OBSTETRICS & GYNECOLOGY

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- Comments from the reviewers and editors (email to author requesting revisions)
- Response from the author (cover letter submitted with revised manuscript)*

**The corresponding author has opted to make this information publicly available.*

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Date: Aug 13, 2021
To: "Huda Al-Kouatly" [redacted]
From: "The Green Journal" em@greenjournal.org
Subject: Your Submission ONG-21-1509

RE: Manuscript Number ONG-21-1509

The Etiology and Outcome of Isolated Fetal Ascites: A Systematic Review

Dear Dr. Al-Kouatly:

Your manuscript has been reviewed by the Editorial Board and by special expert referees. Although it is judged not acceptable for publication in Obstetrics & Gynecology in its present form, we would be willing to give further consideration to a revised version.

If you wish to consider revising your manuscript, you will first need to study carefully the enclosed reports submitted by the referees and editors. Each point raised requires a response, by either revising your manuscript or making a clear and convincing argument as to why no revision is needed. To facilitate our review, we prefer that the cover letter include the comments made by the reviewers and the editor followed by your response. The revised manuscript should indicate the position of all changes made. We suggest that you use the "track changes" feature in your word processing software to do so (rather than strikethrough or underline formatting).

Please be sure to address the Editor comments (see "EDITOR COMMENTS" below) in your point-by-point response.

Your paper will be maintained in active status for 21 days from the date of this letter. If we have not heard from you by Sep 03, 2021, we will assume you wish to withdraw the manuscript from further consideration.

REVIEWER COMMENTS:

Reviewer #1:

Well done well written study
results section: it would be helpful to breakdown the relative contributions of each of the 11 studies to the 315 case total in a table listing each study the year, and the N for each
Conclusion: not sure how your last sentence on genetics is related to the text or is this just a side comment

Reviewer #2:

This is a systematic review of etiologies, progression, and outcomes of isolated fetal ascites. Isolated fetal ascites does represent a clinical conundrum for appropriate counseling, and a systematic review of this nature can be useful. This article would be strengthened by the addition of some comparison analyses and some improved clarity of results reporting.

1. Introduction, lines 100 - 102: 92% underlying etiology identification rate seems high. That may be the highest rate in certain case series, but presenting it this way overestimates. Would recommend presenting a range instead.
2. Methodology is standard and appropriate.
3. Study selection, lines 147 - 151: could you expound how you defined these classifications? Specifically, chylous ascites can be due to genetic etiologies and thus these categories overlap.
4. Methods: you exclude any case reports or case series with fewer than five cases. This essentially excludes a number of etiologies which may be more rare or more morbid. It would be worthwhile to have a supplemental table of the ideologies of excluded cases in order to be fully comprehensive regarding possible ideologies of isolated fetal ascites. For example, gestational alloimmune liver disease is an important etiology with a high recurrence risk that is not mentioned here.
5. Results, line 176 and on: the latest data is presented as a little bit confusing in that the over all frequency of etiology includes, I believe, data from all 11 studies; whereas, the individual etiology data is limited to the individual patient data available from four studies. This should be emphasized. Moreover, lines 176 - 254 essentially recapitulate the data provided in table 2. The data is easier to interpret in table format and is a bit tiring to read as currently written. It should

be summarized rather than explicitly stated in its entirety.
6. Results: I realize you are limited by your available n. However, I think it would be possible to compare incidence of perinatal death and those with and without progression to hydrops. I suspect it would be statistically different and would be useful to know clinically for counseling.
7. Results, line 179: Potter syndrome is a term for a constellation of findings due to a variety of etiologies rather than an etiology and of itself. Based on the supplemental table, I believe this underlying etiology was bilateral dysplastic kidneys. It would be preferable to report the true underlying etiology.
8. Results: a figure representing progression to hydrops and perinatal mortality by etiology group would be useful.
9. Discussion, lines 319 - 320: the hiring to termination of pregnancy almost certainly leads to overestimation of the survival rate, as in general people are more likely to terminate more grave cases. I would be more clear with the admission of this limitation is it is crucial to counseling. I've seen some similar articles report a survival frequency with the denominator of both all pregnancies including terminations and also excluding terminations.

Reviewer #3:

This systematic review is a good attempt at compiling all the studies regarding the etiologies and outcomes in fetal ascites This might be useful in prognostication and counselling in fetuses with this condition
182-260
The termination rates seem to be highly skewed with the genitourinary and storage disorders showing high rates closely followed by genetic and structural disorders
Any criteria for termination or was it patient choice?
276-277
As in all cases of fetal anomalies it is difficult to estimate survival since the termination rates are high
Table 1
The study by Dreux at al(2015) shows that a number of fetuses were terminated from 27- 30 weeks(GUT),31-34weeks(GIT). Fetuses with advanced gestational age tend to be born alive and survive with minimal support unless they have a lethal anomaly.The legality of termination also varies from country to country.The query is whether these babies were liveborn or was feticide carried out prior to termination?

STATISTICS EDITOR COMMENTS:

Lines 171-262 and Table 2: Need to round all %s to nearest integer % and include 95% CIs for each %. The TOP and unknown entries should also have %s and CIs included.

There should be a Table comparing stats of survival by etiology for all comparisons made in Results. The Table should include n/N counts, %s and 95% CIs, along with stats tests. Of note, the difference between cohorts > 24 wks and < 24 wks needs to define which subset those with GA = 24 wks were assigned and the stats result is NS and likely also a reflection of selective loss and TOP rates.

EDITORIAL OFFICE COMMENTS:

1. The Editors of Obstetrics & Gynecology have increased transparency around its peer-review process, in line with efforts to do so in international biomedical peer review publishing. If your article is accepted, we will be posting this revision letter as supplemental digital content to the published article online. Additionally, unless you choose to opt out, we will also be including your point-by-point response to the revision letter. If you opt out of including your response, only the revision letter will be posted. Please reply to this letter with one of two responses:

A. OPT-IN: Yes, please publish my point-by-point response letter.
B. OPT-OUT: No, please do not publish my point-by-point response letter.

