BlueCross BlueShield of Alabama

"Unless otherwise noted, coverage for specific indications is effective the date of the FDA approval of that indication."
"Please check Approved by Governing Bodies for FDA approval date."

Name of Blue Advantage Policy:
Vectibix® (panitumumab)

Policy #: 664
Category: Pharmacology
Effective Date: November 10, 2017
Last Review Date: November 2017

Background:
Blue Advantage *medical policy does not conflict with Local Coverage Determinations (LCDs), Local Medical Review Policies (LMRPs) or National Coverage Determinations (NCDs) or with coverage provisions in Medicare manuals, instructions or operational policy letters. In order to be covered by Blue Advantage the service shall be reasonable and necessary under Title XVIII of the Social Security Act, Section 1862(a)(1)(A). The service is considered reasonable and necessary if it is determined that the service is:*

1. *Safe and effective;*
2. *Not experimental or investigational*;*
3. *Appropriate, including duration and frequency that is considered appropriate for the service, in terms of whether it is:*
   - *Furnished in accordance with accepted standards of medical practice for the diagnosis or treatment of the patient's condition or to improve the function of a malformed body member;*
   - *Furnished in a setting appropriate to the patient's medical needs and condition;*
   - *Ordered and furnished by qualified personnel;*
   - *One that meets, but does not exceed, the patient's medical need; and*
   - *At least as beneficial as an existing and available medically appropriate alternative.*

Routine costs of qualifying clinical trial services with dates of service on or after September 19, 2000 which meet the requirements of the Clinical Trials NCD are considered reasonable and necessary by Medicare. Providers should bill* ***Original Medicare* *for covered services that are related to* ***clinical trials*** *that meet Medicare requirements (Refer to Medicare National Coverage Determinations Manual, Chapter 1, Section 310 and Medicare Claims Processing Manual Chapter 32, Sections 69.0-69.11).*

Description of Procedure or Service:

Vectibix® (panitumumab) is a human IgG2 kappa recombinant, monoclonal antibody that binds specifically to the human epidermal growth factor receptor (EGFR). EGFR is expressed in multiple cell lines including epithelial tissues and is over-expressed in certain cancers. EGFR functions in a complex cascade system that affects gene transcription involved with cellular growth, motility, and proliferation. By binding to EGFR, panitumumab interrupts the cascade which ultimately leads to the development of cancer. The blockage of the epidermal growth factor results in a decrease in tumor growth.

In the EGFR cascade, certain protein including the *KRAS* protein normally function as switches in the kinase pathway activated between cell surface EGFR and downstream signaling. The gene mapping normal, non-mutated KRAS production is referred to as a wild-type gene. Mutations in this KRAS gene occur in 30% to 50% of colorectal cancers as well as other tumor types. These mutations in *KRAS* cause activation of the EGFR pathway beyond the point at which panitumumab would bind with EGFR and interrupt the cascade. This renders panitumumab and other anti-EGFR agents ineffective against those tumors expressing *KRAS* mutations.

Policy:

Effective for dates of service on or after November 10, 2017:

Blue Advantage will treat **Vectibix® (panitumumab)** as a treatment of **stage IV colon, rectal, colorectal, small bowel, anal adenocarcinoma, or appendiceal adenocarcinoma** as a **covered benefit** when **ALL** of the following criteria are met:

- Used as a single agent or as part of combination therapy; **AND**
- NRAS/KRAS gene mutation testing is documented and the tumor is determined to be NRAS/KRAS wild type*****; **AND**
- Vectibix® (panitumumab) is to be used for only one line of therapy; **AND**
- Vectibix® (panitumumab) is NOT used in combination with other monoclonal antibodies or anti-VEGF agents (e.g. bevacizumab); **AND**
- The individual has not received prior treatment with cetuximab** (e.g. Erbitux®)

***Note:** NRAS/KRAS wild-type means the gene is normal or lacking mutations

****Note**: A course of cetuximab discontinued because of adverse reaction, not progressive disease, is not considered prior treatment.

