Clinical Policy Bulletin: Neonatal Hyperbilirubinemia

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Number: 0332

Policy

I. Assessment of Neonatal Hyperbilirubinemia:

Aetna considers measurement of end-tidal carbon monoxide (CO) corrected for ambient CO (ETCOc), used either alone or in combination with the simultaneous measurement of total serum bilirubin (TSB) concentration experimental and investigational because measurement of ETCOc has not been proven to improve prediction of development of significant neonatal bilirubinemia over TSB alone.

Aetna considers genotyping of SLCO1B1 and UGT1A1 experimental and investigational for assessing risk of neonatal hyperbilirubinemia because the clinical value of this approach has not been established.

II. Treatment of Hyperbilirubinemia in Term and Near-Term Infants:

Aetna considers phototherapy medically necessary for term and near-term infants according to guidelines published by the American Academy of Pediatrics (AAP). The following are general age-in-hours specific total serum bilirubin (TSB) threshold values for phototherapy based upon gestational age and the presence or absence of risk factors (isoimmune hemolytic disease, glucose-6-phosphate dehydrogenase [G6PD] deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis, or albumin of less than 3.0 g/dL [if measured]):

<table>
<thead>
<tr>
<th>Age in hours</th>
<th>Total Serum Bilirubin (TSB) Level in mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk*</td>
<td>Low</td>
</tr>
<tr>
<td>24</td>
<td>&gt;12</td>
</tr>
<tr>
<td>48</td>
<td>&gt;15</td>
</tr>
<tr>
<td>72</td>
<td>&gt;18</td>
</tr>
</tbody>
</table>

* Low Risk: ≥38 weeks gestation and without risk factors; Medium Risk: ≥38 weeks gestation with risk factors or 35 to 37 6/7 weeks gestation without risk factors; High Risk: 35 to 37 6/7 weeks gestation with risk factors.

Notes: Prophylactic phototherapy is considered medically necessary for infants showing a rapid rise in bilirubin (greater than 1 mg/dL/hour) and as a temporary measure when one is contemplating exchange transfusion. Clofibrate in combination with phototherapy for neonatal hyperbilirubinemia is considered experimental and investigational.
Aetna considers exchange transfusion medically necessary for term and near-term infants according to guidelines published by the American Academy of Pediatrics (AAP). The following are general age-in-hours specific TSB threshold values for exchange transfusion based upon gestational age and the presence or absence of risk factors (isoimmune hemolytic disease, glucose-6-phosphate dehydrogenase [G6PD] deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis, or albumin of less than 3.0 g/dL [if measured]):

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<td>&gt;25</td>
</tr>
</tbody>
</table>

* Low Risk: ≥38 weeks gestation and without risk factors; Medium Risk: ≥38 weeks gestation with risk factors or 35 to 37 6/7 weeks gestation without risk factors; High Risk: 35 to 37 6/7 weeks gestation with risk factors.

According to available guidelines, inpatient treatment may be considered medically necessary for healthy full-term infants who present with a TSB greater than or equal to 20 mg/dL in the first post-natal week. Inpatient treatment is generally not medically necessary for healthy full-term infants with a TSB less than 20 mg/dL, as these infants can usually be treated with expectant observation or home phototherapy. Inpatient treatment may be medically necessary for preterm infants who present with a TSB greater than or equal to 18 mg/dL. Inpatient treatment is not generally medically necessary for preterm infants who present with a TSB less than 18 mg/dL, as these infants can usually be treated with expectant observation or home phototherapy.

III. Consistent with available guidelines, continued phototherapy is not medically necessary for healthy term infants when the following criteria for discontinuation of phototherapy are met:

<table>
<thead>
<tr>
<th>Age in days</th>
<th>Total Serum Bilirubin (TSB) Level in mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full term infant &gt; 1 day of age</td>
<td>≤14</td>
</tr>
<tr>
<td>Preterm &lt; 5 days old</td>
<td>10 or less</td>
</tr>
<tr>
<td>Healthy preterms &gt; 5 or more days of age</td>
<td>12 or less</td>
</tr>
</tbody>
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A delay in discharge from the hospital in order to observe the infant for rebound once the bilirubin has decreased is not considered medically necessary. According to available guidelines, no further measurement of bilirubin is necessary in most cases.