2. When you submit your revised manuscript, please make the following edits to ensure your submission contains the required information that was previously omitted for the initial double-blind peer review:

* Include your title page information in the main manuscript file. The title page should appear as the first page of the document. Add any previously omitted Acknowledgements (ie, meeting presentations, preprint DOIs, assistance from non-byline authors).
* Funding information (ie, grant numbers or industry support statements) should be disclosed on the title page and in the body text. For industry-sponsored studies, the Role of the Funding Source section should be included in the body text of the manuscript.
* Include clinical trial registration numbers, PROSPERO registration numbers, or URLs at the end of the abstract (if applicable).
* Name the IRB or Ethics Committee institution in the Methods section (if applicable).
* Add any information about the specific location of the study (ie, city, state, or country), if necessary for context.

3. Obstetrics & Gynecology uses an "electronic Copyright Transfer Agreement" (eCTA), which must be completed by all authors. When you uploaded your manuscript, each co-author received an email with the subject, "Please verify your authorship for a submission to Obstetrics & Gynecology." Please check with your coauthors to confirm that they received and completed this form, and that the disclosures listed in their eCTA are included on the manuscript's title page.

4. Standard obstetric and gynecology data definitions have been developed through the reVITALize initiative, which was convened by the American College of Obstetricians and Gynecologists and the members of the Women's Health Registry Alliance. Obstetrics & Gynecology has adopted the use of the reVITALize definitions. Please access the obstetric data definitions at https://www.acog.org/practice-management/health-it-and-clinical-informatics/revitalize-obstetrics-data-definitions and the gynecology data definitions at https://www.acog.org/practice-management/health-it-and-clinical-informatics/revitalize-gynecology-data-definitions. If use of the reVITALize definitions is problematic, please discuss this in your point-by-point response to this letter.

5. Because of space limitations, it is important that your revised manuscript adhere to the following length restrictions by manuscript type: Review articles should not exceed 6,250 words. Stated word limits include the title page, précis, abstract, text, tables, boxes, and figure legends, but exclude references.

6. Specific rules govern the use of acknowledgments in the journal. Please note the following guidelines:

* All financial support of the study must be acknowledged.
* Any and all manuscript preparation assistance, including but not limited to topic development, data collection, analysis, writing, or editorial assistance, must be disclosed in the acknowledgments. Such acknowledgments must identify the entities that provided and paid for this assistance, whether directly or indirectly.
* All persons who contributed to the work reported in the manuscript, but not sufficiently to be authors, must be acknowledged. Written permission must be obtained from all individuals named in the acknowledgments, as readers may infer their endorsement of the data and conclusions. Please note that your response in the journal's electronic author form verifies that permission has been obtained from all named persons.
* If all or part of the paper was presented at the Annual Clinical and Scientific Meeting of the American College of Obstetricians and Gynecologists or at any other organizational meeting, that presentation should be noted (include the exact dates and location of the meeting).
* If your manuscript was uploaded to a preprint server prior to submitting your manuscript to Obstetrics & Gynecology, add the following statement to your title page: "Before submission to Obstetrics & Gynecology, this article was posted to a preprint server at: [URL]."

7. The most common deficiency in revised manuscripts involves the abstract. Be sure there are no inconsistencies between the Abstract and the manuscript, and that the Abstract has a clear conclusion statement based on the results found in the paper. Make sure that the abstract does not contain information that does not appear in the body text. If you submit a revision, please check the abstract carefully.

In addition, the abstract length should follow journal guidelines. The word limit for Original Research articles is 300 words;

Reviews is 300 words; Case Reports is 125 words; Current Commentary articles is 250 words; Executive Summaries, Consensus Statements, and Guidelines are 250 words; Clinical Practice and Quality is 300 words; Procedures and Instruments is 200 words. Please provide a word count.

8. Only standard abbreviations and acronyms are allowed. A selected list is available online at http://edmgr.ovid.com /ong/accounts/abbreviations.pdf. Abbreviations and acronyms cannot be used in the title or précis. Abbreviations and acronyms must be spelled out the first time they are used in the abstract and again in the body of the manuscript.

9. The journal does not use the virgule symbol (/) in sentences with words. Please rephrase your text to avoid using "and/or," or similar constructions throughout the text. You may retain this symbol if you are using it to express data or a measurement.

10. In your Abstract, manuscript Results sections, and tables, the preferred citation should be in terms of an effect size, such as odds ratio or relative risk or the mean difference of a variable between two groups, expressed with appropriate confidence intervals. When such syntax is used, the P value has only secondary importance and often can be omitted or noted as footnotes in a Table format. Putting the results in the form of an effect size makes the result of the statistical test more clinically relevant and gives better context than citing P values alone.

If appropriate, please include number needed to treat for benefits (NNTb) or harm (NNTh). When comparing two procedures, please express the outcome of the comparison in U.S. dollar amounts.

Please standardize the presentation of your data throughout the manuscript submission. For P values, do not exceed three decimal places (for example, "P = .001"). For percentages, do not exceed one decimal place (for example, 11.1%").

11. Please review the journal's Table Checklist to make sure that your tables conform to journal style. The Table Checklist is available online here: http://edmgr.ovid.com/ong/accounts/table_checklist.pdf.

12. Please review examples of our current reference style at http://ong.editorialmanager.com (click on the Home button in the Menu bar and then "Reference Formatting Instructions" document under "Files and Resources). Include the digital object identifier (DOI) with any journal article references and an accessed date with website references. Unpublished data, in-press items, personal communications, letters to the editor, theses, package inserts, submissions, meeting presentations, and abstracts may be included in the text but not in the reference list.

In addition, the American College of Obstetricians and Gynecologists' (ACOG) documents are frequently updated. These documents may be withdrawn and replaced with newer, revised versions. If you cite ACOG documents in your manuscript, be sure the references you are citing are still current and available. Check the Clinical Guidance page at https://www.acog.org/clinical (click on "Clinical Guidance" at the top). If the reference is still available on the site and isn't listed as "Withdrawn," it's still a current document.

