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RESEARCH LETTER

Management of pregnancy and childbirth in Glanzmann thrombasthenia: A case series and review

To the Editor,

Glanzmann thrombasthenia (GT) is a rare inherited platelet disorder caused by defects in αIIbβ3 integrins, leading to impaired platelet aggregation and increased bleeding risk. Bleeding severity varies, and almost all women suffer from abnormal uterine bleeding (AUB).[1] The risk of bleeding extends throughout all phases of pregnancy, wherein both the mother and child are at risk.[2] We conducted a literature search in the PubMed and Embase databases on studies published up to 22 May 2025 using terms related to GT and pregnancy (Supporting Information S1) to evaluate the risks associated with pregnancy and determine the optimal management approach. The study selection and assessment are described in Supporting Information S2. Eventually, eight cohort studies,[1,3–9] one systematic review including 31 case reports[2] and 24 detailed case reports published after the systematic review by Siddiq et al.[10–33] were included, describing a total of 173 pregnancies (summarised in Supporting Information S3 and S4). Additionally, we present a consecutive case series of eight women with GT planning a pregnancy from three tertiary haemophilia centres in Europe between 2017 and 2024 (Table 1 and Supporting Information S5). All women presenting to these centres during this period were included, with the exception of one woman who did not provide informed consent. One woman opted for adoption following preconception counselling (case 1), one woman is actively trying to conceive (case 3) and another woman had a miscarriage (case 4). Five women (case 2 and case 5–8) have given birth. An overview of the challenges in the different phases of pregnancy in these eight women with GT is illustrated in Figure 1.

The pregnancy journey of women with GT starts with the preconception phase, when interrupting contraceptives may lead to worsening of AUB and increased risk of haemorrhagic corpus luteum.[23] The literature on the prevalence and management of AUB in the preconception phase is very scarce, but it is a common problem in women with GT and may lead to iron deficiency anaemia.[9] In addition to the risk of AUB, there are suggestions that reproductive health problems occur more frequently in women with GT: In our review, ART was required in 11 (50%) of 22 pregnancies in which the mode of conception was described.[5,11,15,16,19–21,29,33] This is relatively high compared to infertility rates of 17.5% in the general population.[34] Possible contributing factors are the increased incidence of ovarian haemorrhage and endometriosis in GT.[35–37] In vitro fertilisation and intracytoplasmic sperm injection have been performed safely in women with GT.[11,15,16,19–21] However, oocyte retrieval led to bleeding complications in one out of the three cases in which bleeding outcome was reported.[33] The haemostatic cover provided around this procedure was highly heterogeneous and is summarised in Supporting Information S4B. In addition, consideration should be given in the preconception phase to the need for genetic screening, which is recommended in the case of consanguinity. It is also essential to involve a multidisciplinary team early in the process to ensure comprehensive care and planning.[38]

An important maternal complication during pregnancy is platelet alloimmunisation. Screening for platelet antibodies was carried out in only 59/173 pregnancies (34%). In these 59 cases, the presence of antibodies to human leucocyte antigen (HLA), αIIbβ3 or both was detected in 9 (15%),[2,9,11,33] 7 (12%)[2,16,22] and 5 (8%)[2,12,27] pregnancies respectively. The antibody type was not specified in five pregnancies (8%)[2,5] and the screen was negative in 33 (56%) of reported cases.[2,9,23,25,29,31] For comparison, in general, the prevalence of HLA and αIIbβ3 antibodies in GT patients is 17%–23%[39,40] and 20%–30%, respectively.[41,42] In addition to pregnancy, repeated exposure to platelet transfusions and the presence of type 1 GT are recognised risk factors for alloimmunisation, with the latter being specifically associated with the development of αIIbβ3 antibodies.[43,44] The formation of such antibodies has therapeutic implications, including the potential requirement for HLA-matched platelet transfusions or the use of alternative therapies such as recombinant activated factor VII (rFVIIa). Monitoring for αIIbβ3 antibodies is relevant, as these antibodies can cause fetal and neonatal alloimmune thrombocytopenia (FNAIT) and associated bleeding complications.[45] This may influence the decision regarding the mode of birth. Additionally, fetal monitoring for suspected intracranial haemorrhage and treatment with intravenous immunoglobulin (IVIG) should be considered. Two small cohort studies addressed platelet alloimmunisation and FNAIT.[5,9]

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TABLE 1 Summary of cases.

