



Acta Genet Med Gemelloi 34:179-184 (1985)
© 1985 by The Mendel Institute, Rome

Received 2 November 1984
Final 15 March 1985

Acute Polyhydramnios Complicating Twin Pregnancies

K.T.M. Schneider, K. Vetter, R. Huch, A. Huch

Department of Obstetrics, University of Zurich

Abstract. Acute polyhydramnios in the second trimester is a typical complication in monozygous twin pregnancies. It is caused by a feto-fetal transfusion with anemia on the donor and polycythemia on the recipient twin. Contrary to the chronic hydramnios, there is no increase in malformations. In view of the high mortality rate (100%, according to most authors), the clinical management has to be reconsidered. During the years 1979 to 1983, 10 cases of acute polyhydramnios have been observed at the University Hospital in Zurich. This corresponds to an incidence of 9% in our twin population. All cases investigated were MZ twin pregnancies. With the exception of one patient, who underwent an abortion, all women were hospitalized, had bed rest and received recurrent removals of amniotic fluid and prophylactic tocolysis. The mean gestational age at the time of diagnosis was 23 4/7 weeks and at delivery 30 3/7 weeks. In two cases – one of which is presented in detail – with an unintentional puncture of a placental vessel, the recurrence of the hydramnios did not appear. Eight of 18 newborns survived. No malformations were found. Bed rest, tocolysis and recurrent amniocenteses seem to have a positive influence on the prolongation and outcome of the gestation in acute polyhydramnios.

Key words: Acute polyhydramnios, Twin pregnancy, Feto-fetal transfusion, Amniocentesis

INTRODUCTION

Acute polyhydramnios is defined as condition where the amniotic fluid exceeds 2000 ml or a standard deviation above the amount corresponding to the gestational age and as a rapid development of this increase within a few days [1,2,9,10,15]. This complication is often associated with monozygotic twin pregnancies [2,11,15]. The first description was made by Schatz in 1882 [12]. It is caused by an unidirectional shunt between the two fetal circulations leading to anemia of the donor and to polycythemia of the recipient twin.

The increased urine excretion of the hypervolemic recipient twin results in "acute polyhydramnios" as the principal symptom of the mother. This hypothesis is supported by the fact that the hydramnios is apparent in the amnion of the recipient twin only. The donor twin, with reduced weight compared to the cotwin, shows a much lower urine excretion resulting in an oligohydramnios in his amnion [2,4,6,11,14]. The reported incidence of acute polyhydramnios ranges from 0.7% up to 6% of all twin pregnancies [1,2,9,10,15]. The extreme perinatal mortality of nearly 100% [1,2,4,6,10,15] results from the addition of two problems: the implications of the fetto-fetal transfusion, on the one hand, and the extreme prematurity caused by the hydramnios on the other hand.

Fetal malformations in acute polyhydramnios – in contrast to the incidence in chronic hydramnios – are not increased [1,6,7,10,11,15]. In view of the high mortality rate of two potentially healthy infants, it is important to reconsider the clinical management.

MATERIALS

During the years 1979-1982, 10 cases of acute polyhydramnios were observed at the Department of Obstetrics of the University of Zurich. This represents an incidence of 9% of our total twin births. All cases were monochorial, diamniotic twins (proofed by histologic examination of the placenta). Seven of the 10 women were multiparae. The median time of diagnosis of acute polyhydramnios by clinical examination and ultrasound was in the 23 1/7 week of gestation. At the time of diagnosis the median increase in body weight was 13 kg and the median abdominal circumference 110 cm. The uterine fundus was palpable on the costal arch in all patients at that time (Table 1). Most women suffered from dyspnea and abdominal pain.

MANAGEMENT AND RESULTS

In one patient, an abortion was performed in the 18th week of gestation. To prevent preterm labour, we indicated bed rest and tocolysis in all of these patients. Two women underwent a cerclage and five an induction of lung maturity. With the exception of one patient who had premature rupture of the membranes and thus a spontaneous decompression, we performed repeated amniocenteses in all other cases. The mean amount of amniotic fluid removal was 1300 ml per session. In association with this therapeutic measure the mean date of delivery was in the 31st week of gestation. The gain in gestational age between the appearance of symptoms and the delivery ranged between 4 and 83 days, with a median of 43 days. In 78% of cases the delivery mode was a cesarean section (Table 2).

All neonates were MZ twins. The mean birth weight was 1370 g and the mean difference in weight between the donor and the recipient twin was 29%. Four out of 9 treated twin couples survived, 4 fetuses died in utero, and 6 within 4 days after delivery. Malformations were not found (Table 3). In two cases, the overproduction of the amniotic fluid ceased after an unintentional puncture of a placental vessel and consecutive bleeding into the amniotic fluid.