If the reference you are citing has been updated and replaced by a newer version, please ensure that the new version supports whatever statement you are making in your manuscript and then update your reference list accordingly (exceptions could include manuscripts that address items of historical interest). If the reference you are citing has been withdrawn with no clear replacement, please contact the editorial office for assistance (obgyn@greenjournal.org). In most cases, if an ACOG document has been withdrawn, it should not be referenced in your manuscript.

13. Figure 1: Please upload as a figure file on Editorial Manager.

When you submit your revision, art saved in a digital format should accompany it. If your figure was created in Microsoft Word, Microsoft Excel, or Microsoft PowerPoint formats, please submit your original source file. Image files should not be

copied and pasted into Microsoft Word or Microsoft PowerPoint.

When you submit your revision, art saved in a digital format should accompany it. Please upload each figure as a separate file to Editorial Manager (do not embed the figure in your manuscript file).

If the figures were created using a statistical program (eg, STATA, SPSS, SAS), please submit PDF or EPS files generated directly from the statistical program.

Figures should be saved as high-resolution TIFF files. The minimum requirements for resolution are 300 dpi for color or black and white photographs, and 600 dpi for images containing a photograph with text labeling or thin lines.

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If you choose open access, you will receive an Open Access Publication Charge letter from the Journal's Publisher, Wolters Kluwer, and instructions on how to submit any open access charges. The email will be from publicationservices@copyright.com with the subject line, "Please Submit Your Open Access Article Publication Charge(s)." Please complete payment of the Open Access charges within 48 hours of receipt.

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If you choose to revise your manuscript, please submit your revision through Editorial Manager at http://ong.editorialmanager.com. Your manuscript should be uploaded as a Microsoft Word document. Your revision's cover letter should include the following:

* A confirmation that you have read the Instructions for Authors (http://edmgr.ovid.com/ong/accounts/authors.pdf), and
* A point-by-point response to each of the received comments in this letter. Do not omit your responses to the Editorial Office or Editors' comments.

If you submit a revision, we will assume that it has been developed in consultation with your co-authors and that each author has given approval to the final form of the revision.

Again, your paper will be maintained in active status for 21 days from the date of this letter. If we have not heard from you by Sep 03, 2021, we will assume you wish to withdraw the manuscript from further consideration.

Sincerely,

Torri D. Metz, MD
Associate Editor, Obstetrics

2020 IMPACT FACTOR: 7.661
2020 IMPACT FACTOR RANKING: 3rd out of 83 ob/gyn journals

8/14/2021

Dear Editors,

Thank you very much for your consideration of our paper “The Etiology and Outcome of Isolated Fetal Ascites: A Systematic Review”. We appreciate the time and comments from each reviewer. Please review our responses to each comment below and let us know if you need us to do any further changes. All changes made to the manuscript are tracked. Thank you for the opportunity to improve our paper.

Sincerely,
Huda Al-Kouatly, MD

Reviewer 1

1) Well done well written study. Results section: it would be helpful to breakdown the relative contributions of each of the 11 studies to the 315 case total in a table listing each study the year, and the N for each.

Response: Thank you for your feedback. We have now included the recommended table in the updated manuscript as Table 2. The previous Table 2 in the manuscript has been reassigned as Table 3. We added the following to the result section (line 229-230): “The contribution of each of the 11 studies to the 315 case total is depicted in Table 2.”

Table 2- Contribution of isolated fetal ascites cases from each study in our review

Author (Year)	Cases of isolated fetal ascites
Baccega et al. (2016)	38
Boutall et al. (2011)	24
Catania et al. (2017)	51
Dreux et al. (2015)	70
El Bishry et al. (2008)	11
Favre et al. (2004)	56
Mir et al. (1987)	5
Nose et al. (2011)	12
Schmider et al. (2003)	26
Shweni et al. (1984)	4
Zelop et al. (1994)	18

Total	315

2) Conclusion: not sure how your last sentence on genetics is related to the text or is this just a side comment.

Response: Thank you for your feedback. We replaced this sentence with (line 542-526): "Isolated fetal ascites rarely progresses to fetal hydrops and thus etiological evaluations of isolated fetal ascites should be considered diagnostically distinct from fetal hydrops."

Reviewer 2

1) Introduction, lines 100 - 102: 92% underlying etiology identification rate seems high. That may be the highest rate in certain case series but presenting it this way overestimates. Would recommend presenting a range instead.

Response: Thank you for your feedback and helpful suggestion, we have amended our introduction to now include a range, as suggested. The previous introduction stated "Most often, fetal ascites is caused by an underlying pathological process that can be identified in up to 92% of cases" (line 134-136). The updated introduction now states, (line 147-148) "Most often, fetal ascites is caused by an underlying pathological process that can be identified in 25-92% of cases".

2) Study selection, lines 147 - 151: could you expound how you defined these classifications? Specifically, chylous ascites can be due to genetic etiologies and thus these categories overlap.

Response: We categorized cases according to how the authors in the paper and/or their shared original data classified the etiology of the ascites. When possible, we used the most specific etiology that could be applied. In cases where it was uncertain which category to use, we had a discussion amongst several authors to determine the appropriate category. All cases classified as chylous ascites by the authors are cases that did not have a known genetic diagnosis. We do acknowledge that there is overlap between categories for some cases such as patients with a genetic diagnosis who also had a known structural anomaly.
Therefore, for more clarification, we have added to the method section the following: (line 197-200) "The etiology of ascites was categorized according to how the original authors in the paper and/or their shared original data classified it. In cases where it was uncertain which category to use, we had a discussion among authors (H.A.K, R.H., S.B.) to determine the appropriate category."

3) Methods: you exclude any case reports or case series with fewer than five cases. This essentially excludes a number of etiologies which may be more rare or more morbid. It would be worthwhile to have a supplemental table of the ideologies of excluded cases in order to be fully comprehensive regarding possible ideologies of isolated fetal ascites. For example, gestational alloimmune liver disease is an important etiology with a high recurrence risk that is not mentioned here.