IV. Preterm Infants:

Aetna considers management of physiologic hyperbilirubinemia medically necessary in preterm infants (defined as an infant born prior to 37 weeks gestation) according to guidelines published by the AAP. In preterm infants, phototherapy should be initiated at 50 to 70 % of the maximum indirect levels below:
**Birth weight in grams** | **Maximum Indirect Serum Bilirubin Concentration in mg/dL**
---|---
Uncomplicated | Complicated *

<table>
<thead>
<tr>
<th>Birth weight in grams</th>
<th>Uncomplicated</th>
<th>Complicated *</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1,000</td>
<td>≥12</td>
<td>≥10</td>
</tr>
<tr>
<td>1,000 - 1,250</td>
<td>≥12</td>
<td>≥10</td>
</tr>
<tr>
<td>1,251 - 1,499</td>
<td>≥14</td>
<td>≥12</td>
</tr>
<tr>
<td>1,500 - 1,999</td>
<td>≥16</td>
<td>≥15</td>
</tr>
<tr>
<td>2,000 - 2,500</td>
<td>≥20</td>
<td>≥18</td>
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* Complications include but are not limited to prenatal asphyxia, acidosis, hypoxia, hypoalbuminemia, meningitis, intraventricular hemorrhage, hemolysis, hypoglycemia, or signs of kernicterus.

**V. Home Phototherapy:**

Aetna considers home phototherapy for physiologic jaundice in healthy infants with a gestational age of 35 weeks or more medically necessary if all of the following criteria are met:

A. The infant is otherwise ready to be discharged from the hospital; *and*
B. The infant is feeding well, is active, appears well; *and*
C. TSB is less than 20 to 22 mg/dL in term infants, or less than 18 mg/dL in preterm infants; *and*
D. Arrangements have been made to evaluate the infant within 48 hours after discharge by an early office/clinic visit to the pediatrician, or by a home visit by a well-trained home health care nurse who should be able to:

- Be available for follow-up clinical assessments and blood drawing as determined to be necessary by the responsible physician based on changes in bilirubin levels
- Clinically assess the initial level of jaundice
- Draw blood for bilirubin determinations
- Encourage frequent feedings
- Explain all aspects of the phototherapy system to the parents
- Oversee set-up of the phototherapy system
- Weigh the infant in the home

*Note:* If levels do not respond by stabilizing (+/- 1 mg/dL) or declining, more intensive phototherapy may be warranted.

**VI. Metalloporphyrins:**

Aetna considers the use of metalloporphyrins (e.g., stannsoporfin (tin mesoporphyrin), Stanate, WellSpring Pharmaceutical Corporation, Neptune, NJ) for the treatment of neonatal jaundice experimental and investigational because their safety and effectiveness for this indication has not been established.

**VII. Antenatal Phenobarbital:**

Aetna considers the use of antenatal phenobarbital to reduce neonatal jaundice in red cell isoimmunized pregnant women experimental and investigational because its effectiveness has not been established.
Background

Aetna’s policy on treatment of hyperbilirubinemia in infants is adapted from guidelines from the American Academy of Pediatrics.

There is insufficient evidence to support the use of metalloporphyrins (e.g., stannsoporfin (tin mesoporphyrin), Stanate, WellSpring Pharmaceutical Corporation, Neptune, NJ) for the treatment of neonatal jaundice. Guidelines from the AAP stated: “There is now evidence that hyperbilirubinemia can be effectively prevented or treated with tin-mesoporphyrin, a drug that inhibits the production of heme oxygenase. Tin-mesoporphyrin is not approved by the U.S. Food and Drug Administration. If approved, tin-mesoporphyrin could find immediate application in preventing the need for exchange transfusion in infants who are not responding to phototherapy.” A systematic evidence review prepared for the Cochrane Collaboration (Suresh et al, 2003) concluded that, based upon limitations of the evidence, “[r]outine treatment of neonatal unconjugated hyperbilirubinemia with a metalloporphyrin cannot be recommended at present.”

In a Cochrane review, Thomas et al (2007) stated that neonates from isoimmunized pregnancies have increased morbidity from neonatal jaundice. The increased bilirubin from hemolysis often needs phototherapy, exchange transfusion or both after birth. Various trials in pregnant women who were not isoimmunized but had other risk factors for neonatal jaundice have shown a reduction in need for phototherapy and exchange transfusion by the use of antenatal phenobarbital. A recent retrospective case-controlled study showed reduction in the need for exchange transfusion for the neonates from isoimmunized pregnancies. These investigators evaluated the effects of antenatal phenobarbital in red cell isoimmunized pregnancies in reducing the incidence of phototherapy and exchange transfusion for the neonate. Randomized and quasi-randomized controlled trials of pregnant women established to have红cell isoimmunization in the current pregnancy during their antenatal testing and given phenobarbital alone or in combination with other drugs before birth were selected for review. All 3 review authors independently assessed study eligibility and quality. No studies met the inclusion criteria for this review. The authors concluded that the use of antenatal phenobarbital to reduce neonatal jaundice in red cell isoimmunized pregnant women has not been evaluated in randomized controlled trials.

