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Antenatal Hydronephrosis as a Predictor of Postnatal Outcome: A Meta-analysis

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ABSTRACT

OBJECTIVE. Antenatal hydronephrosis is diagnosed in 1% to 5% of all pregnancies; however, the antenatal and postnatal management of hydronephrosis varies widely. No previous studies define the risk of postnatal pathology in infants with antenatal hydronephrosis. Our objective was to review the current literature to determine whether the degree of antenatal hydronephrosis and related antenatal ultrasound findings are associated with postnatal outcome.

METHODS. We searched Medline (1966–2005), Embase (1991–2004), and the Cochrane Library databases for articles on antenatal hydronephrosis. We required studies to have subjects selected on the basis of documented measurements of antenatal hydronephrosis and followed to a postnatal diagnosis. We excluded case reports, review articles, and editorials. Two independent investigators extracted data.

RESULTS. We screened 1645 citations, of which 17 studies met inclusion criteria. We created a data set of 1308 subjects. The risk of any postnatal pathology per degree of antenatal hydronephrosis was 11.9% for mild, 45.1% for moderate, and 88.3% for severe. There was a significant increase in risk per increasing degree of hydronephrosis. The risk of vesicoureteral reflux was similar for all degrees of antenatal hydronephrosis.

CONCLUSIONS. The findings of this meta-analysis can potentially be used for prenatal counseling and may alter current postnatal management of children with antenatal hydronephrosis. Overall, children with any degree of antenatal hydronephrosis are at greater risk of postnatal pathology as compared with the normal population. Moderate and severe antenatal hydronephrosis have a significant risk of postnatal pathology, indicating that comprehensive postnatal diagnostic management should be performed. Mild antenatal hydronephrosis may carry a risk for postnatal pathology, but additional prospective studies are needed to determine the optimal management of these children. A well-defined prospective analysis is needed to further define the risk of pathology and the appropriate management protocols.
Antenatal hydronephrosis (ANH) affects ~1% to 5% of all pregnancies and is one of the most common birth defects. However, the reported clinical relevance of varying degrees of ANH is unclear. Although the use of prenatal ultrasound as a screening tool for birth defects has not been shown to improve perinatal outcome, more patients are undergoing prenatal counseling for the discovery of ANH. Patients diagnosed with ANH on routine ultrasound often undergo extensive prenatal imaging that may include serial ultrasound and MRI. In addition, patients may undergo postnatal examinations that may include a variable combination of serial renal ultrasound, voiding cystourethrogram (VCUG), diuretic renogram, intravenous pyelogram, and MRI urogram. Although current prenatal testing is mostly noninvasive, much of the postnatal assessment is invasive and exposes the child to radiation or anesthesia that may be unnecessary.

The efficacy and social health care costs of routine prenatal ultrasound as a screening tool for potential postnatal health risks remains controversial. The diagnosis of ANH may cause significant parental anxiety and physician uncertainty with regard to prenatal and postnatal management. Many variations in the definition and management of ANH exist in the literature and clinical practice, including method and frequency of in utero testing, radiographic documentation, classification, or postnatal management. To date, there are no comprehensive prospective studies that determine the risk of pathology with varying degrees of ANH or those aspects of ANH that predict postnatal diagnosis or kidney outcome. To address these questions, we performed a meta-analysis of all published case series of ANH to determine whether or not the degree of ANH is associated with the risk of postnatal pathology.

METHODS

Meta-analysis Search Strategy
In collaboration with a research librarian, we searched Medline (1966–2004), Embase (1991–2004), and the Cochrane Library databases to identify pertinent articles in English. We combined 10 terms for hydronephrosis (hydronephrosis, pelviectasis, pelvicaliectasis, pyelectasis, hydroureteronephrosis, renal pelvic dilation, anterior posterior diameter, oligohydramnios, calyceal dilation, and ureteral dilation) with 6 terms for prenatal (prenatal, newborn, antenatal, fetal, natural history, and ultrasound). We simultaneously searched reference lists of research articles, reviews, and texts to ensure that we acquired all of the relevant articles. We did not contact authors for original data and did not consider unpublished reports.