Blue Advantage will treat **Vectibix®** (**panitumumab)** as a **non-covered benefit** and as **investigational** when the above criteria are not met including, but not limited to **treatment of penile cancer, squamous cell anal carcinoma, and lung cancer.**

Effective for dates of service September 1, 2015and prior to November 10, 2017
Blue Advantage will treat **Vectibix® (panitumumab)** as a **covered benefit** as a treatment for individuals who meet **ALL** of the following criteria:

- As a single agent or as part of combination therapy for Stage IV colon, rectal, colorectal, small bowel, anal adenocarcinoma, or appendiceal adenocarcinoma; **AND**
- KRAS gene mutation testing is documented and the tumor is determined to be KRAS wild type*****; **AND**
- Vectibix® (panitumumab) is to be used for only one line of therapy; **AND**
- Vectibix® (panitumumab) is NOT used in combination with other monoclonal antibodies or anti-VEGF agents (e.g. bevacizumab); **AND**
- The individual has not received prior treatment with cetuximab** (e.g. Erbitux®)

***Note:** KRAS wild-type means the gene is normal or lacking mutations

****Note**: A course of cetuximab discontinued because of adverse reaction, not progressive disease, is not considered prior treatment.

Blue Advantage will treat **Vectibix®** (**panitumumab)** as a **non-covered benefit** and as **investigational** when the above criteria are not met including, but not limited to **treatment of penile cancer, squamous cell anal carcinoma, and lung cancer.**

Blue Advantage does not approve or deny procedures, services, testing, or equipment for our members. Our decisions concern coverage only. The decision of whether or not to have a certain test, treatment or procedure is one made between the physician and his/her patient. Blue Advantage administers benefits based on the members' contract and medical policies. Physicians should always exercise their best medical judgment in providing the care they feel is most appropriate for their patients. Needed care should not be delayed or refused because of a coverage determination.

Key Points:

Colorectal carcinoma

According to the National Institute for Health, colorectal cancer is the fourth most frequently diagnosed cancer and the second leading cause of death in the United States. . It is estimated during 2017, there will be 135,430 Americans diagnosed with colon and rectal cancer and 50,260 will die from colon and rectal cancer combined. Colorectal cancer refers to malignancies originating from the large intestine (colon) or the rectum. The term colorectal cancer does not include anal cancer. Anal cancer refers to malignancies developing from anal tissue (e.g., anus, anal canal or anorectum) which include the opening of the rectum to the outer body. Anal cancer occurs infrequently and represents 4% of all cancers of the lower gastrointestinal tract.

Panitumumab is currently one of two intravenous anti-EGFR therapies approved by the United States (US) Food and Drug Administration (FDA) to treat malignancies. The monoclonal antibodies directed against EGFR include panitumumab and cetuximab. Panitumumab binds

specifically to EGFR, which inhibits the attachment of ligands. This prevents the activation of receptor-associated kinases, resulting in the start of apoptosis and inhibition of cell growth. The blockade of epidermal growth has side effects, with skin rash occurring most frequently. Various studies have drawn correlations between the frequency and severity of rash to tumor response rate.

On September 27, 2006, the FDA provided an accelerated approval for panitumumab (Vectibix®) as a treatment of EGFR-expressing metastatic colorectal carcinoma (mCRC) with disease progression on or following fluoropyrimidine-, oxaliplatin-, and irinotecan-containing chemotherapy regimens. The approval was based on progression-free survival (PFS) data from a randomized controlled trial (RCT) involving 463 individuals with mCRC. Individuals had been previously treated with fluoropyrimidine, oxaliplatin and irinotecan, but progressed on or following therapy. There was a statistically significant PFS prolongation of 96 days with panitumumab compared to 60 days with best supportive care (BSC). Nineteen (8%) partial response rates with a median duration of 17 weeks were noted in individuals randomized to receive panitumumab. There were no measurable responses in the control arm.

The product information label was updated in June 2008, indicating panitumumab as a single agent for the treatment of EGFR-expressing mCRC that has progressed despite standard chemotherapy. Additional warnings were added to the label in 2009, stating there has been increased toxicity with combination chemotherapy. The 2009 label update also included an updated recommendation that due to a lack of benefit, panitumumab was not indicated for mCRC with KRAS mutation in codon 12 or 13. The limitation of use was further amended in 2012, stating panitumumab was not indicated for treatment of individuals with KRAS-mutant mCRC or unknown KRAS-mutation status.

A new indication as first-line treatment, in combination with FOLFOX (fluorouracil, leucovorin, and oxaliplatin), of individuals with wild-type KRAS (exon 2 in codons 12 or 13) mCRC, as determined by an FDA-approved test, was added to the label in May of 2014.