Morris and colleagues (2008) compared aggressive versus conservative phototherapy for infants with extremely low birth weight. These investigators randomly assigned 1,974 infants with extremely low birth weight at 12 to 36 hours of age to undergo either aggressive or conservative phototherapy. The primary outcome was a composite of death or neurodevelopmental impairment determined for 91 % of the infants by investigators who were unaware of the treatment assignments. Aggressive phototherapy, as compared with conservative phototherapy, significantly reduced the mean peak serum bilirubin level (7.0 versus 9.8 mg/dL [120 versus 168 micromol/L], p < 0.01) but not the rate of the primary outcome (52 % versus 55 %; relative risk, 0.94; 95 % confidence interval [CI]: 0.87 to 1.02; p = 0.15). Aggressive phototherapy did reduce rates of neurodevelopmental impairment (26 %, versus 30 % for conservative phototherapy; relative risk, 0.86; 95 % CI: 0.74 to 0.99). Rates of death in the aggressive-phototherapy and conservative-phototherapy groups were 24 % and 23 %, respectively (relative risk, 1.05; 95 % CI: 0.90 to 1.22). In pre-planned subgroup analyses, the rates of death were 13 % with aggressive phototherapy and 14 % with conservative phototherapy for infants with a birth weight of 751 to 1,000 g and 39 % and 34 %, respectively (relative risk, 1.13; 95 % CI: 0.96 to 1.34), for infants with a birth weight of 501 to 750 g. The authors concluded that aggressive phototherapy did not significantly reduce the rate of death or neurodevelopmental impairment. The rate of neurodevelopmental impairment alone was significantly reduced with aggressive phototherapy. This reduction may be offset by an increase in mortality among infants weighing 501 to 750 g at birth.

Guidelines from the Canadian Paediatric Society (2007) found that phenobarbitol, studied as a means of preventing severe hyperbilirubinemia in infants with G6PD deficiency, did not improve clinically important outcomes in a randomized controlled clinical study (Murki et al, 2005).

In a prospective double-blind study, De Luca et al (2008) compared the accuracy of a new transcutaneous bilirubinometer, BiliMed (Medick SA, Paris, France) with BiliCheck (Respirationics, Marietta, GA), a widely available instrument, and with total serum bilirubin (TSB) measurement. A total of 686 healthy newborns needing measurement of their bilirubin were enrolled over a 4-month period.
Serum and transcutaneous bilirubin measurements were taken with both devices within 15 mins. The order of use of the instruments was randomized. The linear regression analysis showed a better correlation between BiliCheck and serum bilirubin ($r = 0.75$) than between BiliMed and serum bilirubin ($r = 0.45$). BiliCheck variability ($+/− 2$ SD of the mean bias from serum bilirubin) was within -87.2 to 63.3 micromol/L, while BiliMed variability was within -97.5 to 121.4 micromol/L. The receiver operating characteristic analysis (for serum bilirubin levels greater than 205.2 micromol/L or greater than 239.4 micromol/L) showed significantly higher areas under the curve for BiliCheck than those for BiliMed ($p < 0.001$). The authors concluded that despite the potential practical advantages of BiliMed, its reduced diagnostic accuracy in comparison with BiliCheck does not justify its use in clinical practice.

Trikalinos et al (2009) reviewed the effectiveness of specific screening modalities to prevent neonatal bilirubin encephalopathy. These researchers identified studies through Medline searches, perusing reference lists and by consulting with United States Preventive Services Task Force (USPSTF) lead experts. They included English-language publications evaluating the effects of screening for bilirubin encephalopathy using early TSB, transcutaneous bilirubin (TcB) measurements, or risk scores. Severe hyperbilirubinemia was used as a surrogate for possible chronic bilirubin encephalopathy (CBE), because no studies directly evaluated the latter as an outcome. These investigators calculated the sensitivity and specificity of early TSB, TcB measurements, or risk scores in detecting hyperbilirubinemia. A total of 10 publications (11 studies) were eligible. Seven (2 prospective) studies evaluated the ability of risk factors ($n = 3$), early TSB ($n = 3$), TcB ($n = 2$), or combinations of risk factors and early TSB ($n = 1$) to predict hyperbilirubinemia (typically TSB greater than 95th hour-specific percentile 24 hours to 30 days post-partum). Screening had good ability to detect hyperbilirubinemia: reported area-under-the-curve values ranged between 0.69 and 0.84, and reported sensitivities and specificities suggested similar diagnostic ability. Indirect evidence from 3 descriptive uncontrolled studies suggested favorable associations between initiation of screening and decrease in hyperbilirubinemia rates, and rates of treatment or re-admissions for hyperbilirubinemia compared with the baseline of no screening. No study assessed harms of screening. The authors concluded that effects of screening on the rates of bilirubin encephalopathy are unknown. Although screening can predict hyperbilirubinemia, there is no robust evidence to suggest that screening is associated with favorable clinical outcomes.

