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CASE REPORT

Relapsing Superwarfarin Coagulopathy in an Unrecognised Early Pregnancy: An Eight-Month Vitamin K_1 Course Ending in Second-Trimester Pregnancy Loss

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ABSTRACT

Background: Superwarfarins are anticoagulant rodenticides whose hepatic sequestration and enterohepatic recirculation produce a vitamin K–dependent coagulopathy lasting months. Exposure during pregnancy is rarely reported.

Objective: To describe relapsing superwarfarin coagulopathy in a woman whose pregnancy was unrecognised at exposure, and to define the endpoint of antidotal therapy.

Case presentation: An 18-year-old woman presented with two weeks of spontaneous ecchymoses, gum bleeding and haematuria. Haemoglobin was 6.0 g/dL with normal platelets and hepatic and renal function; prothrombin time (PT; >90 s) and international normalised ratio (INR; >6) exceeded the analytical ceiling and activated partial thromboplastin time (APTT) was 133.4 s. She later disclosed ingesting a rodenticide three days before onset. After 28 days of plasma, red cells and intramuscular vitamin K_1 she was discharged (INR 5.32) but relapsed 20 days later (haemoglobin 9.4→4.0 g/dL). Ultrasonography for suspected intraperitoneal bleeding instead disclosed an unsuspected live intrauterine pregnancy of 11 weeks, conceived around the time of ingestion. Despite parenteral vitamin K_1 the INR rebounded to 21.6 on day 10 before responding to high-dose infusion. Oral vitamin K_1 was titrated across 16 visits and withdrawn after eight months; the pregnancy was lost as an incomplete abortion at 18 weeks.

Conclusion: Superwarfarin poisoning should be considered in any unexplained vitamin K–dependent coagulopathy; treatment must target the hepatic toxicant reservoir rather than symptom resolution, and maternal biochemical rescue does not guarantee fetal survival.

1. Introduction

Vitamin K antagonists have been used for both medical and industrial purposes for close to a century. Warfarin, the prototype rodenticide, was regarded as close to ideal for pest control because it is colourless, odourless, slow in onset and reversible with a widely available antidote. Increasing rodent resistance in the 1970s prompted the introduction of second-generation long-acting anticoagulant rodenticides, of which brodifacoum, bromadiolone and difenacoum are the most widely distributed. These compounds are described in the literature both as long-acting anticoagulant rodenticides and as superwarfarins; the latter term is used throughout this report. They are so named because they combine markedly greater potency with an extraordinarily prolonged biological persistence that derives from high lipophilicity, avid hepatic binding and enterohepatic recirculation. In a systematic retrospective analysis of 88 published human anticoagulant rodenticide poisonings, brodifacoum and bromadiolone were the two agents most frequently identified, and the aggregate laboratory picture was one of extreme coagulation abnormality, with a median activated partial thromboplastin time (APTT) of 110 seconds, a median prothrombin time (PT) of 100 seconds and a median international normalised ratio (INR) of 9.[1]

The epidemiology of superwarfarin exposure differs strikingly between world regions, and this difference matters at the bedside because it determines the pre-test probability a clinician should attach to the diagnosis. Poison centre data from high-income Western settings are dominated by unintentional exposures in preschool children, most of which are trivial; an analysis of enquiries to a national poison information centre found that the majority of rodenticide contacts involved either small children or domestic animals and that clinically significant poisoning was uncommon.[2] Reports from Asia describe a very different case mix, in which adults predominate and deliberate self-poisoning accounts for a substantial share of hospitalised cases; in the pooled series of 88 published cases, 31 were suicidal and only 52 accidental.[1] Superimposed on this baseline are episodic outbreaks driven by adulterated illicit products. The 2018 Illinois epidemic of brodifacoum-contaminated synthetic cannabinoids generated more than 160 documented cases of bleeding and prolonged coagulopathy and remains the largest emergency department experience of inhalational exposure yet described.[3] That outbreak also exposed a structural weakness in clinical laboratory provision, because quantitative liquid chromatography–tandem mass spectrometry assays for plasma rodenticide concentrations were not available in the hospitals managing the patients.[4] Similar clusters have since been reported elsewhere, and the radiological burden of such poisoning — visceral, muscular and intracranial haemorrhage — has been systematically catalogued.[5] Sporadic community cases continue to be notified, including a cluster of brodifacoum-associated coagulopathy reported from Florida.[6] The list of offending compounds is also still lengthening: poisoning by a butylated hydroxytoluene quinone methide behaving as a superwarfarin has recently been described for the first time in humans, a reminder that a negative screen for the classical rodenticides does not exclude the syndrome.[7]

The clinical phenotype is deceptively ordinary at onset and then extraordinarily varied. Because the vitamin K-dependent clotting factors have half-lives ranging from approximately four to six hours for factor VII to approximately 60 to 72 hours for prothrombin, measurable coagulopathy is delayed after ingestion and overt bleeding is later still; the median latency in the pooled published series

was four days, with a range extending to 30 days.[1] In a retrospective study of 26 children the median incubation period was six days and extended as far as 63 days.[8] Mucocutaneous bleeding predominates, and the deep confluent lesions illustrated in Figure 1A to 1C are its typical cutaneous expression. Ecchymoses, epistaxis, gingival bleeding, haematuria and gastrointestinal blood loss are the usual complaints, and haematuria is the single most frequently reported symptom.[1] Beyond this common core, superwarfarin poisoning has an unsettling capacity to imitate almost any acute surgical or neurological illness. Published presentations include intraspinal haematoma,[9] massive intramuscular haematoma without any external bleeding,[10] haematemesis with oesophageal haematoma,[11] acute epiglottitis,[12] convulsive status epilepticus from intracranial bleeding,[13] spontaneous cerebral haemorrhage,[14] and acute kidney injury attributable to bleeding into the urinary tract and to coagulopathy itself.[15,16]

Diagnosis is difficult for three converging reasons. First, the exposure history is frequently absent, concealed or genuinely unknown to the patient. Covert administration has been documented, and in one report the diagnosis of difenacoum poisoning was reached only after protracted investigation and remained hard to follow up.[17] In another, a man with two weeks of oral bleeding and two days of haematuria was found to have high plasma brodifacoum concentrations acquired through consumption of rodent meat while travelling.[18] Second, quantitative plasma assays for rodenticides are not routinely available and, where they exist, turnaround times of days to weeks make them useless for the first therapeutic decisions.[4] Reports repeatedly emphasise that the diagnosis remains a clinical and exclusionary one at the moment when treatment must start.[19,20] Third, and least appreciated, the very severity of the coagulopathy disables the measurement: in a recently reported bromadiolone cluster the index patient had a PT longer than 180 seconds and an INR that was simply unmeasurable.[21] A number that cannot be measured cannot be trended, and treatment decisions made against a censored value are decisions made partly blind.

