgEnio on clinical endpoints will be assessed in follow-up studies. Likewise, effects of long-term treatment will be analyzed in future studies.
Furthermore, an UpToDate review on “Treatment of moderate persistent asthma in adolescents and adults” (Peters et al, 2018) does not mention extracorporeal immunoadsorption as a therapeutic option.

Treatment of Hepatitis B

Han and colleagues (2018) established a novel HBV specific immunoadsorbent for the removing of HBV particles. The anti-HBsAg monoclonal antibody was immobilized on sepharose beads to produce a sepharose anti-HBs column. Then the immunoadsorbent was evaluated and characterized by scanning electron microscopy. In addition, time-dependent effects of the eradication capacity of anti-HBsAg functionalized sepharose beads against HBV were investigated. Proposed immunoadsorbents exhibited a favorable biocompatibility as well as specificity. With the optimized recycle time, the decontamination performance of HBV particles and quantity of HBsAg were assessed either by real-time quantitative PCR or ELISA, which showed that the immunoadsorbent could remove approximately 90 % of the HBV and 90 % of the HBsAg from human plasma samples. The authors concluded that anti-HBsAg functionalized adsorbents introduced in this work exhibited potential for HBV removal and this approach could establish a novel therapeutic option or at least as a combination supplementary therapy strategy with anti-viral drugs for the treatment regimen of HBV.

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 "+":

<table>
<thead>
<tr>
<th>Code</th>
<th>Code Description</th>
</tr>
</thead>
</table>

CPT codes covered if selection criteria are met:
<table>
<thead>
<tr>
<th>Code</th>
<th>Code Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>36516</td>
<td>Therapeutic apheresis; with extracorporeal immunoadsorption, selective adsorption or selective filtration and plasma reinfusion</td>
</tr>
</tbody>
</table>

Other HCPCS codes related to the CPB:

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>J7500</td>
<td>Azathioprine, oral 50 mg</td>
</tr>
<tr>
<td>J7501</td>
<td>Azathioprine, parenteral, 100 mg</td>
</tr>
<tr>
<td>J7502</td>
<td>Cyclosporine, oral, 100mg</td>
</tr>
<tr>
<td>J7515</td>
<td>Cyclosporine, oral, 25 mg</td>
</tr>
<tr>
<td>J7516</td>
<td>Cyclosporine, parenteral, 250 mg</td>
</tr>
</tbody>
</table>

ICD-10 codes covered if selection criteria are met:

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>D59.3</td>
<td>Hemolytic-uremic syndrome [with clinical evidence of serious bleeding with platelet count below 50,000 or the potential for serious bleeding with platelet count below 20,000]</td>
</tr>
<tr>
<td>D69.3</td>
<td>Immune thrombocytopenic purpura [idiopathic thrombocytopenic purpura (ITP) with clinical evidence of serious bleeding with platelet count below 50,000 or the potential for serious bleeding with platelet count below 20,000]</td>
</tr>
<tr>
<td>G70.01</td>
<td>Myasthenia gravis with (acute) exacerbation</td>
</tr>
<tr>
<td>L10.0 - L10.9</td>
<td>Pemphigus [resistant to standard therapy, including dapsone, corticosteroids, and immunosuppressants (e.g., azathrioprine or cyclosporine)]</td>
</tr>
<tr>
<td>M05.00 - M06.9</td>
<td>Rheumatoid arthritis with rheumatoid factor [moderate to severe rheumatoid arthritis (RA), for reduction of signs and symptoms in members who have failed other treatments (e.g., NSAIDS, methotrexate)]</td>
</tr>
</tbody>
</table>
Systemic lupus erythematosus (SLE) [severe for whom other interventions have been unsuccessful, have become intolerable, or are contraindicated]

<table>
<thead>
<tr>
<th>Code</th>
<th>Code Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>M32.10 - M32.9</td>
<td>Systemic lupus erythematosus (SLE) [severe for whom other interventions have been unsuccessful, have become intolerable, or are contraindicated]</td>
</tr>
</tbody>
</table>

ICD-10 codes not covered for indications listed in the CPB (not all inclusive):

<table>
<thead>
<tr>
<th>Code</th>
<th>Code Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>B16.0 - B16.9</td>
<td>Acute hepatitis B</td>
</tr>
<tr>
<td>B18.0</td>
<td>Chronic viral hepatitis B with delta-agent</td>
</tr>
<tr>
<td>B18.1</td>
<td>Chronic viral hepatitis B without delta-agent</td>
</tr>
<tr>
<td>B19.10 - B19.11</td>
<td>Unspecified viral hepatitis B</td>
</tr>
<tr>
<td>D68.61</td>
<td>Antiphospholipid syndrome</td>
</tr>
<tr>
<td>E78.89</td>
<td>Other lipoprotein metabolism disorders [elevated lipoprotein(a)]</td>
</tr>
<tr>
<td>I50.1 - I50.9</td>
<td>Heart failure</td>
</tr>
<tr>
<td>J45.20 - J45.998</td>
<td>Asthma [allergic]</td>
</tr>
<tr>
<td>L20.0 - L20.9</td>
<td>Atopic dermatitis</td>
</tr>
<tr>
<td>M33.00 - M33.99</td>
<td>Dermatopolymyositis</td>
</tr>
<tr>
<td>Q81.9</td>
<td>Epidermolysis bullosa, unspecified [epidermolysis bullosa acquisita]</td>
</tr>
</tbody>
</table>
The above policy is based on the following references:


43. Colucci WS. Possibly effective emerging therapies for heart failure. UpToDate [online serial]. Waltham, MA: UpToDate; reviewed February 2015.


AETNA BETTER HEALTH® OF PENNSYLVANIA

Amendment to
Aetna Clinical Policy Bulletin Number: 0355 Extracorporeal Immunosorption (Proserba Column)

There are no amendments for Medicaid.