The USPSTF and the Agency for Healthcare Research and Quality (2009) reported on the effectiveness of various screening strategies for preventing the development of CBE. The USPSTF reviewed experimental and observational studies that included comparison groups. For harms associated with phototherapy, case reports or case series were also included. The USPSTF concluded that the evidence is insufficient to assess the balance of benefits and harms of screening for hyperbilirubinemia to prevent CBE.

Hulzebos and associates (2011) examined the relationship between early postnatal dexamethasone (DXM) treatment and the severity of hyperbilirubinemia in extremely low birth weight (ELBW) preterm infants. In 54 ELBW preterm infants, TSB and phototherapy (PT) data during the first 10 days were evaluated retrospectively. These ELBW infants had participated in a randomized controlled trial of early DXM therapy that aimed to evaluate effects on chronic lung disease. Infants had been treated with DXM ($0.25$ mg/kg twice-daily at postnatal day 1 and 2) or with placebo (normal saline). Analysis was performed on an intention-to-treat basis. A total of 25 infants had been randomized into the DXM group; 29 into the placebo group. Mean TSB ($120 +/- 19$ μmol/L versus $123 +/- 28$ μmol/L, DXM versus placebo, respectively) and maximum TSB ($178 +/- 23$ μmol/L versus $176 +/- 48$, DXM versus placebo, respectively) concentrations were similar. Total serum bilirubin concentrations peaked 30 hours earlier in the DXM group ($p ≤ 0.05$). The need for PT as well as the duration of PT were similar in both groups. The authors concluded that early DXM treatment does not affect the severity of neonatal hyperbilirubinemia in ELBW preterm infants. These findings seem compatible with the concept that factors other than bilirubin conjugation capacity are important for the pathophysiology of neonatal jaundice in ELBW preterm infants.

It is also important to note that there are serious health risks associated with corticosteroid therapy. In a Cochrane review on early (less than 8 days) postnatal corticosteroid treatment for preventing chronic lung disease in preterm infants, Halliday et al (2010) concluded that the benefits of early postnatal corticosteroid treatment, especially DXM, may not out-weigh the known or potential adverse effects of this treatment. Although early corticosteroid treatment facilitates extubation and reduces the risk of chronic lung disease and patent ductus arteriosus, it causes short-term adverse effects including gastro
intestinal bleeding, intestinal perforation, hyperglycaemia, hypertension, hypertrophic cardiomyopathy and growth failure. Long-term follow-up studies reported an increased risk of abnormal neurological examination and cerebral palsy. However, the methodological quality of the studies determining long-term outcomes is limited in some cases; the surviving children have been assessed predominantly before school age, and no study has been sufficiently powered to detect important adverse long-term neurosensory outcomes. The authors concluded that there is a compelling need for the long-term follow-up and reporting of late outcomes, especially neurological and developmental outcomes, among surviving infants who participated in all randomized trials of early postnatal corticosteroid treatment.