Superwarfarin exposure during pregnancy occupies an almost empty space in the literature. Whereas the fetal consequences of therapeutic warfarin are well characterised, encompassing embryopathy, growth restriction and catastrophic intracranial bleeding,[22,23] the published experience with second-generation rodenticides in pregnancy is confined to isolated reports. The most informative is that of a woman who developed unexplained epistaxis and haematuria at 34 weeks and 3 days of gestation and was ultimately shown to have brodifacoum poisoning; fetal ultrasonography demonstrated extensive intracranial haemorrhage, and intrauterine fetal demise followed.[24] That report establishes transplacental passage and fetal harm but describes a late-gestation exposure in a pregnancy that was already known. The reason the fetus is disproportionately vulnerable is that the immature fetal liver has a limited capacity for γ-carboxylation, so that fetal haemostasis is compromised at maternal exposures the mother herself tolerates. What happens when the toxicant is on board before the pregnancy is even recognised, and when the maternal coagulopathy relapses across the first and second trimesters, has not been described.

The novelty of the present report lies in four features that have not previously been assembled in a single patient. The pregnancy was discovered incidentally during an ultrasound examination performed to exclude intraperitoneal haemorrhage, so that the coagulopathy led to the diagnosis of pregnancy rather than the reverse. The coagulopathy relapsed after an apparently adequate 28-day inpatient course, and the serial data show that the INR had in fact never normalised at the time of the first discharge. A rebound to an INR of 21.6 occurred on the tenth day of the second admission despite continuous parenteral vitamin K_1, illustrating in a single patient the disproportion between antidote supply and toxicant reservoir. Finally, a complete 16-point, eight-month record of INR-guided oral vitamin K_1 titration is available, spanning the loss of the pregnancy and continuing to withdrawal of therapy. The aim of this report is therefore to describe the clinical course, laboratory trajectory and long-term antidotal management of superwarfarin intoxication complicating an unrecognised early pregnancy, and to argue that the therapeutic target in this condition is the hepatic toxicant reservoir rather than the INR of the day, and that maternal biochemical rescue must not be equated with fetal safety.

2. Case Presentation

Initial presentation

An 18-year-old Balinese woman presented to the emergency department with bluish bruising over her whole body that had appeared two weeks earlier and had worsened over the preceding few days. She had also had bleeding from the gums, which had settled spontaneously, and she had passed reddish urine on the morning of admission. She was unmarried and living with her family; this social detail is recorded because it is relevant both to the initial non-disclosure of the ingestion and to the fact that the pregnancy was neither suspected nor sought at any point during the first admission. She denied melaena. There had been no previous episodes of similar bruising, no history of anticoagulant use of any kind, no previous transfusion, and no family history of a bleeding disorder. The demographic, clinical and baseline laboratory data at first presentation are summarised in Table 1.

On examination, as detailed in Table 1, she was fully alert with a Glasgow Coma Scale score of E4V5M6. Blood pressure was 100/70 mmHg, pulse rate 87 beats per minute, temperature 36.7 °C, respiratory rate 20 breaths per minute and peripheral oxygen saturation 97 % on room air. The conjunctivae were pale, and extensive ecchymoses were present over both upper and lower limbs. Examination of the chest and abdomen was unremarkable and no organomegaly was detected. The distribution and morphology of the cutaneous lesions are shown in Figure 1; the confluent, deeply violaceous plaques over the dorsum of the hand and the fingers in Figure 1A, the large single ecchymosis over the antecubital fossa in Figure 1B and the mottled ecchymoses extending from the leg onto the dorsum of the foot in Figure 1C are characteristic of a plasmatic rather than a platelet-type bleeding disorder.

The complete blood count showed anaemia with a haemoglobin concentration of 6.0 g/dL together with leukocytosis, while the platelet count lay within the reference interval; the abnormal values are flagged in bold red in Table 1 against the reference intervals used for their interpretation. Hepatic and renal function tests were normal. The PT, APTT and INR were all prolonged, the PT and the INR exceeding the upper analytical limits of the laboratory, which reports these assays as ">90 s" and ">6" respectively. The peripheral blood film showed a normochromic normocytic anaemia with neutrophilic leukocytosis and no abnormal cells. She was afebrile and had no localising symptoms or signs of infection, and no source was identified; the neutrophilic leukocytosis was attributed to the ongoing haemorrhage and to resorbing soft-tissue haematoma rather than to sepsis, and no antimicrobial therapy was given. The record does not document whether a pregnancy test was performed at this presentation. Because the gestational age at this point was approximately 4 weeks and 3 days, both possibilities are instructive and are taken up in the Discussion: a test may

never have been requested, or one may have been requested and been negative at a gestation at which a urinary assay can legitimately be so. The chest radiograph was normal. The combination of a severe isolated prolongation of both plasmatic clotting times with a normal platelet count, normal liver function and no history of anticoagulant use narrowed the differential diagnosis sharply, and an acquired deficiency of the vitamin K-dependent factors was considered the most probable mechanism. The complete sequence of events from ingestion to withdrawal of therapy is presented as a visual timeline in Figure 2. For orientation, and using the sonographic dating described below, the gestational ages at the key events were approximately 2 weeks at the ingestion, 4 weeks and 3 days at this first presentation, 8 weeks and 2 days at the first discharge, 11 weeks and 1 day at readmission, 11 weeks and 3 days at the dating scan, and 18 weeks at the loss of the pregnancy.

Table 1. Demographic, clinical and baseline laboratory profile of the patient at first presentation.