Response: We appreciate your feedback. We have added a supplemental table that includes the etiology of all the excluded cases of isolated fetal ascitis. This compromised 182 publications with 169 being a single case report. Since this was 14 pages long we added it at the end of this response document. Thus we added the following to the result section: (line 439-441) "Supplemental Table 11 depicts all the case reports and case series that were excluded in our study with < 5 cases of isolated fetal ascites. We organized this table according to the etiology of the isolated fetal ascites." We added the following to the discussion: (line 508-513) "In our review, we excluded case series with <5 cases. This cutoff was selected to avoid publication bias where rare etiologies are over represented in the literature, particularly as case reports. This does lead to an acknowledged limitation of our review as there are rare diseases such as gestational alloimmune liver disease (GALD) that are known to result in isolated fetal ascites which did not appear in any of the cases series which met our inclusion criteria". We also added the following reference to our manuscript: Sciard C, Collardeau-Frachon S, Atallah A, et al. Prenatal imaging features suggestive of liver gestational allo immune disease. J Gynecol Obstet Hum Reprod. 2019 Jan;48(1):61-64.

4) Results, line 176 and on: the latest data is presented as a little bit confusing in that the overall frequency of etiology includes, I believe, data from all 11 studies; whereas, the individual etiology data is limited to the individual patient data available from four studies. This should be emphasized. Moreover, lines 176 - 254 essentially recapitulate the data provided in table 2. The data is easier to interpret in table format and is a bit tiring to read as currently written. It should be summarized rather than explicitly stated in its entirety.

Response: Thank you for your comment. We stated in line 184-185 of the manuscript, original data was requested from 8 authors and four authors shared their data with us. One author did not have access to their data and the remaining three authors did not respond. The other three authors had complete data available from their published papers. Therefore, we had complete data for 7/11 studies or 75% of cases. We added to the results section: (line 232-233) "Three authors had complete data available from their published papers. Therefore, we had complete data for 7/11 studies or 75% of cases."
We have 10 Supplemental tables for each category of etiology. We summarized the results of all of these in Table 2 in the original manuscript. We tried to synthesize the results section in a way that would be easy for a busy Maternal-Fetal Medicine provider to use when they see a patient with isolated fetal ascites due to a particular etiology. The result section may be lengthy due to numerous etiologies of isolated fetal ascites. We have reorganized table 2 (now Table 3 in the current version) after the comments from the stats editor.

5) I realize you are limited by your available n. However, I think it would be possible to compare incidence of perinatal death and those with and without progression to hydrops. I suspect it would be statistically different and would be useful to know clinically for counseling.

Response: Thank you for the feedback. The live birth rate amongst continuing pregnancies in hydrops cases was 4/13 (31%) and the live birth rate amongst continuing pregnancies without hydrops was 174/242 (72%)%, p=0.0009.
We added the following to the results: (line 398-400) The live birth rate amongst continuing pregnancies in hydrops cases was significantly less than the live birth rate amongst continuing pregnancies without hydrops (4/13, 31% vs 174/242, 72%, p=0.0009).

6) Results, line 179: Potter syndrome is a term for a constellation of findings due to a variety of etiologies rather than an etiology and of itself. Based on the supplemental table, I believe this underlying etiology was bilateral dysplastic kidneys. It would be preferable to report the true underlying etiology.

Response: We appreciate and agree with the reviewer that Potter syndrome is not an etiology by itself. We replaced all categories of Potter syndrome with the etiology: one was bilateral dysplastic kidneys and three were polycystic kidneys based on the ultrasound findings from the authors in supplemental table 1. We also changed this in the results section: (Line 238) This included posterior urethral valves (PUV) (56%), urogenital sinus anomaly (6.7%), lower urinary tract obstruction (5.3%), polycystic/dysplastic kidneys (6%) vesicoureteral reflux (2.7%), and ureteropelvic junction obstruction (2.7%).

7) Results: a figure representing progression to hydrops and perinatal mortality by etiology group would be useful.

Response: Thank you for your helpful feedback. We have added the suggested figure to the manuscript as Figure 2.

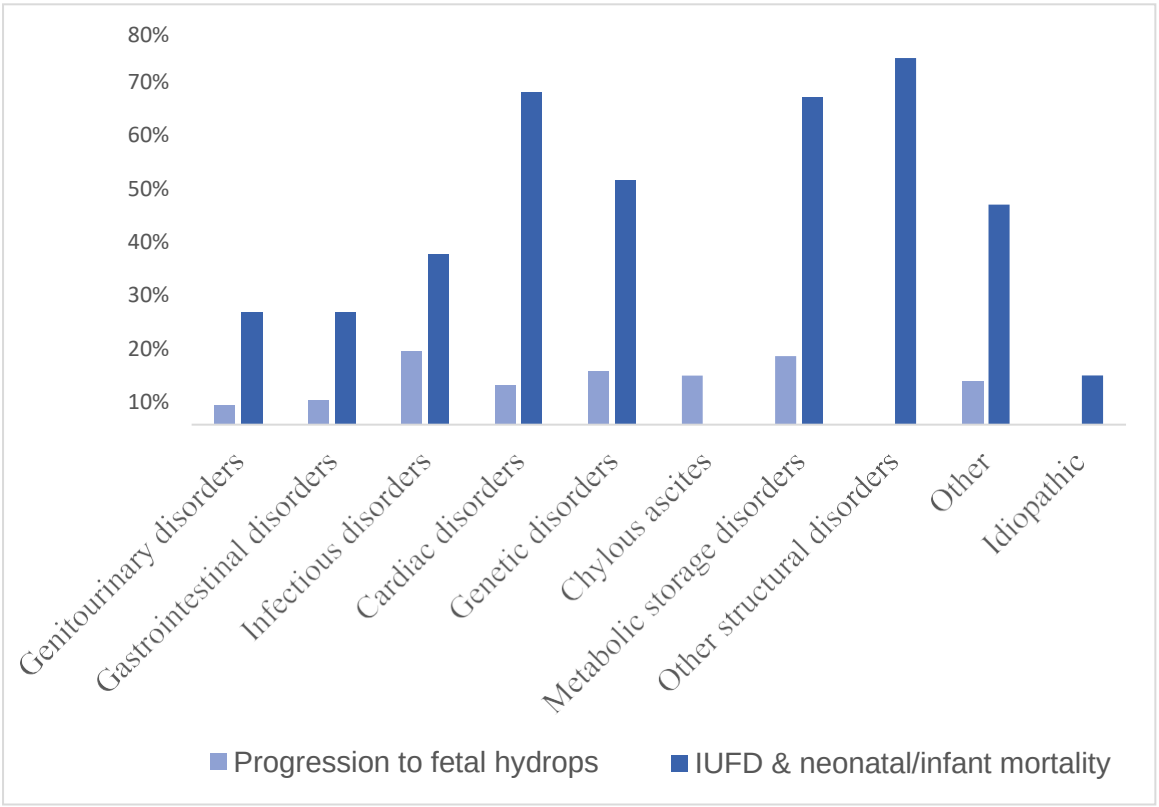

Figure 2 – Rate of progression to fetal hydrops and IUFD and neonatal/infant mortality