One case will be described in detail because of its special course.

Table 1. Characteristics of Pregnant Women with Acute Polyhydramnios

No.	Age (years)	Para	Grav.	Diagn. (weeks)	At the time of diagnosis		
					Increase in weight (kg)	Fundus	Girth (cm)
1	32	1	2	17 + 6	—	cost. arch	—
2	36	3	3	20 + 0	14	"	110
3	30	3	3	24 + 4	7	"	86
4	21	2	2	28 + 3	7	"	118
5	30	3	3	23 + 0	13.5	"	113
6	28	2	3	32 + 4	7	"	—
7	26	2	3	24 + 0	12	"	116
8	29	2	2	21 + 4	—	"	113
9	25	1	1	21 + 0	—	"	—
10	26	1	1	22 + 5	13	"	98
median	28	multipara gravida		23 + 1	13	cost. arch	110

Table 2. Therapeutic Procedure with Acute Polyhydramnios

No.	Bedrest	Tocolysis	Cerclage	Corticosteroids	Decompression (1 total)	Deliv. (weeks)	Range diag.-del. (days)	Ces. sect.
1*								
2	+	+	—	+	3 x (3.6)	31 + 5	83	+
3	+	+	—	—	4 x (4.8)	28 + 6	30	+
4	+	+	—	+	2 x (3.1)	29 + 0	4	—
5	+	+	+	+	17 x (21.0)	33 + 4	74	+
6	+	+	+	—	2 x (2.1)	33 + 3	6	+
7	+	+	—	+	6 x (7.8)	29 + 5	40	+
8	+	+	—	+	PROM	31 + 1	66	+
9	+	+	—	—	1 x (1.3)	22 + 1	8	—
10	+	+	—	—	2 x (2.1)	33 + 4	76	+
median	all	all	2/9	5/9	all	31 + 0	43	7/9

* Therapeutic abortion

Table 3. Acute Polyhydramnios and Fetal Outcome

No.	Sex	Donor/recip.	Weight (g)	* Alive # Dead	Perinatal complications
1	F	R	250	# abortion	
	F	D	180	#	
2	M	R	1610	*	none
	M	D	1550	*	none
3	M	R	600	# in utero	small f.d., anemia
	M	D	540	# 4h p.p.	
4	F	D	790	# 4d p.p.	RDS, infection
	F	R	840	# 1d p.p.	RDS, mec. asp., inf.
5	F	R	2460	*	RDS
	F	D	1370	*	anemia, erythrobl.
6	M	R	2410	*	RDS, icterus
	M	D	1800	*	none
7	F	R	2230	# 2d p.p.	RDS, hyperkal.
	F	D	1180	# 2d p.p.	open ductus
8	M	R	1130	# 3d p.p.	RDS, anem., pneum
	M	R	2550	# in utero	
9	M	D	350	# in utero	
	M	R	485	# in utero	
10	F	R	2250	*	none
	F	D	1410	*	none
g	M/F 10/10	R in 70% Twin A	1420 29% weight difference	# 56%	64%

Table 4. Acute Polyhydramnios: Therapy and Perinatal Mortality (data from the literature)

Author, year	Case (n)	Tocolysis	Decompression	Delivery (weeks)	Interval diag.-del. (days)	Perin. mortal.
Mueller, 1948	1	no	no	18 + 0	28	
Abdul-Karim, 1962	3	no	no	20 + 1	2	A
Conway, 1964	1	no	no	27 + 6	13	L
Sekiya, 1977	1	*	no	*	*	L
Weir, 1979	8	*	no	26 + 1	8	
Galea, 1982	4	*	no	< 28 + 0	*	
Barry, 1958	3	no	yes	27 + 3	29	6/6
Queenan, 1970	3	no	yes	30 + 3	62	5/6
Mills, 1979	1	*	yes	37 + 0	133	0/2
Schmeer, 1981	1	yes	yes	38 + 0	63	1/1
Own results, 1983	9	yes	yes	30 + 3	43	10/18

CASE HISTORY

A 30-years-old 3-para was transferred to us in the 23rd week of gestation with an acute polyhydramnios in a twin pregnancy. The fundus of the otherwise slim woman was found at the height of the costal margin, the abdominal wall was tightly strained with an abdominal circumference of 113 cm and an increase in body weight of 13.5 kg.

The ultrasound examination showed an extreme hydramnios in the amnion of the bigger twin. Beside bed rest, tocolysis and cerclage, 17 amniocenteses were performed, removing a total of 21 liters of amniotic fluid.