Domain	Parameter	Finding	Reference interval
Demographics	Age	18 years	–
	Sex	Female	–
	Ethnicity	Balinese	–
	Marital status	Unmarried	–
History	Presenting complaint	Bluish bruising over the whole body for 2 weeks, worsening	–
	Mucosal bleeding	Gum bleeding (resolved); reddish urine on the day of admission	–
	Melaena	Denied	–
	Previous similar episodes	None	–
	Anticoagulant or antiplatelet use	None	–
	Previous transfusion	None	–
	Family history of bleeding	None	–
Vital signs and examination	Level of consciousness	Compos mentis, GCS E4V5M6	E4V5M6
	Blood pressure	100/70 mmHg	90–120 / 60–80 mmHg
	Pulse rate	87 beats/min	60–100 beats/min
	Respiratory rate	20 breaths/min	12–20 breaths/min
	Axillary temperature	36.7 °C	36.5–37.5 °C
	Oxygen saturation (room air)	97 %	≥95 %
	Conjunctivae	Pale (anaemic)*	Not pale
	Skin	Ecchymoses over both upper and lower limbs*	No lesions
	Chest and abdomen	Unremarkable; no organomegaly	Normal
Haematology	Haemoglobin	6.0 g/dL*	12.0–16.0 g/dL
	Leukocyte count	Leukocytosis (absolute value not recorded)*	4.0–11.0 × 10^3/µL
	Platelet count	Within reference interval	150–450 × 10^3/µL
	Peripheral blood film	Normochromic normocytic anaemia; neutrophilic leukocytosis*	Normal morphology
Haemostasis	Prothrombin time	>90 s (above analytical ceiling)*	10–14 s
	Activated partial thromboplastin time	133.4 s*	24–36 s
	International normalised ratio	>6 (above analytical ceiling)*	0.9–1.2
Organ function	Liver function tests	Normal	Within reference interval
	Renal function tests	Normal	Within reference interval
Imaging	Chest radiograph	Normal	Normal

**Notes: GCS, Glasgow Coma Scale. Values outside the reference interval are shown in bold red. The laboratory reported the prothrombin time and international normalised ratio at the upper limit of the assay; the true values are unknown and are at least as abnormal as shown. Reference intervals are the conventional adult intervals used for clinical interpretation at the reporting laboratory.*

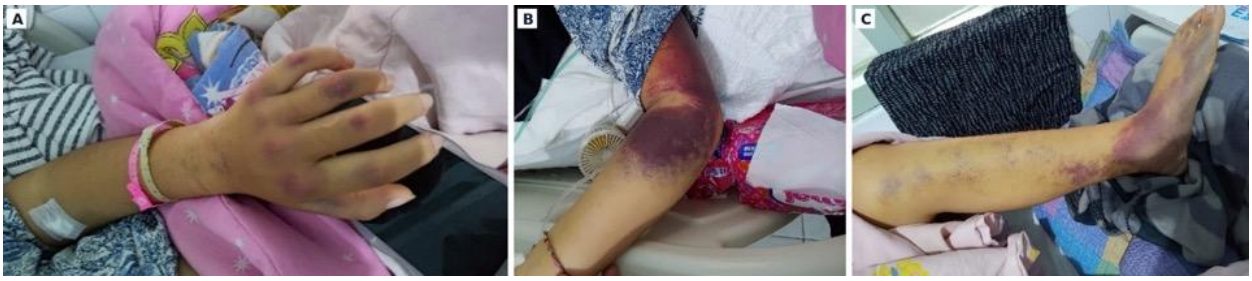

Figure 1. Spontaneous ecchymoses at first presentation. (A) Confluent violaceous ecchymoses over the dorsum of the right hand and fingers. (B) A single large ecchymosis over the antecubital fossa and proximal forearm. (C) Mottled ecchymoses extending along the lower leg onto the dorsum of the foot. The lesions are deep, confluent and palpable rather than petechial, a distribution characteristic of a plasmatic (coagulation factor) rather than a platelet-type bleeding disorder.

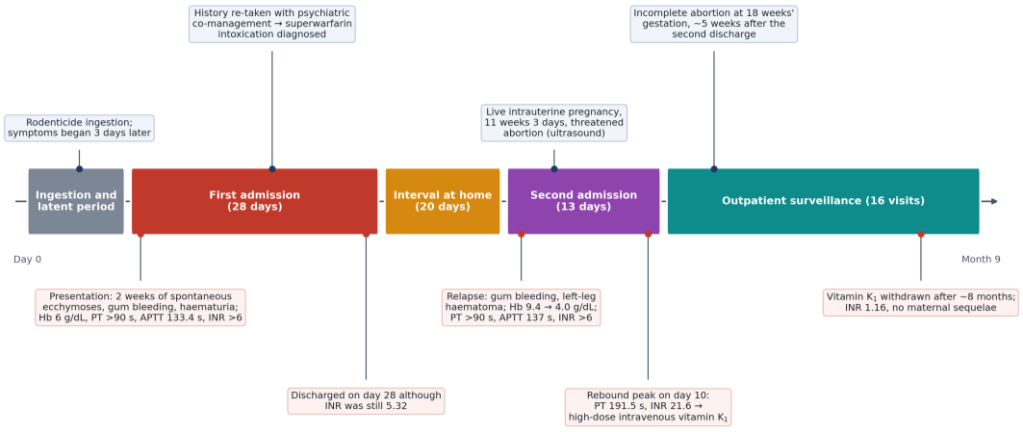

Figure 2. Visual timeline of the clinical course from rodenticide ingestion to withdrawal of vitamin K_1 approximately nine months later. Coloured bands mark the five phases of the illness and are not drawn to a linear time scale. Callouts above the axis record diagnostic and obstetric events; callouts below the axis record haemostatic events. The 20-day interval at home separates two episodes of severe coagulopathy. The incomplete abortion at 18 weeks' gestation occurred approximately five weeks after the second discharge, while maternal coagulation was improving under outpatient vitamin K_1 therapy.

Disclosure of exposure and first admission

After the history had been taken again in more detail, and with the patient under joint care with the psychiatry service, she disclosed that she had swallowed rat poison three days before the bruising appeared. On the basis of the history, physical findings and laboratory results, superwarfarin intoxication was diagnosed clinically. It should be stated plainly that the diagnosis rests on three grounds and on no laboratory confirmation of the toxicant: a disclosed ingestion with a latency consistent with the published range, a biochemical pattern of isolated vitamin K-dependent factor deficiency, and — established only in retrospect — a duration of vitamin K_1 dependence that no other plausible aetiology would produce. No plasma rodenticide assay was available. She was treated with transfusion of packed red cells at one to two units per day until the haemoglobin concentration exceeded 10 g/dL, fresh frozen plasma at five units per day, and vitamin K_1 2 mg every eight hours by the intramuscular route, with daily monitoring of the coagulation profile. Two features of this initial regimen are commented on in the Discussion: the intramuscular route was used at a time when the PT was above the analytical ceiling, and the dose was an order of magnitude below the maintenance requirements reported for this poisoning. No injection-site haematoma was documented, although the record is not explicit on the point.