The Panitumumab Advanced Colorectal Cancer Evaluation (PACCE) study was a phase IIIb randomized, open-label clinical trial evaluating cohorts of a chemotherapy regimen including bevacizumab, with and without panitumumab as first-line treatment of individuals with previously untreated metastatic colorectal carcinoma (mCRC). Investigators chose a 5-FU, leucovorin and oxaliplatin-based regimen (Ox-CT; n= 823) or a 5-FU, leucovorin and irinotecan-based regimen (Iri-CT; n= 230), each with bevacizumab. Individuals were randomized to receive the selected regimen, or chemotherapy with the addition of panitumumab. A statistically significant difference in PFS in favor of the control arm (without panitumumab) was unveiled at the first planned interim analysis which resulted in a discontinuation of panitumumab. In the final analysis, median PFS was 10.1 months for panitumumab and 11.7 months for the control group (hazard ratio [HR], 1.19; 95% confidence interval [CI], 0.79 to 1.79). In a safety analysis of 804 individuals in the Ox-CT cohort and 224 individuals in the Iri-CT cohort, both groups had more adverse events (AEs) of grade 3 or higher in the panitumumab cohorts compared to the control groups (Ox-CT 367 (90%) versus 305 (77%), respectively; Iri-CT 100 (90%) versus 71 (63%) respectively). Serious AEs included diarrhea, infections and pulmonary embolism. Seven (1%) deaths were attributed to be panitumumab related. The authors concluded the decreased

PFS and increased serious AEs do not support panitumumab in combination with bevacizumab and oxaliplatin- or irinotecan-based chemotherapy as a treatment for mCRC. "Administration of chemotherapy and dual EGFR/VEGF inhibition should be conducted only in a research setting."

The National Comprehensive Cancer Network® (NCCN®) colon and rectal Clinical Practice Guidelines in Oncology™ (V2.2017) "Strongly recommends against the use of therapy involving the concurrent combination of an anti-EGFR agent (cetuximab or panitumumab) and an anti-vascular endothelial growth factor (VEGF) agent (bevacizumab)."

The phase III, Panitumumab Randomized Trial in Combination with Chemotherapy for Metastatic Colorectal Cancer to Determine Efficacy (PRIME) study randomized 1183 individuals with untreated metastatic colorectal carcinoma to either regimen of FOLFOX4 or to FOLFOX4 plus panitumumab. The primary endpoint was PFS. A total of 1096 (93%) of the participants had KRAS information available. No KRAS mutations in exon 2 were noted in 656 participants (60%) and KRAS mutations in exon 2 were noted in 440 individuals (40%). PFS was significantly improved in those with wild-type (WT) KRAS and who were treated with FOLFOX4 + panitumumab versus FOLFOX4 alone (median PFS 9.6 months vs. 8.0 months; HR, 0.80; 95% CI, 0.66 to 0.97; P=0.02). There was a nonsignificant improvement in overall survival of 4.2 months (23.9 vs. 19.7 months, P=0.07) for those treated with FOLFOX4 + panitumumab vs. FOLFOX4 alone. Participants with mutant KRAS had PFS significantly reduced with the addition of panitumumab to FOLFOX4 compared to FOLFOX4 alone (median PFS 7.3 months vs. 8.8 months; HR, 1.29; 95% CI, 1.04 to 1.62; P=0.02).

The medically necessary criteria for excluding previous use of EGFR monoclonal antibody therapy prior to treatment with panitumumab is based on the selection criteria in the pivotal trial as well as the expert view of medical practitioners practicing in the clinical area of oncology, and who have familiarity with the available evidence. The NCCN® Clinical Practice Guidelines (CPG) for colon cancer and rectal cancer (V2.2017) includes the following clarification:

> There are no data, nor is there compelling rationale, to support the use of panitumumab after clinical failure on cetuximab, or the use of cetuximab after clinical failure on panitumumab. As such, the use of one of these agents after therapeutic failure on the other is not recommended.

In a small retrospective series, Saif (2010) reported results of 15 individuals treated with panitumumab after having failed prior cetuximab therapy. Eleven individuals were evaluable for responses and tissue samples from three tumors were retrospectively assessed for KRAS status. Three individuals had minor radiographic improvement and 3 other individuals had stable disease (SD). Median duration of SD was 4 months with a range from 2 to 8 months. The investigators concluded the evidence may suggest panitumumab, after cetuximab failure, may "exert antitumor activity through different mechanisms, although, further work is required to investigate this potentially interesting issue."