Inclusion and Exclusion Criteria
We reviewed all of the articles obtained from the literature search and only included studies that met the following ANH criteria: (1) diagnosed in utero by ultrasound; (2) reported anterior posterior diameter (APD) or a specific APD range; and (3) reported postnatal diagnosis of hydronephrosis. We included women of all races/ethnicities and ages. We excluded studies that (1) were case reports, editorials, or review articles, (2) lacked reported APD, (3) did not report postnatal follow-up or diagnosis, (4) only reported on patients with multisystem congenital malformations, coexisting chromosomal abnormality, history of fetal intervention, or fetal termination, or (5) selected subjects on the basis of postnatal diagnosis.

Data Extraction
Two investigators (R.S.L. and H.T.N.) independently screened the abstracts and the articles considered for inclusion and performed independent data extraction. Investigator consensus (R.S.L., H.T.N., D.D.K., and M.C.) reconciled any differences in data acquisition.

The degree of hydronephrosis classified by APD characterized the ANH data across the studies. Using the current literature on APD measurements, we developed 3 major classifications of ANH: mild ANH, moderate ANH, and severe ANH (Table 1). To accommodate reported APD ranges that crossed between major classifications, we defined 2 additional ANH classifications, mild-moderate ANH and moderate-severe ANH (Table 1).

In addition to APD measurements, we attempted to extract other ultrasound findings, such as renal calyceal dilation, hydroureteronephrosis, renal echogenicity, renal parenchymal thinning, bladder dilation, posterior urethral dilation, and amniotic fluid level. We also attempted to extract the gestational age at diagnosis, gender, laterality, and presence or absence of bilateral hydronephrosis.

We classified the postnatal diagnoses into 6 categories: (1) normal or transient hydronephrosis (resolved without intervention); (2) ureteropelvic junction obstruction (UPJ); (3) vesicoureteral reflux (VUR); (4) urethral obstruction, such as posterior urethral valves or urethral atresia; (5) ureteral obstruction, such as ureterocele, ectopic ureterocele, and obstructing megaloureter; and (6) others (multicystic dysplastic kidney.

<table>
<thead>
<tr>
<th>TABLE 1 Classification of ANH by APD</th>
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<tbody>
<tr>
<td>Classification</td>
</tr>
<tr>
<td>Mild</td>
</tr>
<tr>
<td>Mild/moderate</td>
</tr>
<tr>
<td>Moderate</td>
</tr>
<tr>
<td>Moderate/severe</td>
</tr>
<tr>
<td>Severe</td>
</tr>
</tbody>
</table>
prune belly syndrome, hydrometrocolpos, and bladder agenesis).

We considered patients with bilateral ANH as 1 subject. When possible, we based our analysis on the renal unit with the worse degree of ANH and its corresponding outcome. We excluded patients that were lost to follow-up, excluded by the original author in each study, or had indeterminate prenatal or postnatal data. We considered a patient as lost to follow-up if there was no postnatal follow-up or the postnatal diagnosis was unavailable. Indeterminate patients did not have extractable APD data or did not have a prenatal diagnosis of hydronephrosis.

Statistical Analysis
Logistic regression was used to estimate the risk of any postnatal pathology for different degrees of ANH. Robust SEs based on generalized estimating equations with a working independence correlation structure were used to account for correlated outcomes within each study. Similar methods were used to estimate the risks of specific pathologic diagnoses for different degrees of ANH. Tests for trend in the risks of postnatal pathologies with increasing degree of ANH were conducted using equally spaced scores in these logistic regression models.

RESULTS
We screened 1645 citations and found 17 case series that met inclusion and exclusion criteria. A total of 1678 subjects of 104 572 subjects screened (1.6% prevalence) had ANH (Table 2). The studies analyzed did not use the same ultrasound criteria or prenatal and postnatal imaging protocols to define, detect, and follow ANH (Table 3). We were unable to cluster the studies by prenatal or postnatal diagnostic protocols. Prenatal imaging protocols varied widely with regard to the timing of the initial and follow-up prenatal ultrasound and the number of follow-up prenatal ultrasounds. As for postnatal diagnostic imaging, 15 of 17 studies performed a renal ultrasound sometime within the first 7 days after birth. The use of further diagnostic studies, such as VCUG or functional studies (diuretic renogram or intravenous pyelogram), varied significantly between studies, and 2 studies did not document their postnatal protocol. The length of follow-up for each study was not available. We stratified the subjects using the ANH categories defined in Table 1 regardless of the classification of ANH by the study.