The serial haemostatic values and the haemostatic support given on each sampling day during the first admission are set out in Table 2 and displayed graphically in Figure 3A. Three features of that trajectory deserve emphasis. The PT and INR remained at or above the analytical ceiling of the laboratory on days 1, 3, 6, 10 and 13, so that the true degree of coagulopathy during the first fortnight is unknown and can only be described as at least as severe as the censored values shown. The APTT, which was not censored, fell from 133.4 seconds on day 1 to 87.7 seconds by day 6 and to 41.0 seconds by day 15, indicating a real but incomplete response. Most importantly, correction was transient and repeatedly lost. The pattern is most easily seen at the two points where the PT falls below the analytical ceiling between censored neighbours: 50.0 seconds on day 8 with an INR of 5.01, lying between values above the ceiling on days 6 and 10, and 50.05 seconds on day 24 immediately after the transfusion given that day. Both are the expected signature of a short-lived correction by transfused factors superimposed on an uncorrected underlying deficiency rather than laboratory error. After each episode of plasma support the values improved and then deteriorated again, so that on day 17 and day 18 the INR had risen to 10.60, and on day 21 the PT reached its highest recorded value for that admission of 104 seconds. The intramuscular vitamin K_1 dose was increased from 2 mg to 10 mg every eight hours on day 21 in response, and further fresh frozen plasma was given on days 24 and 26.

Table 2. Serial haemostatic parameters and haemostatic support during the first admission (days 1–28)

Day	PT (s)	APTT (s)	INR	Haemostatic support recorded on that day
1	>90*	133.4*	>6*	FFP 5 units/day + vitamin K1 2 mg q8h IM; PRC 1–2 units/day until Hb >10 g/dL
3	>90*	88.6*	>6*	Continued
6	>90*	87.7*	>6*	FFP 5 units
8	50.0*	51.2*	5.01*	Continued
10	>90*	64.8*	>6*	FFP 10 units
13	>90*	66.9*	>6*	FFP 4 units
15	67.0*	41.0*	6.90*	Continued
17	99.6*	39.8*	10.60*	FFP 5 units
18	99.6*	39.8*	10.60*	FFP 5 units
21	104.0*	60.9*	8.00*	Vitamin K1 increased to 10 mg q8h IM
24	50.05*	40.6*	5.06*	FFP 5 units
26	61.0*	55.7*	6.22*	FFP 5 units
28	64.8*	44.1*	5.32*	Discharged on vitamin K1 10 mg q8h orally

Notes: PT, prothrombin time; APTT, activated partial thromboplastin time; INR, international normalised ratio; FFP, fresh frozen plasma; PRC, packed red cells; Hb, haemoglobin; IM, intramuscular; q8h, every eight hours. All values shown were outside the reference interval. Reported at the upper analytical limit of the assay; the true value is unknown. The value of 50.05 s recorded on day 24 is reproduced exactly as documented and its precision reflects the source record rather than the analytical resolution of the assay.