Price and colleagues (2014) reported results from the first direct comparison trial which was an open-label, randomized, multi-center phase III study that compared panitumumab to cetuximab in individuals with chemotherapy-refractory metastatic colorectal cancer. A total of 1010

individuals were enrolled with 999 actually starting treatment. The panitumumab treatment group included 499 participants with 500 participants assigned to the cetuximab treatment group. The primary endpoint of overall survival showed panitumumab was noninferior to cetuximab (P=0.0007). The median overall survival was 10.4 months in the panitumumab group and 10.0 months in the cetuximab group (HR, 0.97; 95% CI, 0.84-1.11). The incidence of adverse reactions was generally similar. The noted differences were a lower occurrence rate of grade 3-4 infusion reaction with panitumumab (<0.5%) compared to cetuximab (2%) and a higher occurrence rate of grade 3-4 hypomagnesaemia with panitumumab (7%) compared to the cetuximab (3%) cohorts. The authors concluded that although the results suggest noninferiority between the two anti-EGFR monoclonal antibodies, the study did not "directly address the sequencing and timing of therapy" and additional research is needed. Additional studies are also needed to confirm the interchangeability of anti-EGFR monoclonal antibodies in other clinical indications.

Panitumumab is indicated for individuals with tumors that express the wild-type KRAS gene. The American Society of Clinical Oncology issued a provisional, consensus clinical opinion based on systematic reviews of literature primarily from phase II and III clinical trials involving individuals with metastatic colorectal cancer:

> All patients with metastatic colorectal carcinoma who are candidates for anti-EGFR antibody therapy should have their tumor tested for KRAS mutations in a CLIA-accredited laboratory. If KRAS mutation in codon 12 or 13 is detected, then patients with metastatic colorectal carcinoma should not receive anti-EGFR antibody therapy as part of their treatment.

The ASCO published a provisional opinion update in 2016 regarding extended RAS mutation testing in metastatic CRC to predict response to anti-EGFR monoclonal antibody therapy. The opinion was based on evidence from 13 articles on KRAS mutations (11 systematic reviews, 2 health technology assessments) and 2 articles on NRAS testing. The opinion stated that:

> Subgroup analyses of patients with any of the less common RAS mutations are small, and there is inadequate evidence to provide a definitive opinion on the lack of benefit for the use of anti-EGFR antibodies for patients whose cancer harbors any specific RAS mutation other than the exon 2 KRAS mutation.

The updated NCCN (V.2.2017) guidelines include recommendations for NRAS/KRAS gene testing for all stage IV colon and rectal disease.

Studies of metastatic colorectal carcinoma treatment have shown there are subsets of individuals who are not as responsive to anti-EGFR monoclonal antibodies. To understand the variation, there is ongoing research into the genetic signaling pathways that promote the growth of specific cells. The Kirsten rat sarcoma virus also known as the KRAS gene is being analyzed for mutations and correlation of response to anti-EGFR monoclonal antibodies. The desired goal for KRAS status mutation analysis is to identify individuals who would not respond to anti-EGFR monoclonal antibody therapy, thereby saving them the time, expense and unnecessary toxicity of ineffective therapies.

Freeman and colleagues analyzed gene mutations in metastatic colorectal tumor samples from three phase II panitumumab studies. Progression-free survival (PFS) was favored in individuals with wild-type KRAS compared with individuals with mutant KRAS (HR 0.4; 95% CI, 0.2-0.7; p=0.002). Median PFS was 16.2 weeks for the wild-type KRAS versus 7.4 weeks for the mutant KRAS cohort. In a systematic review and meta-analysis, Adelstein and colleagues (2011) analyzed the treatment effect of KRAS mutation in response to anti-EGFR antibodies to treat metastatic colorectal cancer (CRC). Data analyzed from 11 studies included 8924 individuals treated with anti-EGFR therapy. With anti-EGFR therapy, the hazard ratio for progressive disease in individuals with KRAS wild-type was 0.80 (4436 individuals 95% CI, 0.64, 0.99) and 1.11 (3119 individuals, 95% CI, 0.97, 1.27) in individuals with the mutant KRAS. The authors concluded the status of KRAS mutation modified the treatment effect of anti-EGFR therapy in the treatment of metastatic CRC.