Of the 1678 patients with ANH, 246 were lost to follow-up, and 124 were excluded because of indeterminate prenatal data, leaving 1308 patients for analysis. Sixty-five patients had bilateral disease.22–25 Data for each renal unit in these 65 patients was not obtainable but was reported by the authors as a single subject.

Table 4 shows the number of patients in each category of ANH in each of the 17 studies and the percentage of patients in each study with any postnatal pathology. Only 4 studies had patients in each of the 3 major categories of ANH.9,24,26,27 Four studies had patients in mild and moderate-severe categories.4,23,28,29 One study solely contributed patients to the mild-moderate ANH classification.30 In Table 4, postnatal pathology is grouped into those who had any pathology versus those who had no pathology or transient postnatal hydronephrosis. Taking all of the patients with any degree of ANH, 36% had pathology discovered during postnatal management.

We determined the risk of any pathology (UPJ, VUR, posterior urethral valves, ureteral obstruction, and other) for each degree of ANH (Table 5). The overall risk

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### Table 2

<table>
<thead>
<tr>
<th>Source</th>
<th>Disposition of Subjects in Included Studies</th>
<th>No. Patients Screened</th>
<th>No. Patients With ANH per Study</th>
<th>No. Lost to Follow-up</th>
<th>No. Excluded</th>
<th>No. Included</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obido et al40</td>
<td>1999–2002</td>
<td>7416</td>
<td>115</td>
<td>49</td>
<td>0</td>
<td>66</td>
</tr>
<tr>
<td>Kapadia et al9</td>
<td>1996–1999</td>
<td>17 850</td>
<td>117</td>
<td>15</td>
<td>1</td>
<td>101</td>
</tr>
<tr>
<td>Ismaili et al30</td>
<td>1998–2000</td>
<td>5643</td>
<td>258</td>
<td>45</td>
<td>0</td>
<td>213</td>
</tr>
<tr>
<td>Sairam et al4</td>
<td>1994–1998</td>
<td>11465</td>
<td>268</td>
<td>38</td>
<td>3</td>
<td>227</td>
</tr>
<tr>
<td>Chowdhary et al58</td>
<td>1997–1998</td>
<td>6810</td>
<td>38</td>
<td>0</td>
<td>0</td>
<td>38</td>
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<tr>
<td>Fasolato et al24</td>
<td>1994–1995</td>
<td>1809</td>
<td>51</td>
<td>0</td>
<td>1</td>
<td>50</td>
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<tr>
<td>Langer et al26</td>
<td>1989–1991</td>
<td>2170</td>
<td>95</td>
<td>6</td>
<td>0</td>
<td>89</td>
</tr>
<tr>
<td>Adra et al10</td>
<td>1989–1993</td>
<td>84</td>
<td>84</td>
<td>16</td>
<td>0</td>
<td>68</td>
</tr>
<tr>
<td>Tam et al18</td>
<td>1989–1992</td>
<td>125</td>
<td>125</td>
<td>0</td>
<td>46</td>
<td>79</td>
</tr>
<tr>
<td>Lam et al41</td>
<td>1987–1990</td>
<td>16 991</td>
<td>60</td>
<td>0</td>
<td>23</td>
<td>37</td>
</tr>
<tr>
<td>Wilhelm et al22</td>
<td>5 y</td>
<td>78</td>
<td>78</td>
<td>0</td>
<td>13</td>
<td>65</td>
</tr>
<tr>
<td>Rosendahl14</td>
<td>1983–1987</td>
<td>4856</td>
<td>27</td>
<td>0</td>
<td>5</td>
<td>22</td>
</tr>
<tr>
<td>Kent et al27</td>
<td>1991–1997</td>
<td>14 700</td>
<td>40</td>
<td>0</td>
<td>3</td>
<td>37</td>
</tr>
<tr>
<td>Arger et al30</td>
<td>2 y</td>
<td>3530</td>
<td>39</td>
<td>8</td>
<td>9</td>
<td>22</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>104 572</td>
<td>1678</td>
<td>246</td>
<td>124</td>
<td>1308</td>
</tr>
</tbody>
</table>
of any pathology was 11.9% (95% confidence interval [CI]: 4.5–28.0) for mild ANH, 45.1% (95% CI: 25.3–66.6) for moderate ANH, and 88.3% (95% CI: 53.7–98.0) for severe ANH. The risk of postnatal pathology rose significantly with increasing degree of ANH (P < .001).