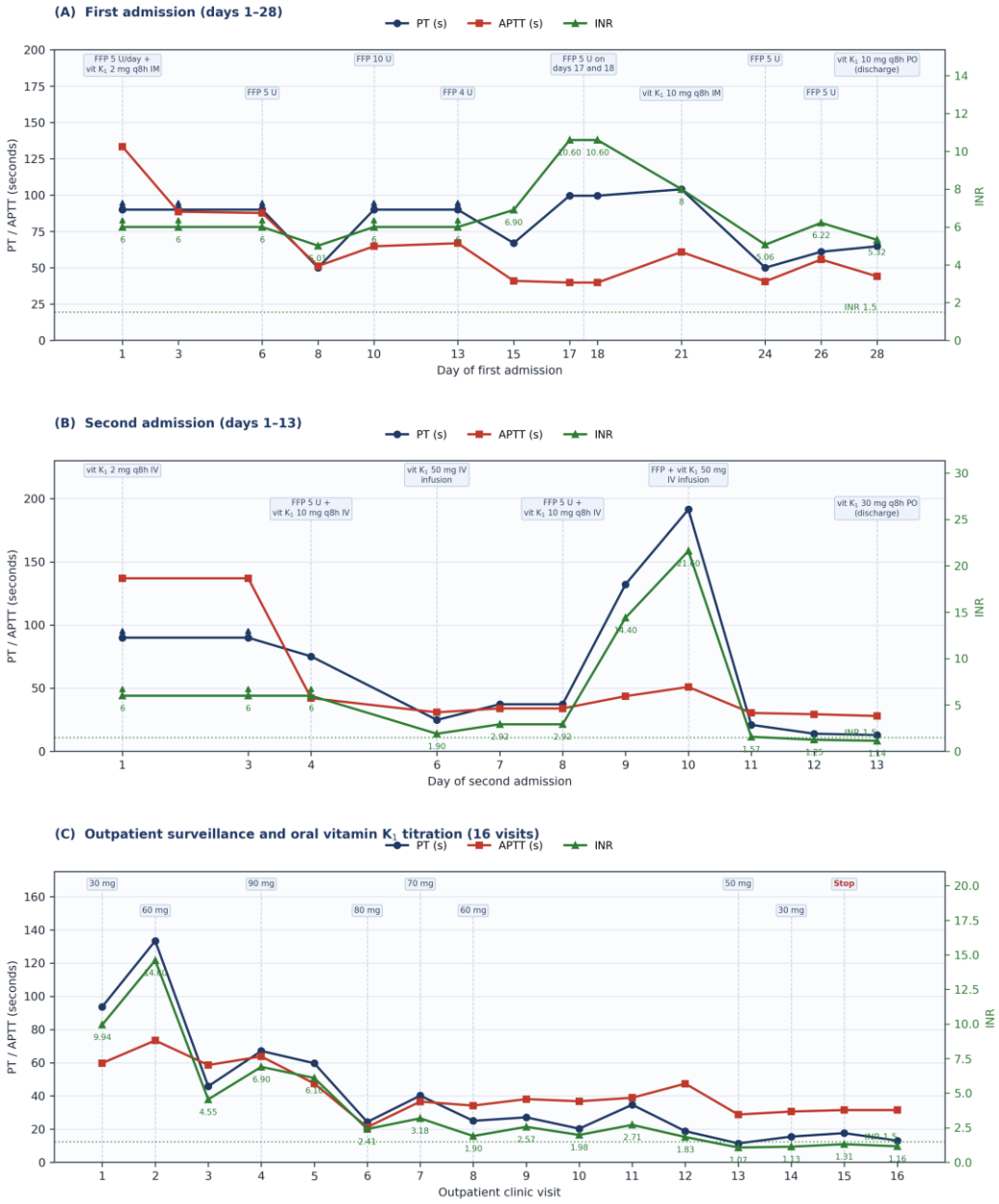

Figure 3. Serial haemostatic trajectories with concurrent treatment. (A) First admission, days 1–28. (B) Second admission, days 1–13. (C) Outpatient surveillance across 16 clinic visits with the oral vitamin K_1 dose as charted. Prothrombin time and activated partial thromboplastin time are read against the left axis and the international normalised ratio against the right axis; the dotted horizontal line marks an international normalised ratio of 1.5. Upward arrows denote values reported at the upper analytical limit of the assay (prothrombin time >90 s, international normalised ratio >6), whose true magnitude is unknown. Note that correction in panel A was never complete, that the international normalised ratio at discharge on day 28 was 5.32, and that in panel B the international normalised ratio rebounded from 2.92 on day 8 to 21.60 on day 10 despite continuous parenteral vitamin K_1. The visit axis in panel C is ordinal rather than linear in time, and the pregnancy loss is therefore not marked on it. In panel A the fresh frozen plasma given on days 17 and 18 is shown as a single merged annotation to avoid overlap; the individual episodes are listed separately in Table 2.

After approximately one month of treatment the patient's symptoms had settled and she was discharged on day 28 on oral vitamin K_1 10 mg every eight hours with instructions to attend the outpatient clinic for regular review. It is important to record precisely what her coagulation status was at that moment, because the subsequent course turns on it: on day 28 the PT was 64.8 seconds, the APTT 44.1 seconds and the INR 5.32 (Table 2, final row; Figure 3A). Two things should be said about that value. It was not an off-treatment measurement: from day 21 onwards she had been receiving vitamin K_1 10 mg every eight hours, so 5.32 was the INR achieved on 30 mg of antidote per day. And clinical haemostasis had been achieved — she had stopped bleeding — but the laboratory had not returned to normal at any point during that admission.

Second admission and the incidental diagnosis of pregnancy

Twenty days after discharge the patient was readmitted with spontaneous gum bleeding together with bruising and swelling of the left leg. The swelling was painless and did not improve with rest. Laboratory tests on readmission showed anaemia with a haemoglobin concentration of 9.4 g/dL, leukocytosis with neutrophil predominance, and a severely prolonged coagulation profile with a PT greater than 90

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seconds, an APTT of 137.3 seconds and an INR greater than 6, while renal and hepatic function remained normal. On the second day of this admission, at approximately 11 weeks and 2 days of gestation although the pregnancy was not yet known, she became visibly more pale and the haemoglobin concentration fell abruptly to 4.0 g/dL. She was transfused, but the record does not state the number of units given on this occasion, the transfusion threshold or target applied, or whether any assessment of the fetus was made at that moment; the pregnancy was identified the following day. The serial values and the corresponding treatment for this admission are given in Table 3 and plotted in Figure 3B.

Because no external source could account for a fall of that magnitude, abdominal ultrasonography was performed to look for intraperitoneal bleeding. No free fluid was demonstrated in the hepatorenal, splenorenal or paracolic spaces. The examination instead revealed a single live intrauterine fetus. The patient was referred to the obstetric service, and transabdominal sonography demonstrated a gestational sac with a crown–rump length of 4.67 cm, corresponding to a gestational age of 11 weeks and 3 days, with a positive fetal heart beat. She was diagnosed as gravida 1, para 0, at 11 weeks and 3 days with a single live fetus and threatened abortion. It should be recorded that the notes carry this diagnostic label without describing the finding that ordinarily defines it: vaginal bleeding is nowhere documented during this admission, the haemorrhagic manifestations being gingival bleeding, ecchymoses and a left-leg haematoma, and no assessment of the cervical os is recorded. The label is reproduced here as it was made by the obstetric service, and its basis is examined in the Discussion. The relevant sonographic images are presented in Figure 4; the gestational sac containing a fetus of appropriate size for dates is seen within the uterine cavity in Figure 4A, and colour and pulsed-wave Doppler interrogation confirming fetal cardiac activity is shown in Figure 4B. Treatment was intensified to fresh frozen plasma together with intravenous vitamin K_1 10 mg every eight hours. Beyond this, the record contains no account of specifically obstetric management: no progestogen or activity-restriction advice is documented, and no interval fetal sonography was performed between the dating scan and the eventual loss.

Table 3. Serial haemostatic parameters and vitamin K_1 regimen during the second admission (days 1–13)

Day	PT (s)	APTT (s)	INR	Treatment recorded on that day
1	>90*	137.0*	>6*	Vitamin K1 2 mg q8h IV
3	>90*	137.0*	>6*	Continued
4	75.2*	42.2*	>6*	FFP 5 units + vitamin K1 10 mg q8h IV (pregnancy identified)
6	24.9*	30.9	1.90*	Vitamin K1 50 mg IV infusion
7	37.3*	34.0	2.92*	Continued
8	37.3*	34.0	2.92*	FFP 5 units + vitamin K1 10 mg q8h IV
9	132.2*	43.7*	14.40*	Vitamin K1 10 mg q8h IV
10	191.5*	51.0*	21.60*	FFP + vitamin K1 50 mg in 100 mL NaCl 0.9 % IV over 1 h
11	21.0*	30.4	1.57*	Continued
12	14.0	29.4	1.25*	Continued
13	12.9	28.0	1.14	Discharged on vitamin K1 30 mg q8h orally

**Notes: PT, prothrombin time; APTT, activated partial thromboplastin time; INR, international normalised ratio; FFP, fresh frozen plasma; IV, intravenous; q8h, every eight hours. Values outside the reference interval (PT 10–14 s, APTT 24–36 s, INR 0.9–1.2) are shown in * mark. \Reported at the upper analytical limit of the assay.*