8). Discussion, lines 319 - 320: the hiring to termination of pregnancy almost certainly leads to overestimation of the survival rate, as in general people are more likely to terminate more grave cases. I would be more clear with the admission of this limitation is it is crucial to counseling. I've seen some similar articles report a survival frequency with the denominator of both all pregnancies including terminations and also excluding terminations.

Response: Thank you for your helpful suggestion. We have added to the results section the overall survival rate and the survival rate when termination of pregnancy is excluded: (Line 384-387) "The overall survival rate amongst cases of isolated fetal ascites with a known outcome was 58% (178/308). When termination of pregnancy was excluded, the survival rate amongst fetuses with isolated fetal ascites was 70% (178/255) (Table 3)."

Reviewer 3

1). The termination rates seem to be highly skewed with the genitourinary and storage disorders showing high rates closely followed by genetic and structural disorders Any criteria for termination or was it patient choice?

Response: The data is compiled from 11 different studies worldwide. Unfortunately, we do not have information regarding the specific criteria for pregnancy termination in each of the individual studies. We do acknowledge it could have varied significantly from country to country. Also, there was no statistically significant difference in the rate of TOP among the different etiologies aside form infectious, chylous and idiopathic being lower. We added that to the result section under the stat editor answers as a stat table was requested for the same.

2) As in all cases of fetal anomalies it is difficult to estimate survival since the termination rates are high Table 1 The study by Dreux at al(2015) shows that a number of fetuses were terminated from 27- 30 weeks(GUT),31-34weeks(GIT). Fetuses with advanced gestational age tend to be born alive and survive with minimal support unless they have a lethal anomaly. The legality of termination also varies from country to country. The query is whether these babies were liveborn or was feticide carried out prior to termination?

Response: I reached out to Dr Dreux seeking clarification and she has provided the following response "In France, TOP can be processed at any time of the pregnancy if pathology is considered very severe. The limit is the birth. Parent's request must be considered by a multidisciplinary prenatal center. And feticide is done if all practitioners agree." Therefore, we can conclude from Dr Dreux's response that fetuses with advanced gestational age were not born alive. There were two other authors where termination of pregnancy also occurred at greater than 24 weeks and we had original data from both. Dr Favre is a member of the same group as Dr Dreux and Dr Boutall in South Africa had the following response "None of the terminations after 24 weeks were feticides. In our department if the fetus is not expected to survive for any significant amount of time (usually due to pulmonary hypoplasia) we

offer an induction of labor as we have very limited feticide resources. All of these fetuses demised at birth. Those that had an Apgar score above 0 were classified as neonatal demise". Therefore, with the above responses and data we have, we are confident that those who had pregnancy termination beyond 24 weeks were not born alive.

Statistical Editor

1). Lines 171-262 and Table 2: Need to round all %s to nearest integer % and include 95% CIs for each %. The TOP and unknown entries should also have %s and CIs included.

Response: Below is the revised table (previously Table 2, now Table 3). We rounded all percentages to the nearest integer and added the corresponding confidence intervals.