In a prospective-retrospective analysis of the PRIME study, Douillard and colleagues (2013) investigated if other activating RAS mutations, besides the KRAS mutation in exon 2, may be negative predictive biomarkers for anti-EGFR therapy. The updated analysis showed a significant improvement in overall survival of 4.4 months (23.8 months vs. 19.4 months, P=0.03) in those treated with FOLFOX4 + panitumumab vs. FOLFOX4 alone. In this exploratory analysis, RAS status had been evaluated in 90% (1060 participants) of the total 1183 participants in the PRIME trial. There were 512 (48%) individuals who had nonmutated RAS, which meant no KRAS or NRAS mutations in exons 2, 3, or 4. Mutated RAS (any KRAS or NRAS mutations in exon 2, 3, or 4) were identified in 548 (52%) participants. A total of 108 (17%) of individuals without mutated KRAS in exon 2 had other RAS mutations in other exons. This subgroup of participants had non-significantly shorter PFS and OS in the panitumumab + FOLFOX4 treatment group compared to the FOLFOX4 cohort. The authors note the data "further assesses the hypothesis that additional activating RAS mutations predict unresponsiveness to panitumumab treatment." However, these findings need to be confirmed with meta-analyses or pooled trial data of anti-EGFR therapy.

NCCN clinical practice guidelines (V2.2017) recommend metastatic colorectal tumor tissue to be genotyped for RAS mutations (KRAS and NRAS). Individuals with "any known KRAS mutation (exon 2 or non-exon 2) or NRAS mutation should not be treated with either cetuximab or panitumumab, either alone or in combination with other anticancer agents, because they have virtually no benefit and the exposure to toxicity and expense cannot be justified."

The data suggest that the metastatic colorectal population with wild-type KRAS mutation status benefited more from panitumumab alone as compared with those with the activating KRAS mutation. Therefore, analysis of KRAS status is recommended to facilitate treatment plans.

Squamous cell anal cancer is the most common histologic form of anal cancer. Adenocarcinoma and melanoma of the anal canal represents infrequently occurring subtypes of anal cancer. The NCCN Anal Carcinoma Clinical Practice Guidelines (V2.2017) recommends management of anal adenocarcinoma according to the NCCN Rectal Cancer Clinical Practice Guidelines (2017). Specialty consensus opinion also supports the NCCN recommendations to treat stage IV anal adenocarcinoma similar to stage IV colorectal adenocarcinoma. Of note, the NCCN guidelines

do not recognize the use of panitumumab for treatment of squamous cell anal cancer as an off-label indication.

Other Carcinomas
Penile cancer is a rare disease with squamous cell carcinoma being the most common type of penile cancer. Due to the rarity of this disease, there are no large randomized trials investigating the use of panitumumab as a treatment. The updated National Comprehensive Cancer Network® (NCCN®) Clinical Practice Guidelines in Oncology for penile cancer (2017) no longer include the off-label recommendation for panitumumab as a single agent in second-line therapy as palliative therapy. The guidelines recommend clinical trials for second-line or palliative therapies as "No standard second-line systemic therapy exists."

Waddell and colleagues (2013) investigated panitumumab as first-line therapy for advanced oesophagogastric carcinoma in a phase III open label trial known as REAL3. Participants were randomized to epirubicin, oxaliplatin, and capecitabine (EOC) or to a modified dose EOC with panitumumab (mEOC+P). The primary endpoint was overall survival. However, after a preplanned review by the independent data monitoring committee reported a decreased OS with panitumumab compared to EOC alone, the trial was halted. Additionally, there was an increased incidence of adverse events with the panitumumab regimen, which included grade 3 and 4 diarrhea, mucositis, rash and hypomagnesemia. Based on the inferior OS and PFS, the authors concluded panitumumab could not be recommended when combined with EOC in unselected individuals with advanced oesophagogastric adenocarcinoma. Additionally, studies to identify biomarkers that may identify a subgroup of individuals who may benefit from the panitumumab are recommended.