Case	Age	Gravity and parity	Genetic variants	Preconception challenges	Mode of conception	Platelet antibodies	Antenatal history	Gestational age at birth	Mode of birth and anaesthesia	Management	PPH (mL)	Neonatal outcome
1	43	G0P0	ITGB3, compound heterozygous missense mutation	Severe menorrhagia and ovarian bleeds requiring platelet transfusion	—	HLA and αIIbβ3 antibodies	—	—	—	Patient opted for adoption	—	—
2	30	G2P0	ITGA2B, homozygous deletion mutation	Ovarian endometriosis, haematosalpinges, early pregnancy loss, subfertility Unacceptable high bleeding risk of oocyte retrieval for IVF	IVF with donor oocytes (haemostatic cover unknown)	No HLA or αIIbβ3 antibodies previous to pregnancy	No complications	37 + 2	El CS General anaesthesia	During delivery: 3 HLA-matched platelets Day 1–7: HLA-matched platelets twice daily Day 8–10: HLA-matched platelets once daily, TXA Day 11–17: TXA	700	Healthy neonate, normal platelet count
3	37	G3P0	ITGA2B, homozygous deletion	Severe bleeding phenotype, requiring blood transfusion following IUI	Not able to become pregnant yet following IUI (haemostatic cover with TXA and rFVIIa)	No HLA antibodies, not tested for αIIbβ3 antibodies	—	—	—	—	—	—
4	27	G1P0	ITGB3, homozygous missense mutation	Severe bleeding phenotype, requiring regular blood transfusions, known with severe menorrhagia and ovulation bleeds.	Natural	No HLA or αIIbβ3 antibodies previous to pregnancy	Severe vaginal bleed requiring 3x rFVIIa. TXA and 2 units of RBCs. Miscarriage in first trimester	—	—	—	—	—
5	38	G2P0	ITGA2B, compound heterozygous	Severe gastro-intestinal bleed, menorrhagia	Natural	Not reported	Vaginal bleed at 14 weeks GA, pre-eclampsia	37 + 1	Em CS General anaesthesia	During delivery: TXA, rFVIIa 90 mcg/kg once Day 1–14: TXA	300	Healthy neonate, normal platelet count
	40	G3P1			Natural	Not reported	Pre-eclampsia	36 + 0	Em CS General anaesthesia	During delivery: TXA, rFVIIa 90 mcg/kg once Day 1–14: TXA	420	Healthy neonate

TABLE 1 (Continued)

Case	Age	Gravity and parity	Genetic variants	Preconception challenges	Mode of conception	Platelet antibodies	Antenatal history	Gestational age at birth	Mode of birth and anaesthesia	Management	PPH (mL)	Neonatal outcome
6	29	G2P0	ITGB3, homozygous missense mutation	Mild bleeding phenotype, known with menorrhagia	Natural	No HLA or αIIbβ3 antibodies	Uncomplicated	38 + 0	Em CS NR	During delivery: TXA, 4 HLA-matched PLTs Day 1 until discharge: daily HLA-matched PLTs, TXA	1000	Healthy neonate
7	36	G2P0	No pathogenic variants in coding region of ITGA2B or ITGB3, diagnosis based on LTA	Severe epistaxis, daily gum bleeding, easy bruising	Natural	HLA-I and HPA-15 antibodies, no αIIbβ3 antibodies	Recurrent epistaxis managed with TXA and iron deficiency managed by oral iron	38 + 3	El CS NR	During delivery: TXA, 4 HLA-matched PLT, 1 RBC Day 1–5: daily HLA-matched PLTs, TXA	1700	Healthy neonate, normal platelet count
8	40	G2P0	ITGA2B, splice site mutation	Gingival bleeds and menorrhagia.	IVF	No HLA antibodies. αIIbβ3 antibodies became detectable 2 months after delivery (not detectable during pregnancy)	No complications	38 + 0	Vaginal, Remifentanil iv	During delivery: 3 HLA-matched PLTs, TXA, rFVIIa 6 mg (repeated after 3 h) Day 1: 1 HLA-matched PLT Day 2: rFVIIa 6 mg TXA continued till lochia were minimal	1600	Severe thrombocytopenia and bruises

Abbreviations: El CS, elective caesarean section; Em CS, emergency caesarean section; GA, gestational age; HLA, human leucocyte antigen; IUI, intrauterine insemination; IVF, in vitro fertilisation; NR, not reported; PLT, platelet transfusion; PPH, post-partum haemorrhage; RBC, red blood cell; rFVIIa, recombinant activated factor VII; TXA, tranexamic acid.