The risks and 95% CIs for specific pathologic diagnoses for each degree of ANH are also shown in Table 5. For all of the pathologic diagnosis other than VUR, there was a significant increase in risk with increasing degree of ANH. There was no evidence of a trend in the risk of VUR across different degrees of ANH (P = .10).

We attempted to extract other antenatal ultrasound findings, such as renal calyceal dilation, hydrourereteronephrosis, renal echogenicity, renal parenchymal thinning, bladder dilation, posterior urethral dilation, amni-
Table 5: Risk of Pathology by Degree of ANH

<table>
<thead>
<tr>
<th>Postnatal Pathology, % (95% CI)</th>
<th>Degree of ANH</th>
</tr>
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</table>
|                                | Mild (N = 587) | Mild-Moderate (N = 213) | Moderate (N = 235) | Moderate-Severe (N = 179) | Severe (N = 94) | Trend P
| Any Pathology                  | 11.9 (4.5–28.0) | 39.0 (32.6–45.7) | 45.1 (25.3–66.6) | 72.1 (47.6–88.0) | 88.3 (53.7–98.0) | <.001
| UPJ                            | 4.9 (2.0–11.9) | 13.6 (9.6–18.9) | 17.0 (7.6–33.9) | 36.9 (17.9–61.0) | 54.3 (21.7–83.6) | <.001
| VUR                            | 4.4 (1.5–12.1) | 10.8 (7.3–15.7) | 14.0 (7.1–25.9) | 123.8 (4.7–15.0) | 8.5 (4.7–15.0) | .10
| PUV                            | 0.2 (0.0–1.4) | 0.9 (0.2–3.7) | 0.9 (0.2–2.9) | 6.7 (2.5–16.0) | 5.3 (1.2–21.0) | <.001
| Ureteral obstruction            | 1.2 (0.2–8.0) | 11.7 (8.1–16.8) | 9.8 (6.3–14.9) | 10.6 (7.4–15.0) | 5.3 (1.4–18.2) | .025
| Othera                          | 1.2 (0.3–4.0) | 1.9 (0.7–4.9) | 3.4 (0.5–19.4) | 5.5 (3.0–10.2) | 14.9 (3.6–44.9) | .002

PUV indicates posterior urethral valve.

a Pointwise 95% CIs were estimated by logistic regression with robust SEs based on generalized estimating equations with a working independence correlation structure to adjust for clustering by study for all degrees of ANH except mild-moderate. Because only 1 study had subjects with mild-moderate ANH, the pointwise 95% CIs had to be estimated using logistic regression with unadjusted SEs.

b Testing for trend in risks with increasing degree of ANH using logistic regression with robust SEs based on generalized estimating equations with a working independence correlation structure.

c Includes prune belly syndrome, VATER syndrome, solitary kidney, renal mass, and unclassified.

otonic fluid level, gestational age at diagnosis, gender, laterality, and presence or absence of bilateral hydronephrosas, and correlate these to outcome. However, because of the variability in reporting and lack of data, we were unable to perform an appropriate analysis.

Discussion

The precision and value of antenatal ultrasound as a screening modality remains controversial. Our analysis of 1308 subjects with varying degrees of ANH suggests that the risk of a pathologic postnatal outcome of ANH may be quantified by the measurement of APD. For instance, there are only 3 studies among those reporting 1308 subjects with varying degrees of ANH. In our analysis, the overall risk of VUR in the ANH population (8.6%) is quantifiably higher than the general population incidence (1%).