At the beginning of the 28th week a sonographic examination showed a hydrops in the recipient twin. In the 30th week a placental vessel was punctured unintentionally with a marked bleeding into the amniotic fluid. Immediately after this event we observed a decrease of the hydrops in the recipient twin and a stop of the overproduction of amniotic fluid. Subsequently, both twins had a normal development but kept their difference in size. In the 34th week of gestation the membranes ruptured prematurely and green amniotic fluid was discharged. The fetuses were delivered by cesarean section.

The first twin had a weight of 2040 g and normal Apgar score and pH values. The neonate developed an early icterus and a transitory respiratory distress syndrome. The second twin had a weight of 1370 g only; Apgar score and pH values were normal. Because of a hematocrit of 30% he received a blood transfusion. He developed hypoproteinemia, hypocalcemia and an icterus prolongatus.

The further clinical course was uneventful. Both children are now two years old, body weight and length have adapted, and they show a normal development without sequelae.

DISCUSSION

In comparing our results with those of other investigators, we noted (Table 4) that if amniocenteses were performed for decompression, a higher gestational age could be achieved. Survivors could only be found in this group. Premature labour caused by the hyperdistention of the uterus [3,14] may be prevented by this procedure. The maternal or fetal risk of repeated amniocenteses is assessed to be justifiable today [5]. Whether prophylactic tocolysis has added to the result delaying the date of delivery beyond the 28th week of gestation must remain an open question.

In two cases with remarkable differences in size, a stagnation of excessive fluid production could be noticed. In both cases, the progress stopped after an unintentional puncture of a placental vessel. Ultrasound revealed unexpectedly strong bleedings into the amniotic fluid. In the literature, the stagnation of acute polyhydramnios was described only after the intrauterine death of the recipient twin with cardiac insufficiency [13]. In the reported case with extreme fetal hydrops we could document a remission. All four infants were born alive several weeks after cessation of the polyhydramnios. It is not evident if and in which manner a puncture of a placental vessel can stop fetofetal transfusion. However, the striking coincidence between bleeding episodes and onset of remission in these two cases suggests a possible connection.

CONCLUSION

Altogether our therapeutic concept in acute hydramnios including bed rest, prophylactic tocolysis, repeated amniocenteses and delivery at an optimal time in view of lung maturity and hemodynamics is confirmed by the results. Because of the severity of obstetrical and usually also neonatal problems in the surviving twins, treatment of these patients should take place in obstetrical centers with an attached neonatal intensive care unit.

REFERENCES

1. Abdul-Karim R, Iskandar G (1962). Acute Hydramnsio. *Obstet Gynecol* 20:486-489.
2. Barry AP (1958): Hydramnsio. *Obstet Gynecol* 11:667-675.
3. Caldeyro-Barcia R, Pose SV, Alvarez H (1957): Uterine Contractility in Polydramnios and the Effects of Withdrawal of the Excess of Amniotic Fluid. *Am J Obst Gynecol* 73:1238-1254.
4. Conway CF (1964): Transfusion Syndrome in Multiple Pregnancy. *Obstet Gynecol* 23:745-751.
5. Editorial (1978): an assessment of the hazards of amniocentesis. Report to the Medical Research Counciln by their working party on amniocentesis. *Br J Obstet Gynecol* 85:2.
6. Galea P, Scott JM, Goel KM (1982): Feto-fetal transfusion syndrome. *Arch Dis Child* 57:781-783.
7. Kirkinen P (1978): Polyhydramnion. A clinical study. *Ann Chir Gynecol* 67:117-122.
8. Mills WG (1979): Correspondence to: Acute polyhydramnios: A complication of monozygous twin pregnancy. *Br J Obstet Gynecol* 87:256.
9. Mueller PF (1948): Acute hydramnios. *Am J Obstet Gynecol* 56:1069-1076.
10. Pitkin RM (1976): Acute polyhydramnios recurrent in successive pregnancies. Management with multiple amniocenteses. *Obstet Gynecol* 48:42s-43s.
11. Queenan JT, Gadow EC (1970): Polyhydramnios: Chronic versus acute. *Am J Obstet Gynecol* 108:349-355.
12. Schatz F, cited by Conway CF [4].
13. Schmeer G, Bucchia I, Brehm H (1981): Intrauteriner Fruchttod des einen Zwillings nach Polyhydramnion und speatere Spontangeburt des ueberlebenden Zwillings. *Geb Fra* 41:809-810.
14. Sekija S, Hafez ESE (1977): Physiomorphology of twin transfusion syndrome. *Obstet Gynecol* 50:288-292.
15. Weir PE, Ratten GJ, Beischer NA (1979): Acute polyhydramnios. A complication of monozygous twin pregnancy. *Br J Obstet Gynecol* 86:849-853.

Correspondence: Dr. K.T.M. Schneider, Frauenklinikstr. 10, 8091 Zurich, Switzerland.