Table 3 in revised manuscript: Outcomes by etiology of isolated fetal ascites

Etiology	Total Cases N (% [95%-CI])	Progression to Hydrops N / Total* (% [95%-CI] p-value)	Pregnancy Outcome N (Overall % [95%-CI] p-value) / {Continuing Pregnancies With Known Outcomes Only}†				
			TOP	Unknown	IUFD	Neonatal/Infant Death‡	Live Birth without Reported Neonatal/Infant Death
Total	315 (100%)	17/259 (7% [3.9-10.3])	53 (17% [12.9-21.4])	7 (2% [0.9-4.5])	35 (11% [7.9-15.1]) / {14% [9.8-18.6]}	42 (13% [9.8-17.6]) / {16% [12.1-21.6]}	178 (57% [50.8-62.1]) / {70% [63.8-75.4]}
Genitourinary Disorders	75 (24% [19.2-28.9])	2/47 (4% [0.5-14.5] p=0.37)	26 (35% [24-46.5] p=1)	2 (3% [0.3-9.3] p=0.78)	7 (9% [3.8-18.3] p=0.37) / {15% [6.2-28.3] p=0.7}	4 (5% [1.5-13.1] **p=0.01**) / {9% [2.4-20.4] p=0.07}	36 (48% [36.3-59.8] p=0.84) / {77% [62-87.7] p=1}
Gastrointestinal Disorders	62 (20% [15.4-24.5])	3/56 (5% [1.1-14.9] p=0.48)	9 (15% [6.9-25.8] p=0.37)	0 (0% [0-5.8] p=0.21)	2 (3% [0.4-11.2] **p=0.02**) / {4% [0.5-13] **p=0.01**}	10 (16% [8-27.7] p=0.83) / {19% [9.4-32] p=0.77}	41 (66% [53-77.7] p=1) / {77% [63.8-87.7] p=1}
Infectious Disorders	28 (9% [6-12.6])	4/26 (15% [4.4-34.9] p=0.98)	1 (4% [0.1-18.3] **p=0.03**)	1 (4% [0.1-18.3] p=0.88)	8 (29% [13.2-48.7] p=1) / {31% [14.3-51.8] p=1}	1 (4% [0.1-18.3] p=0.09) / {4% [0.1-19.6] p=0.05}	17 (61% [40.6-78.5] p=1) / {65% [44.3-82.8] p=1}
Cardiac Disorders	28 (9% [6-12.6])	2/25 (8% [1-26] p=0.78)	2 (7% [0.9-23.5] p=0.12)	1 (4% [0.1-18.3] p=0.88)	8 (29% [13.2-48.7] p=1) / {32% [14.9-53.5] p=1}	9 (32% [15.9-52.4] p=1) / {36% [18-57.5] p=1}	8 (29% [13.2-48.7] **p=0.03**) / {32% [14.9-53.5] p=0.5}
Genetic Disorders	24 (8% [4.9-11.1])	2/18 (11% [1.4-34.7] p=0.9)	6 (25% [9.8-46.7] p=0.91)	0 (0% [0-14.2] p=0.57)	3 (13% [2.7-32.4] p=0.73) / {17% [3.6-41.4] p=0.78}	6 (25% [9.8-46.7] p=0.97) / {33% [13.3-59] p=0.98}	9 (38% [18.8-59.4] p=0.25) / {50% [26-74] p=1}
Chylous Ascites	20 (6% [3.9-9.6])	2/20 (10% [1.2-31.7] p=0.87)	0 (0% [0-16.8] **p=0.02**)	0 (0% [0-16.8] p=0.63)	0 (0% [0-16.8] p=0.09) / {0% [0-16.8] p=0.05}	0 (0% [0-16.8] p=0.05) / {0% [0-16.8] **p=0.02**}	20 (100% [83.2-100] p=1) / {100% [83.2-100] p=1}
Metabolic Storage Disorders	11 (3% [1.8-6.2])	1/7 (14% [0.4-57.9] p=0.93)	5 (45% [16.7-76.6] p=1)	0 (0% [0-28.5] p=0.78)	0 (0% [0-28.5] p=0.27) / {0% [0-45.9] p=0.41}	4 (36% [10.9-69.2] p=0.99) / {67% [22.3-95.7] p=1}	2 (18% [2.3-51.8] **p=0.03**) / {33% [4.3-77.7] p=0.48}
Other Structural Disorders	13 (4% [2.2-7])	0/8 (0% [0-36.9] p=0.58)	3 (23% [5-53.8] p=0.84)	2 (15% [1.9-45.4] p=1)	1 (8% [0.2-36] p=0.57) / {13% [0.3-52.7] p=0.7}	5 (38% [13.9-68.4] p=1) / {63% [24.5-91.5] p=1}	2 (15% [1.9-45.4] **p=0.01**) / {25% [3.2-65.1] p=0.26}
Other	12 (4% [2-6.6])	1/11 (9% [0.2-41.3] p=0.84)	1 (8% [0.2-38.5] p=0.37)	0 (0% [0-26.5] p=0.76)	2 (17% [2.1-48.4] p=0.86) / {18% [2.3-51.8] p=0.82}	3 (25% [5.5-57.2] p=0.94) / {27% [6-61] p=0.91}	6 (50% [21.1-78.9] p=1) / {55% [23.4-83.3] p=1}
Idiopathic	42 (13% [9.8-17.6])	0/41 (0% [0-8.6] p=0.05)	0 (0% [0-8.4] **p=0.0002**)	1 (2% [0.1-12.6] p=0.76)	4 (10% [2.7-22.6] p=0.49) / {10% [2.7-23.1] p=0.3}	0 (0% [0-8.4] p=0) / {0% [0-8.6] **p=0.0003**}	37 (88% [74.4-96] p=1) / {90% [76.9-97.3] p=1}

* Calculated using pregnancies with known outcomes and cases of TOP which progressed to hydrops prior to TOP.

† Confidence intervals calculated with Clopper-Pearson exact binomial interval. Two tailed Fisher Exact P-values calculated from hypergeometric distribution. Continuing pregnancies with known outcomes only excluded the TOP and unknown categories.

‡ There were a total of 4 infant deaths at ≤3 months of age. Two were from the gastrointestinal etiology, 1 from the genetic etiology and 1 from the other category.

0 Abbreviations: IUFD; intrauterine fetal demise, TOP; termination of pregnancy

2) There should be a Table comparing stats of survival by etiology for all comparisons made in Results. The Table should include n/N counts, %s and 95% CIs, along with stats tests. Of note, the difference between cohorts > 24 wks and < 24 wks needs to define which subset those with GA = 24 wks were assigned and the stats result is NS and likely also a reflection of selective loss and TOP rates.

Response: We added to Table 3 above all the stats of survival by etiology. We added the following to the method section: (line 221-224) Two tailed Fisher Exact P-values calculated from hypergeometric distribution was used to compare outcome by etiology. Confidence intervals were calculated with Clopper-Pearson exact binomial interval. A p value <0.05 was considered statistically significant. The following was added to the result section: (line 397-408)
There was no difference in the rate of progression to hydrops for any etiology (Figure 2). Comparing all etiologies, pregnancy termination was less frequent if isolated fetal ascites was due to an infectious, chylous or idiopathic etiology (Table 3). Among all continuing pregnancies with known outcome, IUFD was less frequent when isolated fetal ascites was due to a gastrointestinal etiology (4%, p=0.01). Compared to all etiologies, neonatal/infant death was less likely when isolated fetal ascites was due to a genitourinary etiology (5%, 0.01) and live birth without reported neonatal/infant death was less likely in cardiac (29%, p=0.03), metabolic storage (18%, p=0.03) and other structural disorders (15%, p=0.01).

Thank you for this clarifying comment. Patients with a gestational age of 24 weeks exactly were included in the > 24-week group. We have now amended the greater than (>) symbol to the greater than or equal to symbol (≥) throughout the text and tables.

Author's comments & changes:
On further review of our supplemental tables, we noted that four of the reported neonatal deaths occurred at > 28 days of life. These were 2 cases from the gastrointestinal etiology, 1 from the genetic etiology and 1 from the other category. Therefore, we have amended the tables and text of our manuscript to add the term infant death when infant death occurred. We updated our methods section to state (line 213) "Pregnancy outcome was classified as live birth, neonatal or infant death, intrauterine fetal demise (IUFD), termination of pregnancy (TOP) and unknown". In the results section we added (line 391-392) "We had a total of 35 IUFD, 38 neonatal deaths and 4 infant deaths by 3 months of age occurred (Table 3)".