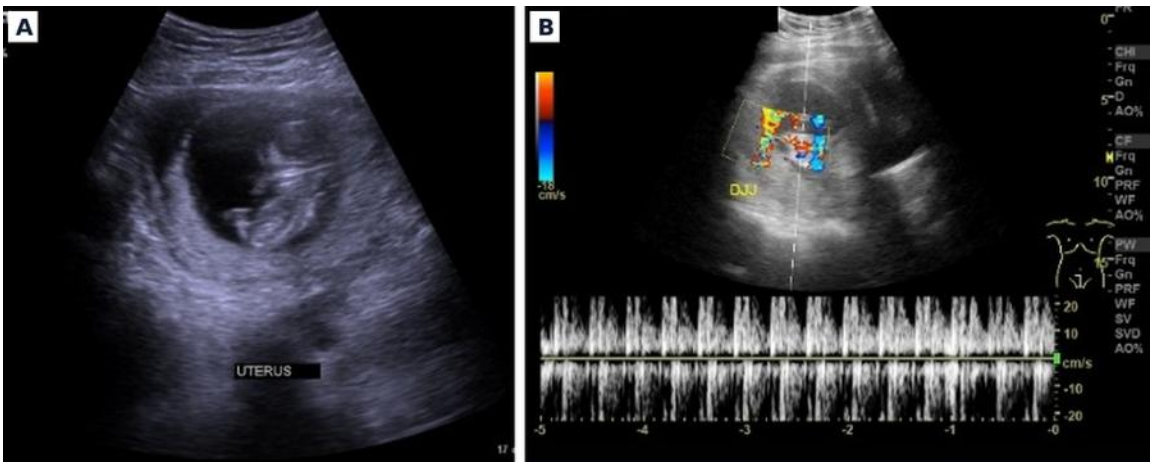

Figure 4. Transabdominal ultrasonography performed to exclude intraperitoneal haemorrhage, which instead demonstrated an unsuspected intrauterine pregnancy. (A) Gestational sac within the uterine cavity containing a single fetus with a crown–rump length of 4.67 cm, corresponding to 11 weeks and 3 days of gestation. (B) Colour and pulsed-wave Doppler interrogation of the fetal heart confirming cardiac activity. All patient identifiers embedded in the original images have been removed.

The haemostatic response during the second admission was biphasic and instructive. As shown in Figure 3B, the initial response to intravenous vitamin K_1 and plasma was brisk: the INR fell from a censored value of more than 6 on days 1 to 4 to 1.90 on day 6 after a 50 mg intravenous infusion of vitamin K_1, the PT falling from more than 90 seconds to 24.9 seconds and the APTT from 137 seconds to 30.9 seconds over the same interval. Correction was not sustained. As shown in Figure 3B and set out row by row in Table 3, despite continued parenteral vitamin K_1 the INR rose again to 2.92 on days 7 and 8, and then abruptly to 14.40 on day 9 and 21.60 on day 10, the PT reaching 191.5 seconds — the highest value recorded at any point in this patient's illness. These two values were not repeated, and the record does not state

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whether the sample was drawn from a peripheral vein or from the line through which plasma and vitamin K_1 were being infused; both possibilities are considered in the limitations. Further fresh frozen plasma was given together with vitamin K_1 50 mg in 100 mL of 0.9 % sodium chloride infused over one hour. The response to this escalation was rapid and, on this occasion, durable: by day 11 the INR had fallen to 1.57, by day 12 to 1.25 and by day 13 to 1.14, with the PT returning to 12.9 seconds and the APTT to 28.0 seconds.

Outpatient course and outcome

The patient was discharged after two weeks of this second admission on oral vitamin K_1 30 mg every eight hours. She attended the haematology and medical oncology outpatient clinic weekly for a complete blood count and coagulation profile. The oral vitamin K_1 dose was adjusted at each visit against the measured INR. The sixteen paired observations of coagulation status and prescribed dose are tabulated in Table 4 and plotted in Figure 3C.

Table 4. Outpatient haemostatic surveillance and titration of oral vitamin K_1 over 16 clinic visits

Clinic visit	PT (s)	APTT (s)	INR	Oral vitamin K1 dose as charted	Comment
1	93.7*	59.8*	9.94*	30 mg	First visit after second discharge
2	133.4*	73.4*	14.60*	60 mg	Peak outpatient coagulopathy; dose escalated
3	45.8*	58.6*	4.55*	Unchanged	
4	67.1*	63.8*	6.90*	90 mg	Highest dose recorded
5	59.7*	47.5*	6.10*	Unchanged	
6	24.3*	21.2*	2.41*	80 mg	Sustained improvement begins; taper started
7	40.2*	36.6*	3.18*	70 mg	
8	25.0*	34.2	1.90*	60 mg	
9	27.1*	38.1*	2.57*	Unchanged	
10	20.3*	36.8*	1.98*	Unchanged	
11	34.7*	38.9*	2.71*	Unchanged	
12	18.8*	47.4*	1.83*	Unchanged	
13	11.4	28.8	1.07	50 mg	Coagulation profile fully normal
14	15.5*	30.6	1.13	30 mg	
15	17.6*	31.5	1.31*	Stopped	Vitamin K1 withdrawn after ~8 months
16	13.1	31.5	1.16	None	Normal profile without antidote

*Notes: PT, prothrombin time; APTT, activated partial thromboplastin time; INR, international normalised ratio. Values outside the reference interval (PT 10–14 s, APTT 24–36 s, INR 0.9–1.2) are shown in *. Doses are reproduced exactly as documented in the outpatient record; the discharge prescription at the end of the second admission was 30 mg every eight hours. The incomplete abortion at 18 weeks' gestation occurred during this outpatient period but has not been assigned to a numbered visit, because clinic attendance intervals were not uniform and calendar dates were not recorded against each visit.*

The pattern shown in Figure 3C recapitulates the inpatient experience on a longer time base. At the first clinic visit the INR was 9.94 and at the second it had risen to 14.60 with a PT of 133.4 seconds, so that the recorded dose was increased from 30 mg to 60 mg and subsequently to 90 mg by the fourth visit. From the fifth visit onward the trajectory was one of progressive and sustained improvement, the INR falling below 2.5 by the sixth visit and remaining below 3.0 thereafter apart from transient values of 3.18 and 2.71 at the seventh and eleventh visits. As the coagulation profile improved the dose was reduced in steps to 80 mg, 70 mg, 60 mg and 50 mg, then to 30 mg by the fourteenth visit, and was discontinued altogether at the fifteenth visit, approximately eight months after the index admission, the patient having been followed for approximately nine months in total. The final two observations after withdrawal of the antidote, an INR of 1.31 at the fifteenth visit and 1.16 at the sixteenth (Table 4, final two rows; Figure 3C, right-hand end of the green trace), are the critical ones, because they demonstrate for the first time in this patient's illness a normal coagulation profile that was not being sustained by exogenous vitamin K_1. The calendar interval between those two visits is not recorded, which limits the reassurance they provide; on the weekly schedule described at discharge it would have been about one week. No further bleeding occurred and no residual sequelae were identified. The record does not document whether psychiatric follow-up continued beyond the second admission, whether contraception was discussed during the many months in which she carried a hepatic burden of a fetotoxic compound, or what pre-pregnancy counselling she received after the loss.

