



32 Oxytocin regimens to prevent uterine atony after vaginal delivery: a 3-arm double-blind RCT

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OBJECTIVE: Oxytocin use to prevent post partum hemorrhage is a common obstetric intervention but optimal dosing is unknown. We compared 2 higher dose regimens to our routine regimen for vaginal deliveries.

STUDY DESIGN: Double-masked randomized trial of three oxytocin regimens: 80U, 40U or 10U of oxytocin in 500ml crystalloid solution given after delivery as a 1-hour infusion. Any uterotonic or other treatment for hemorrhage could be used as indicated - additional oxytocin was discouraged during the 1-hour infusion. The primary composite outcome was treatment for uterine atony or hemorrhage including additional uterotonics, tamponade, surgical or interventional treatment, or blood transfusion. Secondary outcomes included dose-response and safety. To detect a 33% decrease in the primary outcome (relative to the 10U group) with 80% power and 2-tailed alpha of 0.05, a total of 1800 patients were required. At planned interim review (n=1200), the 40U arm was stopped for fertility; enrollment continued in the 10U and 80U dose arms.

RESULTS: 1798 out of 2869 women were randomized and analyzed; 59% were African-American and 37% nulliparous; mean gestational age was 39 weeks. The groups had similar demographics and risk factors for atony. Results for selected pre-specified outcomes are presented in the table. The relative risk (95% CI) of the primary outcome was 0.93 (0.62-1.40) for the 80U and 0.94 (0.61-1.47) for the 40U group. Treatment with additional oxytocin was less frequent in the 80U compared to 10U group (RR 0.41; 0.19-0.88) as was the frequency of hematocrit drop >6% (0.83; 0.69-0.99). No such differences were observed between the 40U and 10U groups. Surgical or other treatments were rare and similar across groups as were safety outcomes.

CONCLUSIONS: Compared with our usual 10U oxytocin regimen, higher dose regimens were safe but did not reduce the incidence of the primary composite outcome. Higher dose oxytocin may reduce need for additional oxytocin and incidence of drop in hematocrit > 6%.

Table: Results for primary and selected secondary outcomes

Outcome	Incidence % (or Mean ± SD)			P-values for comparisons	
	80U (N=658)	40U (N=481)	10U (N=659)	80 vs. 10U	Trend
Primary composite	6	6	7	0.745	0.744
Any Uterotonic	6	6	7	0.580	0.578
Additional oxytocin*	1	2	3	0.018	0.019
Methergine*	4	4	4	0.672	0.673
Hemabate*	3	3	3	0.533	0.525
Blood Transfusion*	<1	<1	1	0.564	0.559
2^o outcomes					
Hematocrit change (%)	-3.9 ± 3.0	-4.0 ± 3.0	-4.2 ± 2.9	0.084	0.091
Hematocrit drop >6%	23	27	28	0.045	0.046
EBL >500cc	4	5	6	0.070	0.191
Safety					
Fluid overload	<1	0	0	0.500	0.366
Pressor treatment	14	12	13	0.416	0.409

* Indicates exact test; EBL=Estimated blood loss

33 Diastolic cardiac pathology predicts clinical twin-twin transfusion syndrome in discordant monochorionic/diamniotic twins

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OBJECTIVE: Twin-Twin Transfusion Syndrome (TTTS) occurs in 10% of monochorionic/diamniotic (MC/DA) gestations. However some pregnancies may be affected by discordant amniotic fluid and/or growth without TTTS; others with early presumed TTTS will not progress. We hypothesized that "recipient" diastolic pathology would be more likely to be present in true stage I-II TTTS.

STUDY DESIGN: We evaluated echocardiograms of all pregnancies with suspected TTTS seen at our institution Jan 2006- July 2009. Pregnancy outcomes and Quintero stage at presentation and on followup were recorded. Tricuspid inflow and venous Doppler (umbilical, ductus venosus, inferior vena cava) signals were evaluated by qualitative and quantitative methods and left ventricular isovolumic relaxation time (LVIVRT) was measured.

RESULTS: 220 pregnancies were evaluated for suspected TTTS;123 had echos available. 8 had severe TTTS (Stage III-V) and were excluded. The remainder comprise the study group (n=115). Mean GA at evaluation was 20.2 wks (15.9-28.7). 41 did not have or develop TTTS (Group A), and 64 had TTTS Stage I or II (Group B) at presentation; 10 developed TTTS after initial evaluation. 9/64 with TTTS Stage I or II progressed after initial evaluation. There was a significant difference between groups A and B in the number of fetuses with right ventricular diastolic pathology (34% vs 87%, P<0.001, sensitivity 87%, specificity 63%). Adding LVIVRT >50ms as an abnormal finding, the sensitivity of diastolic pathology for predicting presence of TTTS increased to 90% (specificity 55%). Diastolic pathology was seen in 100% of recipients in which development or progression of TTTS was documented. Quantification of tricuspid inflow time as a percent of cardiac cycle in the suspected recipient revealed a shorter filling time in fetuses with TTTS (31 +/-6% vs 37 +/-4%, P<0.001).

CONCLUSIONS: Diastolic pathology was present in the majority of pregnancies with TTTS but absent in most referred MC/DA pregnancies without TTTS. Diastolic pathology may be suggestive of risk for development or progression of TTTS in MC/DA pregnancies.