We updated our abstract as follows:
Line 111-114: Two tailed Fisher Exact P-values calculated from hypergeometric distribution was used to compare outcome by etiology. Confidence intervals were calculated with Clopper-Pearson exact binomial interval.
Line 130: In the majority of cases, fetal ascites does not progress to fetal hydrops.

Supplemental Table 11: Excluded studies in our review with < 5 cases of isolated fetal ascites

Category	Author	Year	Number of Cases of Fetal Ascites	Etiology
Genitourinary	Kobata	1982	1	Prune belly syndrome due to distal urethral obstruction
	Kramer	1985	1	Prune belly syndrome
	Kunt	2020	1	Prune belly syndrome with overlapping

				presentation of partial urorectal septum malformation sequence
	Johnson	1982	1	Prune belly syndrome with urethral hypoplasia
	Perez-Brayfield	2001	1	Prune belly syndrome with severe urethral hypoplasia
	Sakamoto	1987	1	Prune Belly Syndrome
	Simon	1986	1	Prune belly syndrome with cryptorchidism
	Smythe	1981	1	Prune belly syndrome
	Bataille	2007	1	Fetal bladder rupture
	Bracero	2011	1	Spontaneous fetal bladder rupture
	Brunner	2017	1	Spontaneous fetal bladder rupture
	Lowenstein	2003	1	Congenital bladder perforation
	Magawa	2018	1	Spontaneous bladder rupture due to posterior urethral valves
	Oki	2016	1	Fetal bladder rupture
	Padwell	1987	1	Fetal bladder rupture
	Singh	2013	1	Fetal bladder rupture
	Son	2010	1	Bladder rupture secondary to neuropathic bladder in a fetus with meningomyelocele
	Spasojevic	2009	1	Idiopathic bladder rupture
	Bettelheim	2000	1	Lower urinary tract obstruction with fetal bladder rupture
	Chen	1997	1	Posterior urethral valves with fetal bladder rupture
	Cortes-Osorio	2012	1	Posterior urethral valves
	Hecher	1991	1	Posterior urethral valves
	Kelly	1989	1	Posterior urethral valves
	Lacher	2007	1	Posterior urethral valves with bladder rupture
	Scott	1976	1	Posterior urethral valves
	Sofia Mercy	2015	1	Posterior urethral valves
	Vasconcelos	2014	1	Posterior urethral valves with prune belly syndrome
	Camanni	2009	1	Persistent urogenital sinus
	Gul	2008	1	Persistent urogenital sinus
	Loganathan	2014	1	Persistent urogenital sinus
	Nigam	2014	1	Persistent urogenital sinus and cloaca
	Pauleta	2010	1	Persistent urogenital sinus
	Adams	1998	1	Urinary Ascites with persistent cloaca
	Chilakala	2012	1	Ruptured remnant of urachal diverticulum

	Isguder	2020	1	Urorectal septum malformation sequence
	Lee	2013	1	Partial urorectal septum malformation sequence
	Machin	1985	1	Bilateral multicystic kidneys
	Pappas	1961	1	Fetal megabladder
	Persutte	1989	1	Fetal obstructive uropathy
	Rosenberg	1960	1	Urachal cyst
	Soman	2006	1	Urinary hydrocolpos
	Staboulidou	2006	1	Sinus urogenitalis
Gastrointestinal	Agarwal	2000	1	Idiopathic meconium peritonitis
	Agrawala	2005	1	Meconium peritonitis due to colonic atresia
	Chen	2004	1	Meconium peritonitis
	Claudio	2018	1	Meconium peritonitis
	Foster	1987	3	Meconium peritonitis
	Koyanagi	2012	1	Meconium peritonitis
	Garb	1980	1	Meconium peritonitis
	Leppert	1984	1	Meconium peritonitis
	Lin	2007	1	Meconium peritonitis due to intrauterine intussusception
	Lin	1992	1	Meconium peritonitis
	Marcellin	2012	2	Meconium peritonitis in both twins with twin-to-twin transfusion syndrome
	Okawa	2008	1	Meconium peritonitis
	Rachagan	1989	1	Meconium peritonitis due to meconium ileus
	Seow	2000	1	Meconium peritonitis due to terminal ileum perforation
	Sengupta	2015	1	Meconium Peritonitis
	Shimokawa	1986	1	Meconium peritonitis
	Skoll	1987	1	Meconium peritonitis due to multiple congenital atresias of the bowel
	Taba	2010	1	Meconium peritonitis due to intrauterine volvulus
	Wall	1959	1	Meconium peritonitis
	Cruickshank	1921	1	Persistent cloaca with imperforate hymen
	Chen	2010	1	Persistent cloaca
	Morikawa	2006	1	Persistent cloaca
	Motoi	2009	1	Persistent cloaca
	Ohno	2000	1	Cloacal anomaly
	Jeican	2016	1	Ileal atresia