The obstetric outcome was less favourable. Although maternal haemostasis was progressively restored, the pregnancy could not be maintained and ended as a spontaneous incomplete abortion at 18 weeks of gestation. Because 18 weeks of gestation is 6 weeks and 4 days beyond the dating scan performed on the third day of the second admission, the loss occurred approximately five weeks after that discharge, during the outpatient surveillance period and while maternal coagulation was steadily improving. It has deliberately not been anchored to a numbered clinic visit in Table 4 or Figure 3C, for the reason given in the limitations.

3. Discussion

Why the diagnosis took two weeks

This patient bled for a fortnight before she reached hospital and disclosed her exposure only after the history had been taken a second time in a different clinical context. That sequence is the rule rather than the exception. In the pooled analysis of 88 published human anticoagulant rodenticide poisonings the median latency between exposure and presentation was four days and extended to 30 days, and multi-organ bleeding with haematuria as the commonest single symptom was the dominant picture.[1] The delay has a straightforward pharmacological explanation. Superwarfarins do not consume clotting factors; they prevent the regeneration of the reduced vitamin K required for their post-translational γ-carboxylation, so that circulating functional factor levels decline only as existing molecules are cleared. Because factor VII has the shortest half-life the PT moves first, and clinically evident bleeding follows several days later still. A patient who ingests a rodenticide therefore

feels entirely well throughout the period in which the diagnosis would be easiest to make.

The second obstacle is that the exposure history is often unavailable. Deliberate self-poisoning is under-reported for obvious reasons, and there are well-documented instances of covert administration in which the diagnosis of difenacoum poisoning was reached only after prolonged investigation and in which follow-up itself proved difficult.[17] Exposure may also be entirely unsuspected by the patient: a 37-year-old man with two weeks of oral bleeding and two days of haematuria was eventually shown to have high plasma brodifacoum concentrations acquired by eating rodent meat during travel, a route no history-taking algorithm would generate.[18] The practical lesson from this case is that the psychiatric consultation was not an ancillary service but a diagnostic intervention: the exposure was disclosed only once the patient was interviewed in a setting that made disclosure safe.

The third obstacle is analytical. Quantitative plasma rodenticide measurement by liquid chromatography–tandem mass spectrometry is the only method that identifies the offending compound, estimates the ingested dose and, most usefully, indicates when antidotal therapy may safely stop; yet it was unavailable in the hospitals managing the largest reported outbreak of this poisoning, and an explicit action plan has had to be proposed to make it accessible.[4] Where such assays are available they change management materially: in a recently reported bromadiolone cluster the index patient's serum concentration was 150 ng/mL and those of two household members were 250 and 450 ng/mL, allowing the source to be traced and public health action to be taken.[21] In our patient no rodenticide assay was performed at any stage, and neither the specific compound nor its plasma concentration is known. This limitation is shared by most reports from settings comparable to ours and is discussed further below.

The biochemical signature and its differential diagnosis

The mechanism that produces the laboratory picture is summarised in Figure 5. Reduced vitamin K hydroquinone is the obligatory cofactor for γ-glutamyl carboxylase, which converts specific glutamate residues in prothrombin and in factors VII, IX and X, and in proteins C, S and Z, into γ-carboxyglutamate. Those residues create the high-affinity calcium-binding sites that allow these proteins to dock onto anionic phospholipid membranes at sites of injury.[25] During carboxylation the hydroquinone is oxidised to vitamin K 2,3-epoxide, which must be reduced back to the quinone and then to the hydroquinone for the cycle to continue, a reduction catalysed principally by vitamin K epoxide reductase complex subunit 1. Superwarfarins inhibit this enzyme, so that epoxide accumulates and functionally decarboxylated proteins are secreted. Because tissue stores of vitamin K are small, this deficiency state can develop within days.[25] A second, quinone reductase pathway mediated by NAD(P)H:quinone oxidoreductase 1, drawn on the left of Figure 5, can regenerate the hydroquinone independently of the epoxide reductase; it is quantitatively minor under physiological conditions but comparatively resistant to inhibition, and it is this bypass, shown in green in Figure 5, that high-dose vitamin K_1 exploits. The therapeutic logic of giving tens of milligrams of vitamin K_1 daily for months, rather than the 10 mg used to reverse warfarin, follows directly from that pharmacology.

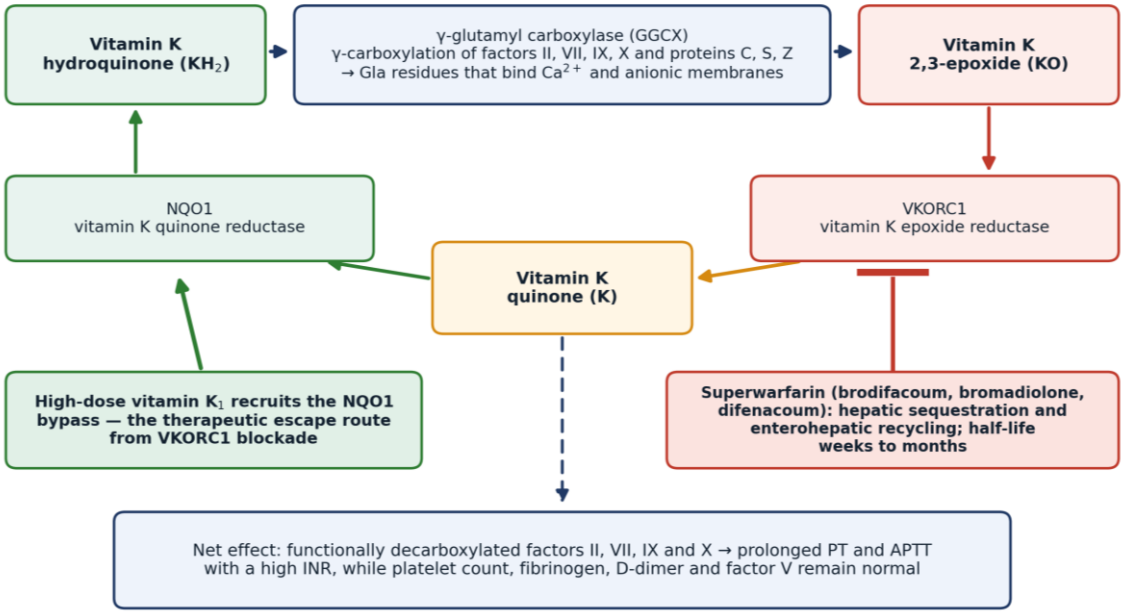

Figure 5. The hepatic vitamin K cycle, its blockade by superwarfarins and the NAD(P)H:quinone oxidoreductase 1 bypass. Reduced vitamin K hydroquinone is the cofactor for γ-glutamyl carboxylase, which carboxylates factors II, VII, IX and X and proteins C, S and Z; the hydroquinone is oxidised to the 2,3-epoxide in the process and must be recycled by vitamin K epoxide reductase complex subunit 1. Superwarfarins inhibit that enzyme (red bar) and, because they are sequestered in the liver and undergo enterohepatic recirculation, sustain the blockade for weeks to months. High-dose vitamin K_1 acts through the quinone reductase pathway, which is comparatively resistant to inhibition. The net biochemical result is a grossly prolonged prothrombin time and activated partial thromboplastin time with a preserved platelet count, fibrinogen, D-dimer and factor V. This is an original schematic prepared by the authors.