A phase III study investigating cisplatin and fluorouracil with or without panitumumab as a treatment for individuals with recurrent or metastatic squamous-cell carcinoma of the head and neck (SPECTRUM) was reported by Vermorken (2013). A total of 327 individuals were randomized to the panitumumab treatment group and 330 were assigned to the control group. The primary endpoint of the open-label study was overall survival (OS). The median OS for the panitumumab cohort was 11.1 months compared to 9.0 months in the control group (HR, 0.873; 95% CI, 0.729-1.046; P=0.1403). There were 14 (4%) deaths in the treatment group, with 5 (2%) deaths attributed to panitumumab. In the control, group, there were 8 (2%) treatment related deaths. The authors concluded that the addition of panitumumab to chemotherapy did not improve OS.

In 2015, two open-label, randomized, controlled, phase 2 trials were reported addressing the use of panitumumab for unresected, locally advanced, squamous-cell carcinoma of the head and neck. The first, reported by Mesia, the CONCERT-1 trial, involved 150 subjects aged 18 years and older with stage III, IVa, or IVb, previously untreated, measurable (≥ 10 mm for at least one dimension), locally advanced squamous-cell carcinoma of the head and neck (non-nasopharyngeal) and an Eastern Cooperative Oncology Group (ECOG) performance status of 0-1. Subjects were randomly assigned in a 2:3 ratio to receive treatment with either open-label chemoradiotherapy (n=63; 3 cycles of cisplatin at 100 mg/m2) or panitumumab plus chemoradiotherapy (n=87; 3 cycles of intravenous panitumumab 9.0 mg/kg every 3 weeks plus cisplatin at 75 mg/m2). At 2 years, local-regional control was reported to be 68% (95% CI; 54-

78) in the chemoradiotherapy group and 61% (95% CI; 50-71) in the panitumumab plus chemoradiotherapy group. The most frequent grade 3-4 adverse events were dysphagia (27% of subjects in the chemoradiotherapy group vs. 40% in the panitumumab plus chemoradiotherapy group), mucosal inflammation (24% vs. 55%, respectively), and radiation skin injury (13% vs. 31%, respectively). Serious adverse events were reported in 32% of subjects in the chemoradiotherapy group vs. 43% of subjects in the panitumumab plus chemoradiotherapy group. The authors concluded that the addition of panitumumab to standard fractionation radiotherapy and cisplatin did not confer any benefit.

The second report, by Giralt, provided results of the CONCERT-2 trial. This trial used the same inclusion and exclusion criteria as the CONCERT-1 trial but involved randomly assigning subjects in a 2:3 ratio to either chemoradiotherapy (n=61; 2 cycles of cisplatin at 100 mg/m^2 during radiotherapy) or to radiotherapy plus panitumumab (n=90; 3 cycles of panitumumab at 9 mg/kg every 3 weeks administered with radiotherapy). The authors reported that local-regional control at 2 years was 61% (95% CI, 47-72) in the chemoradiotherapy group and 51% (95% CI, 40-62) in the radiotherapy plus panitumumab group. The most frequent grade 3-4 adverse events were mucosal inflammation (40% of subjects in the chemoradiotherapy group vs. 42% of subjects in the radiotherapy plus panitumumab group), dysphagia (20% vs. 40%, respectively), and radiation skin injury (11% vs. 24%, respectively). Serious adverse events were reported in 40% of subjects in the chemoradiotherapy group vs. 34% of subjects in the radiotherapy plus panitumumab group. The authors concluded that panitumumab cannot replace cisplatin in combined treatment with radiotherapy.

There are ongoing trials studying the expanded use of panitumumab for additional indications, such as solid tumors including lung cancer, and at different stages of various diseases. The use of panitumumab in combination with a variety of chemotherapy or biologic agents is also under investigation.

Key Words:

Metastatic colorectal cancer (CRC), KRAS mutation analysis, monoclonal antibody, human epidermal growth factor receptor (EGFR), non-small-cell lung cancer, Vectibix, panitumumab

Approved by Governing Bodies:

On September 27, 2006, the U.S. FDA approved panitumumab (Vectibix™; a product of Amgen, Inc) for the treatment of patients with EGFR-expressing, metastatic colorectal cancer with disease progression on or following fluoropyrimidine-, oxaliplatin-, and irinotecan-containing chemotherapy regimens. .

Benefit Application:

Coverage is subject to member's specific benefits. Group specific policy will supersede this policy when applicable.

Current Coding:

HCPCS:

J9303 Injection, Panitumumab, 10 mg

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Policy History:

Medical Policy Group, September 2016
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