In a Cochrane review, Gholitabar et al (2012) examined the safety and effectiveness of clofibrate in combination with phototherapy versus phototherapy alone in unconjugated neonatal hyperbilirubinemia. Randomized controlled trials were identified by searching MEDLINE (1950 to April 2012) before being translated for use in The Cochrane Library, EMBASE 1980 to April 2012 and CINAHL databases. All searches were re-run on April 2, 2012. These investigators included trials where neonates with hyperbilirubinemia received either clofibrate in combination with phototherapy or phototherapy alone or placebo in combination with phototherapy. Data were extracted and analyzed independently by 2 review authors (MG and HM). Treatment effects on the following outcomes were determined: mean change in bilirubin levels, mean duration of treatment with phototherapy, number of exchange transfusions needed, adverse effects of clofibrate, bilirubin encephalopathy and neonatal mortality. Study authors were contacted for additional information. Studies were analyzed for methodological quality in a “Risk of bias” table. A total of 15 studies (2 including preterm neonates and 13 including term neonates) were included in this review. All but 1 of the included studies were conducted in Iran. For preterm neonates, there was a significantly lower bilirubin level in the 100 mg/kg clofibrate group compared to the control group with a mean difference of -1.37 mg/dL (95% CI: -2.19 mg/dL to -0.55 mg/dL) (-23 µmol/L; 95% CI: -36 µmol/L to -9 µmol/L) after 48 hours. For the term neonates, there were significantly lower bilirubin levels in the clofibrate group compared to the control group after both 24 and 48 hours of treatment with a weighted mean difference of -2.14 mg/dL (95% CI: -2.53 mg/dL to -1.75 mg/dL) (-37 µmol/L; 95% CI: -43 µmol/L to -30 µmol/L) and -1.82 mg/dL (95% CI: -2.25 mg/dL to -1.38 mg/dL) (-31 µmol/L; 95% CI: -38 µmol/L to -24 µmol/L), respectively. There was a significantly lower duration of phototherapy in the clofibrate group compared to the control group for both preterm and term neonates with a weighted mean difference of -23.82 hours (95% CI: -30.46 hours to -17.18 hours) and -25.40 hours (95% CI: -28.94 hours to -21.86 hours), respectively. None of the studies reported on bilirubin encephalopathy rates, neonatal mortality rates, or the levels of parental or staff satisfactions with the interventions. The authors concluded that there are insufficient data from different countries on the use of clofibrate in combination with phototherapy for hyperbilirubinemia to make recommendations for practice. They stated that there is a need for larger trials to determine how effective clofibrate is in reducing the need for, and duration of, phototherapy in term and preterm infants with hyperbilirubinemia.

Watchko and Lin (2010) noted that the potential for genetic variation to modulate neonatal hyperbilirubinemia risk is increasingly being recognized. In particular, polymorphisms across 3 genes involved in bilirubin production and metabolism: (i) glucose-6-phosphate dehydrogenase (G6PD), (ii) uridine diphosphate glucuronosyl transferase 1A1 (UGT1A1), and (iii) solute carrier organic anion transporter polypeptide 1B1 (SLCO1B1)] may interact with each other and/or environmental contributors to produce significant hyperbilirubinemia. Variant gene co-expression including compound and synergistic heterozygosity enhances hyperbilirubinemia risk, contributing to the etiologic heterogeneity and complex nature of neonatal jaundice.

Liu et al (2013) examined if 3 variants (388 G>A, 521 T>C, and 463 C>A) of SLCO1B1 are associated with neonatal hyperbilirubinemia. The China National Knowledge Infrastructure and MEDLINE databases were searched. These researchers performed a systematic review with meta-analysis including genetic studies, which assessed the association between neonatal hyperbilirubinemia and 388 G>A, 521 T>C, and 463 C>A variants of SLCO1B1 between January of 1980 and December of 2012. Data selection and extraction were performed independently by 2 reviewers. A total of 10 articles were included in the study. The results revealed that SLCO1B1 388 G>A is associated with an increased risk of neonatal hyperbilirubinemia (odds ratio [OR], 1.39; 95% CI: 1.07 to 1.82) in Chinese neonates, but not in white, Thai, Latin American, or Malaysian neonates. The SLCO1B1 521 T>C mutation showed a low risk of neonatal hyperbilirubinemia in Chinese neonates, while no significant associations were found in Brazilian, white, Asian, Thai, and Malaysian neonates. There were no significant differences in SLCO1B1 463 C>A between the hyperbilirubinemia and the control group.
The authors concluded that the findings of this study demonstrated that the 388 G>A mutation of the SLCO1B1 gene is a risk factor for developing neonatal hyperbilirubinemia in Chinese neonates, but not in white, Thai, Brazilian, or Malaysian populations; the SLCO1B1 521 T>C mutation provides protection for neonatal hyperbilirubinemia in Chinese neonates, but not in white, Thai, Brazilian, or Malaysian populations.