As expected, our meta-analysis confirms that severe ANH carries a significant risk of a postnatal pathologic outcome (88.3%). The primary debate centers on the postnatal outcome of mild or moderate ANH. We demonstrate a significant risk of pathology in both of these categories (11.9% and 45.1%, respectively), which potentially indicates that more thorough postnatal diagnostic management should be considered when confronted with a child with this condition.

Clinicians often use other aspects of the prenatal ultrasound, such as renal calyceal dilation, renal echogenicity, renal parenchymal thinning, hydrourerateronephrosis, bladder dilation, posterior urethral dilation, amniotic fluid level, gender, gestational age, and laterality to help provide an assessment of risk for postnatal pathology and to plan postnatal management. Although these additional ultrasound parameters are reported as potential predictors of postnatal pathology, the articles published to date have not provided the appropriate detail to determine what additional prenatal or postnatal tests should be conducted.

For instance, there are only 3 studies among those considered that reported results on amniotic fluid levels. Of these studies, only 2 had subjects with oligohydramnios. Because of the sparseness of the data and the fact that all patients with oligohydramnios had postnatal pathology, the same techniques used for estimating CIs for the risk by degree of ANH could not be applied in the setting of oligohydramnios. Similar lapses in the data are seen for the other parameters considered. Undoubtedly, these additional parameters play a crucial role in determining the risk of postnatal pathology. Further rigorous prospective analysis is needed to determine the prognostic value of these prenatal ultrasound findings.

In addition, there is a lack of conformity in the definition of ANH. A survey study of European pediatric urologists and nephrologists demonstrates a lack of consensus in defining ANH and in management. The literature we reviewed demonstrated no conformity in the definition of ANH. We were unable to apply current grading standards, such as the Society of Fetal Urology grading of hydronephrosis, to the reported studies or analysis, because the studies did not report all of the required data for this grading system. As a result, it was necessary to create a rigorous but arbitrary definition of ANH based on the 1 factor most consistently reported: APD. Nevertheless, based on this review, we do not feel that the grading of ANH will be based solely on APD.

Likewise, there are major variations in the method of prenatal screening and postnatal diagnostic follow-up. Our analysis demonstrated significant variability in the timing of the initial and follow-up ultrasound and in the number of follow-up prenatal ultrasounds (Table 3). Reports demonstrate that the degree of ANH can change in utero and that the amount of change may be a significant indicator of postnatal outcome. In addition, prenatal ultrasound performed in the third trimester as opposed to earlier in the pregnancy may be more predictive of postnatal outcome. However, because of the inconsistent reporting and variable prenatal protocols, we were unable to determine whether or not timing of prenatal ultrasound or serial ultrasound influenced outcome or provided additional prognostic information. The
incongruity of the screening protocols reviewed may introduce substantial heterogeneity into the study and challenges the accuracy of APD as the sole indicator of postnatal outcome.

Similarly, standardized protocols for postnatal management of ANH and independent standardized outcome measures of ANH do not exist. Table 3 demonstrates a significant variation in the postnatal diagnostic testing protocol in each study, which may bias the outcome toward one diagnosis over another. Most importantly, the definition by each protocol of significant postnatal hydronephrosis varies, which is a requirement in many protocols for further postnatal diagnostic investigation. Patients who are classified as normal or transient after the initial postnatal ultrasound and do not undergo further diagnostic investigation may actually have postnatal pathology. Many studies show that a negative postnatal ultrasound is not a reliable indicator or predictor for the exclusion of VUR.\(^4\)\(^4\)\(^5\)\(^6\)\(^7\) The variability in postnatal protocols may have introduced a selectivity bias toward a diagnosis of normal/transient as opposed to VUR, particularly if the patients are not adequately studied or followed in the postnatal period. Similarly, some postnatal protocols are selective in their performance of a functional test to determine whether the postnatal hydronephrosis is secondary to UPJ obstruction. The ability to definitively diagnose UPJ obstruction is debated on many levels, including the timing and method of testing, to the functional definition of obstruction.\(^4\)\(^6\)\(^8\)\(^9\)\(^1\)\(^0\)

These 2 variations in postnatal protocols may result in underdiagnosing pathology and may, therefore, have underestimated the risks of pathology downward. At the same time the postnatal diagnosis may not be the most important outcome measure, because some of the pathologic diagnoses may resolve without intervention or damage to the health of the child. Rather, the need for surgical intervention or the onset of renal damage may be more appropriate outcome measures. However, these outcome measures have their own inherent subjective biases.