	Machin	1985	1	Small bowel atresia
	Osmulikevici	2017	1	Jejunal atresia
	Wilson	2017	1	Jejunal atresia
	Tan	2019	1	Jejunal atresia after fetoscopic laser ablation of twin to twin transfusion syndrome
	Ozlu	2019	1	Intestinal atresia
	Sciard	2019	3	Liver gestational alloimmune disease
	Casaccia	2006	1	Prenatal rectal perforation
	Chilukuri	1990	1	Spontaneous rupture of the common bile duct
	DeRusso	2003	1	Intestinal malrotation and omental cyst
	Fletcher	1964	1	Liver giant cell transformation
	Giancotti	2011	1	Malformation of intrahepatic bile ducts
	Hersh	1983	1	Omphalocele
	Rosgaard	1996	1	Ductal plate malformation of the liver
Infectious	Binder	1988	1	CMV
	Chan	2020	1	CMV
	Chen	2010	1	CMV
	Chou	2001	1	CMV
	Frank	1966	1	CMV
	Fujioka	2017	2	CMV
	Finegold	1982	1	CMV with paucity of bile ducts
	Stocker	1985	1	CMV
	Symonds	1974	1	CMV and urethral atresia
	Szeifert	1985	1	CMV
	Blaakaer	1986	1	Toxoplasmosis
	Vanhaesebrouck	1988	1	Toxoplasmosis
	Cho	2013	1	Hepatitis A
	McDuffie	1999	1	Meconium peritonitis after maternal Hepatitis A
	Ling	2006	1	Hepatitis C
	Pradhan	2012	1	Hepatitis E
	Aziz	1974	1	Congenital Syphilis
	Quinlivan	1998	1	Congenital listeriosis
Cardiac	Allan	1981	1	Subendocardial fibroelastosis
	Arger	1979	1	Premature closure of the foramen ovale
	Matsubara	2008	1	Congenital atrioventricular block
	Muraoka	2017	1	Fetal left ventricular non-compaction cardiomyopathy

	Balci	1999	1	Fraser syndrome
Genetic	Cardoso	2019	1	Cri-du-Chat Syndrome
	Chen	2004	1	Complete trisomy 9
	Chen	1997	1	Cutis marmorata telangiectasia congenita
	Chen	2010	1	Partial trisomy 16p and partial monosomy 22q
	Chen	2009	1	46,XX,DER(13;21)(Q10;Q10),+21
	Chen	2014	1	Mosaic tetrasomy 9p
	Chen	1999	1	Bilateral renal agenesis with partial trisomy 13 and partial trisomy 16
	Duncan	1996	1	Down syndrome
	Entezami	1996	1	Xerocytosis
	Sanchez	2005	1	Xerocytosis
	Alessandri	2008	1	Perlman Syndrome
	Grootenboer	2001	1	Hereditary stomatocytosis
	Basu	2003	1	Hereditary stomatocytosis
	Greenberg	1986	2	Perlman syndrome
	Itai	2018	1	Intestinal pseudo-obstruction associated with Leigh syndrome
	Lienhardt	1999	1	Gunther disease
	Hengstschlager	2003	1	Supernumerary aberrant chromosome 9
	Hoagland	1988	1	Trisomy 18 with lower urinary tract obstruction due to severe prostatic hypoplasia with an angulated urethra
	Rijhsinghani	2002	1	Cystic fibrosis with meconium ileus
	Straub	1983	1	Conradi's disease
	Scarbrough	1988	1	1q deletion
	Schmider	2001	1	Primary lymphangiectasia
	Wax	1992	1	Turner Syndrome
	Tikanoja	2004	1	Mulibrey nanism with fetal growth restriction
Chylous	An	2017	1	Fetal chylous ascites due to Multifocal Kaposiform Hemangioendothelioma
	Chereau	2007	1	Chylous ascites
	Babic	2012	1	Chylous ascites
	Chye	1997	3	Chylous ascites
	Chen	1998	1	Chyloperitoneum and omental cysts
	Fung	1997	1	Chylous ascites
	Leung	2001	2	Chyloperitoneum
	Mitsunaga	2001	2	Chylous ascites
	Pai	2017	1	Chylous ascites due to fetal neuroblastoma

	Pan	1995	1	Chylous ascites
	Sarno	1990	1	Chyloperitoneum
	Winn	1990	2	Chylous ascites
Storage Disorders	Colin	2016	1	Niemann-Pick type C disease
	Maconochie	1989	1	Niemann-Pick type C disease
	Spiegel	2009	4	Niemann-Pick type C disease
	Ben-Haroush	2003	1	Wolman disease
	Dos Santos	1998	1	Galactosialidosis
	Lee	2015	1	Type 2 sialidosis
	Machin	1985	1	Lysosomal storage disease
	Saxonhouse	2003	1	Mucopolysaccharidosis type VII
	Poulain	1995	1	Sialic acid storage disease
	Zigman	2018	1	Infantile free sialic acid storage disease
Structural	Chou	1992	1	Type III bilateral congenital cystic adenomatoid malformation of the lung.
	Bonnefoy	2011	1	Lobar bronchial atresia
	Degani	1985	1	Fetal ovarian cyst
	Diamond	2003	1	Congenital cystic adenomatoid malformation
	Gilboa	2007	1	Hydrometrocolpos and uterine didelphys
	Gosavi	2017	1	Congenital High Airway Obstruction Syndrome
	Isnard	2007	1	Congenital cystic adenomatoid malformation of the lung
	Ito	2002	1	Fetal ovarian cysts
	Jacquemyn	1998	1	Imperforate hymen
	Meizner	1990	1	Extralobar lung sequestration
	Morrison	1998	3	Laryngeal atresia or stenosis
	Omenaca	1976	1	Right diaphragmatic hernia
	Sakamoto	1987	1	Polysplenia syndrome
	Travan	2016	1	Isolated hypoplasia of abdominal wall muscles
	Zhou	2009	1	Tracheal agenesis
Idiopathic	Chen	1995	1	Idiopathic
	Chen	2007	1	Idiopathic
	Chiang	2004	1	Idiopathic
	Chou	2001	1	Idiopathic
	Defoort	1989	1	Idiopathic
	Kuniakova	2019	1	Idiopathic
	Nwosu	1994	1	Idiopathic

	Mueller-Heubach	1983	2	Idiopathic
	Okamura	1988	3	Idiopathic
	Richards	1990	1	Idiopathic (resolved after administration of betamethasone & prednisone, maternal lupus with fetal heartblock)
	Sakamoto	1987	1	Idiopathic
	Winn	1990	2	Idiopathic
Other	Cozzi	2010	1	Fetal abdominal lymphangioma
	Isin	1997	1	Maternal-fetal trauma
	McKinney	2000	1	Ritodrine exposure
	Rossi	2010	1	Umbilical arterial necrotic vasculopathy
	Schurr	2008	1	Infantile myofibroma
	Shima	2003	1	Congenital fibrosarcoma of the jejunum with meconium peritonitis
	Stiller	1996	1	ABO incompatibility
Total			204	

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