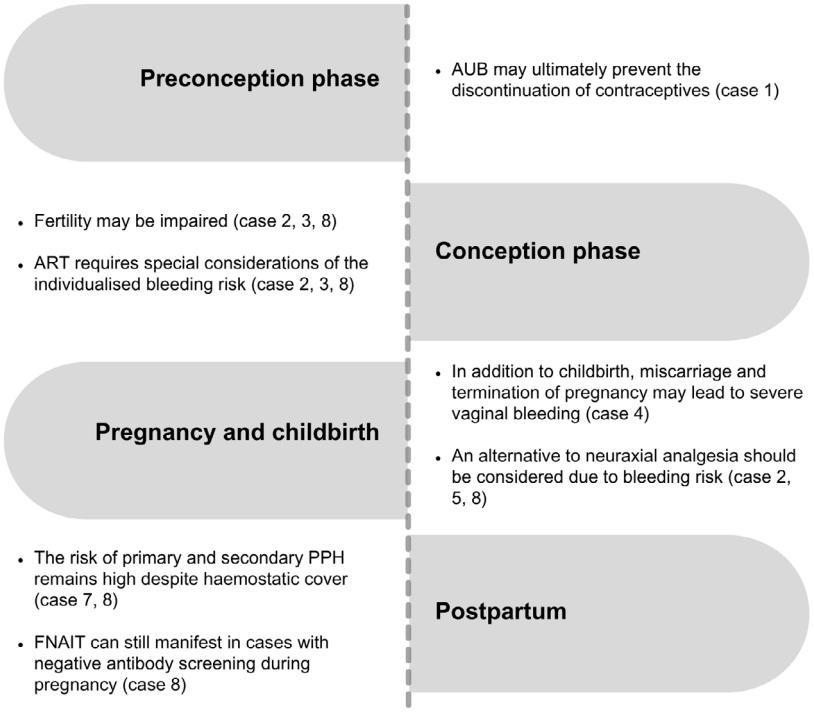


FIGURE 1 Summary of the most important challenges during the preconception phase, conception, pregnancy and childbirth in women with Glanzmann thrombasthenia. ART, assisted reproductive therapy; AUB, abnormal uterine bleeding; FNAIT, fetal and neonatal alloimmune thrombocytopenia; GT, Glanzmann thrombasthenia; PPH, post-partum haemorrhage.

Platelet alloimmunisation occurred in 6/27 (22%) pregnancies. Four women were treated with IVIG during pregnancy, resulting in one healthy neonate, while the neonatal outcomes of the remaining three cases are unknown. In the other two cases, the presence of platelet alloimmunisation was identified only after delivery. Both neonates were diagnosed with FNAIT without bleeding complications and were successfully treated with platelet transfusions and IVIG. Additionally, the use of IVIG is reported in five pregnancies in case reports, resulting in five healthy neonates (including one twin pregnancy) and one neonate with FNAIT without bleeding complications.[2,16,33] Besides antibody screening, haemoglobin and iron levels should be optimised during pregnancy, since anaemia is a known risk factor for post-partum haemorrhage (PPH).[46]

Of 141 pregnancies in which the mode of birth was documented, there were 75 (53%) vaginal births and 66 (47%) caesarean sections (CS) reported. The anaesthetic technique for CS was only documented in 11 cases, of which 91% received general anaesthesia.[11,12,14,16,21,23,25,27–29,32] The mode of birth and anaesthesia should be discussed in a multidisciplinary team ahead of the estimated delivery date. Overall, the mode of birth should be determined by obstetric indication. However, in cases of FNAIT or if the child is at risk of GT, CS is preferred to avoid the use of instrumentation and decrease the risk of neonatal bleeding. Additionally, epidural and spinal anaesthesia should be avoided.[45]