Petersen and colleagues (2014) stated that extreme hyperbilirubinemia (plasma bilirubin greater than or equal to 24.5 mg/dL) is an important risk factor for severe bilirubin encephalopathy. Several risk factors for hyperbilirubinemia are known, but in a large number of patients, a causal factor is never established. UGT1A1 is the rate-limiting enzyme in bilirubin's metabolism. The genotype of Gilbert syndrome, the UGT1A1*28 allele, causes markedly reduced activity of this enzyme, but its association with neonatal hyperbilirubinemia is uncertain and its relationship with extreme hyperbilirubinemia has not been studied. These researchers examined whether the UGT1A1*28 allele is associated with extreme hyperbilirubinemia. The UGT1A1*28 allele was assessed in a case-control study of 231 white infants who had extreme hyperbilirubinemia in Denmark from 2000 to 2007 and 432 white controls. Cases were identified in the Danish Extreme Hyperbilirubinemia Database that covers the entire population. Genotypes were obtained through the Danish Neonatal Screening Biobank. Subgroup analysis was done for AB0 incompatible cases. No association was found between the UGT1A1*28 allele and extreme hyperbilirubinemia. With the common genotype as reference, the odds ratio of extreme hyperbilirubinemia was 0.87 (range of 0.68 to 1.13) for UGT1A1*28 heterozygotes and 0.77 (range of 0.46 to 1.27) for homozygotes. Also, no association was found for AB0 incompatible cases. The authors concluded that the UGT1A1*28 allele was not associated with risk for extreme hyperbilirubinemia in this study.

Travan et al (2014) examined if UGT1A1 promoter polymorphisms associated with Gilbert Syndrome (GS) occur with a greater frequency in neonates with severe hyperbilirubinemia. In a case-control study performed at a single hospital center in Italy, 70 subjects with severe hyperbilirubinemia (defined as bilirubin level greater than or equal to 20 mg/dL or 340 μmol/L) and 70 controls (bilirubin level less than 12 mg/dL or 210 μmol/L) were enrolled. Both case and control subjects were full term newborns. Polymerase chain reaction analysis on blood spot was performed to determine the frequency of UGTA1A1 promoter polymorphisms in cases and controls. No statistical difference in the prevalence of UGTA1A1 gene variants was found between cases and controls (p = 1). Thirteen infants homozygous for (TA)7 polymorphism associated with GS were in the case group (18.6 %) and 14 in the control group (20.0 %). A heterozygous group was also equally distributed between cases (44.3 %) and controls (42.9 %). No (TA)8 repeat was found in the 2 groups. The authors concluded that in this study population, GS polymorphism alone did not appear to play a major role in severe neonatal hyperbilirubinemia in neonates without signs of hemolysis.

An UpToDate review on “Evaluation of unconjugated hyperbilirubinemia in term and late preterm infants” (Wong and Bhutani, 2015) does not mention genotyping of SLCO1B1 and UGT1A1 as management tools.

CPT Codes / HCPCS Codes / ICD-9 Codes

CPT codes covered if selection criteria are met:

82247 Bilirubin; total
82248 direct
36450 Exchange transfusion, blood; newborn

HCPCS codes covered if selection criteria are met:

E0202 Phototherapy (bilirubin) light with photometer
S9098 Home visit, phototherapy services (e.g., Bili-lite), including equipment rental, nursing services, blood draw, supplies, and other services, per diem

HCPCS codes not covered for indications listed in the CPB:
J2560 Injection, phenobarbital sodium, up to 120 mg

ICD-9 codes covered if selection criteria are met:

773.0 Hemolytic disease due to Rh isoimmunization
773.1 Hemolytic disease due to ABO isoimmunization
773.2 Hemolytic disease due to other and unspecified isoimmunization
773.4 Kernicterus due to isoimmunization
774.0 - 774.7 Other prenatal jaundice

ICD-9 codes not covered for indications listed in the CPB:

656.20 - 656.23 Isoimmunization from other and unspecified blood-group incompatibility affecting management of mother [not covered for the use of antenatal phenobarbital in red cell isoimmunized pregnant women]

Other ICD-9 codes related to the CPB:

767.0 Subdural and cerebral hemorrhage
768.5 - 768.6 Birth asphyxia
768.7 Hypoxic-ischemic encephalopathy (HIE)
768.9 Unspecified birth asphyxia in liveborn infant
772.10 - 772.14 Intraventricular hemorrhage
775.6 Neonatal hypoglycemia
775.7 Late metabolic acidosis of newborn
799.01 - 799.02 Asphyxia and hypoxemia

The above policy is based on the following references:


60. Wong RJ, Bhutani VK. Evaluation of unconjugated hyperbilirubinemia in term and late preterm infants. UpToDate [online serial]. Waltham, MA: UpToDate; reviewed January 2015.