In addition, the decision to place a child on prophylactic antibiotics because of VUR risk before postnatal diagnostic imaging is controversial. The articles considered in this meta-analysis did not address this controversy. To date, there were no large comprehensive prospective studies that determine the risk of VUR with varying degrees of ANH. Numerous small series demonstrated that children with ANH have an increased risk of VUR as compared with the general population.\(^4\)\(^8\)\(^2\)\(^3\)

However, although the data were limited, children with ANH and VUR seemingly have a more benign course with a higher resolution rate of VUR as compared with children discovered to have VUR after a febrile infection.\(^5\)\(^4\)\(^5\)\(^6\) The decision to place a child with ANH on prophylactic antibiotics remains controversial, and we were unable to clarify this controversy through this analysis.

We could not estimate the health care burden of screening patients with varying degrees of ANH. The articles published to date do not address the cost-effectiveness of serial prenatal ultrasound or extensive postnatal diagnostic evaluations and its relationship to postnatal outcome for different degrees of ANH. Further prospective work in this area is clearly needed. Ideally, a multicenter prospective study with strict prenatal and postnatal protocols would be needed to define the prognostic ability of ANH. These studies need to determine which parameters of a prenatal ultrasound are predictive, the timing and number of prenatal ultrasounds needed, the requisite postnatal diagnostic imaging protocol, and the overall cost-effectiveness of the investigation.

Regardless, our analysis demonstrates a significant increased risk of postnatal pathology for any grade of ANH as compared with the normal population. In addition, when categorizing ANH by a measurable parameter, APD, a significant increase in the risk for all pathologies exists with each increasing grade of ANH, except for VUR. Our analysis provides realistic estimates of risk for each degree of ANH that may be of value in directing the prenatal and postnatal management of these patients.

Despite the issues discussed earlier, this analysis improves our ability to counsel families by providing them with realistic estimates of postnatal pathology, which may help decrease parental anxiety about ANH. This analysis was unable to determine whether the diagnosis of ANH or associated findings of ANH would lead to poor child or kidney-specific postnatal or long-term outcome. In addition, we were unable to determine whether more antenatal imaging was helpful. The study was unable to provide further insight into these aspects of prenatal counseling. Although no consensus for prenatal and postnatal follow-up exists, on discovery of ANH we recommend that during the prenatal period patients be followed by a center that specializes in maternal fetal medicine and prenatal imaging and undergo multidisciplinary prenatal counseling. Children with bilateral severe ANH or a solitary kidney with any grade of ANH should undergo a postnatal ultrasound before discharge from the hospital. Children with all other grades of ANH should undergo a postnatal ultrasound within the first month of life. Children with persistent moderate to severe ANH should undergo a VCUG and a functional study as indicated. Children with mild ANH that persists postnatally or children with resolved ANH should be considered for further diagnostic imaging on a case-by-case basis until appropriate guidelines have been determined by a rigorous prospective study.
CONCLUSIONS

Children with any degree of ANH are at greater risk of postnatal pathology as compared with the normal population. Moderate and severe ANH have a considerable risk of pathology, indicating that comprehensive postnatal diagnostic management should be performed. Mild ANH may carry a risk for postnatal pathology, but further prospective studies are needed to determine the optimal management of these children. To further define the risk of pathology and the appropriate management protocols for different degrees of ANH, a well-defined prospective analysis of the relationship between the parameters of prenatal ultrasound needs to be performed.

ACKNOWLEDGMENTS

We thank Alison Clap for her assistance with the literature search and Dr David Wypij for his assistance with study design and statistics. We also thank Drs Stuart Bauer, Joseph Borer, Bartley Cilento, David Diamond, Carlos Estrada, Craig Peters, and Alan Retik for their insightful comments, guidance, and suggestions.

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