Prophylactic treatment to prevent PPH was administered in the majority of pregnancies (58/79, 73%) when reported. This treatment most often involved platelet transfusion (1–6 units, mean 3.4 units), which was administered in 37 (64%) pregnancies. rFVIIa was administered in 8 (14%), a combination of both in 11 (19%) and 1-deamino-8-D-arginine vasopressin in 2 (3%). Administration of antifibrinolytics was only reported in 24 pregnancies (41%), with a median duration of 11.5 days (range 7–49 days) after childbirth.[2,9,11,12,14–20,22–28,30–33,47] In 21 (27%) pregnancies, no prophylactic treatment was administered.[2,9,10,13,20,21,24,29] Overall, PPH occurred in 84 of 145 deliveries (60%) in which the presence of PPH was described. This included 45 (31%) cases of primary PPH, 21 (14%) secondary PPH and 6 (4%) both primary and secondary PPH. In 12 pregnancies (8%), the type of PPH was not specified. The estimated blood loss was documented in 27 pregnancies, with an average of 1115 mL (range 100–6000 mL).[2,12,13,16,17,19,23,25,27,28,30–33] Primary PPH required red blood cell transfusion in 25/45 (56%), platelet transfusion in 19/45 (42%) and rFVIIa in 10/45 (22%) women. Secondary PPH occurred at a range from 3 days to 3 weeks post-partum and was mostly treated with platelet transfusion (10/21, 48%) or rFVIIa (5/21, 24%). The prevalence of PPH in women with GT is higher than the general population (60%–67% vs. 6.4% respectively) and prophylactic treatment to prevent primary PPH is very heterogeneous.[48] From our literature review, it is not possible to draw conclusions on the optimal way to prevent PPH. Mitigating measures to prevent secondary PPH should include tranexamic acid continuation until lochia are minimal, education and pro-active planning including timely start of hormonal methods of contraception especially in women with severe phenotype.

Although women with GT also have venous thromboembolism risk factors such as PPH, hospitalisation, CS and haemostatic treatments, thrombosis during or after childbirth has not been reported.[49] Additionally, the use of thrombosis prophylaxis in GT was not described in the literature. Outside the scope of pregnancy, thrombosis in GT is extremely rare following the administration of haemostatic therapy such as rFVIIa.[50]

The prevalence of miscarriages in women with GT was 15%[1,3,4,6,7,9] which is comparable to the general population.[50] However, the rates of neonatal complications are higher.[1,3,4,6,7,9] Neonatal outcome was documented for 149 children of 143 pregnancies (six twin pregnancies), of whom 32 (22%) had complications. There were five stillbirth of which four due to intracranial haemorraghe.[2,21,22] Furthermore one neonatal death occurred.[2] In addition there were 17 cases of FNAIT and nine neonatal bleeds.[2,3,5,9,16,32] In 37 pregnancies with reported neonatal outcomes and platelet alloimmunisation status, complications occurred in 45% among the 22 women with antibodies, whereas no complications were seen in the 15 women without antibodies. This finding implies an association between the presence of maternal platelet antibodies and fetal/neonatal outcomes. We recommend that platelet counts should be checked in all neonates.

In conclusion, there is a lack of uniformity in the management of pregnancy and childbirth in women with GT and an unrecognised risk of impaired fertility. Pregnancy in women with GT is associated with a higher maternal and neonatal complication risk, the latter especially in women with maternal platelet antibodies. The management of pregnancy and childbirth requires a multidisciplinary approach and advance planning in a tertiary haemophilia centre. Moreover, there is a need for an international prospective registry and consensus guidelines.

AUTHOR CONTRIBUTIONS

KHGR and OT wrote the main text. OT, KHGR and QVT obtained the clinical data. KHGR conducted the literature search and analysed the data. LB, MH, PR, MK, SS, KPMvG, REGS and QVT reviewed the paper. All authors approved the final version of the paper.

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childbirth, FNAIT, Glanzmann thrombasthenia, platelet alloimmunisation, pregnancy

CONFLICT OF INTEREST STATEMENT

The authors have no competing interests.

PATIENT CONSENT STATEMENT

All patients provided informed consent prior to their participation in the study.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.