The resulting laboratory phenotype is highly specific if it is read as a pattern rather than as a list of abnormal values: both plasmatic clotting times are prolonged, often grossly, while the platelet count, fibrinogen and factor V are preserved. In the paediatric series of 26 children every patient had a prolonged PT, a raised INR, a prolonged APTT and reduced activity of the vitamin K-dependent factors.[8] The principal alternative diagnoses in a young woman are set out in Table 5. Disseminated intravascular coagulation, which is the diagnosis most often considered in an obstetric context, is excluded here by the normal platelet count, by the absence of any recognised trigger at first presentation, and above all by

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a time course of eight months that is wholly incompatible with a consumptive process; it should be stated that fibrinogen and D-dimer were not measured in this patient, so the exclusion does not rest on them. In a retrospective study of 163 fetal deaths, probable disseminated intravascular coagulation was present in only 7.4 % of women and confirmed disseminated intravascular coagulation in 1.8 %, and where the initial coagulation screen was normal no subsequent test became abnormal.[26] Liver disease is excluded by normal transaminases and preserved factor V. Congenital haemophilia does not prolong the PT and would not present for the first time at the age of 18 in a woman with no family history. Acquired haemophilia A, as a disease entity, produces an isolated prolongation of the APTT that fails to correct on mixing; no mixing study was performed in this patient, and acquired haemophilia was excluded here on the grossly prolonged PT alone. Vitamin K deficiency from other causes remains the closest mimic, as the third row of Table 5 makes clear, and it should be noted that a prolonged PT in hyperemesis gravidarum has been shown to be a usable indicator of vitamin K deficiency in pregnancy, so that the biochemical pattern alone does not distinguish nutritional deficiency from poisoning.[27] The discriminating features are the magnitude and, above all, the durability of the abnormality.

Table 5. Differential diagnosis of an acquired prolongation of both the prothrombin time and the activated partial thromboplastin time in a young pregnant woman

Condition	PT	APTT	Platelets	Fibrinogen / D-dimer	Mixing study	Discriminating feature	Fit with this case
Superwarfarin (long-acting anticoagulant rodenticide) poisoning	Markedly prolonged	Prolonged	Normal	Normal	Corrects	Isolated deficiency of factors II, VII, IX and X with normal factor V; abnormality persists for months	Complete
Warfarin ingestion	Prolonged	Prolonged	Normal	Normal	Corrects	Rapid and durable response to vitamin K1 and plasma; relapse within 12–16 h does not occur	Excluded — no access to warfarin; response was not durable
Dietary or malabsorptive vitamin K deficiency	Prolonged	Prolonged or normal	Normal	Normal	Corrects	Underlying malnutrition, cholestasis, antibiotic use or hyperemesis; corrects within 24–48 h of vitamin K1	Excluded — normal nutrition, no cholestasis, no durable correction
Advanced liver disease	Prolonged	Prolonged	Often low	Fibrinogen low	Partial correction	Abnormal transaminases and bilirubin; factor V also reduced; stigmata of chronic liver disease	Excluded — normal liver function tests
Disseminated intravascular coagulation	Prolonged	Prolonged	Low*	Fibrinogen low, D-dimer high*	Variable	Consumptive picture with thrombocytopenia; obstetric triggers include abruption, fetal death, sepsis	Excluded on the normal platelet count, the absence of a trigger and an eight-month course
Congenital haemophilia A or B	Normal	Prolonged	Normal	Normal	Corrects	Lifelong history, male predominance, family history, isolated factor VIII or IX deficiency	Excluded — normal PT, no lifelong or family history
Acquired haemophilia A	Normal	Prolonged	Normal	Normal	Does not correct*	Autoantibody to factor VIII, often postpartum or autoimmune; inhibitor demonstrable	Excluded — PT also grossly prolonged
Heparin exposure	Normal or mildly prolonged	Prolonged	Normal or low	Normal	Corrects with protamine	Recent hospital or catheter exposure; thrombin time prolonged	Excluded — no prior admission or anticoagulant exposure
HELLP syndrome	Normal or mildly prolonged	Normal or mildly prolonged	Low*	Usually normal until advanced	Corrects	Haemolysis with raised lactate dehydrogenase and transaminases, thrombocytopenia; almost always after 20 weeks	Excluded — normal platelet count and liver function; gestation far too early
Acute fatty liver of pregnancy	Prolonged	Prolonged	Often low*	Fibrinogen low*	Partial correction	Hypoglycaemia, raised transaminases and bilirubin, renal impairment, encephalopathy; third trimester	Excluded — normal liver and renal function; gestation far too early

Notes: PT, prothrombin time; APTT, activated partial thromboplastin time. The PT, APTT, platelet, fibrinogen/D-dimer and mixing-study columns describe the pattern expected for each condition and are not results obtained in this patient; fibrinogen, D-dimer, mixing studies and individual factor assays were not performed. The pattern that identifies vitamin K-dependent factor deficiency is a grossly prolonged PT and APTT with a normal platelet count, normal fibrinogen and D-dimer, correction on mixing, and preserved factor V activity. Individual factor assays and mixing studies were not performed in this patient; the entries in the final column indicate how each alternative was excluded on the available clinical and laboratory grounds.

The censored INR: a measurement problem with clinical consequences

On five of the thirteen sampling days of the first admission, and on three of the eleven days of the second, this patient's PT and INR were reported at the upper limit of the assay rather than as true values. In Figure 3A and Figure 3B these observations are marked with upward arrows, and in Table 2 and Table 3 they are recorded as ">90" and ">6" respectively. The clinical consequence is that between day 1 and day 13 of the first admission the treating team had no way of knowing whether the patient was improving, static or deteriorating on the two variables they were nominally following. This is not a local idiosyncrasy: in the pooled series the reported INR ranged as high as 38.2,[1] and in the bromadiolone cluster the index patient's INR was simply unmeasurable at a PT greater than 180 seconds.[21] Three practical inferences follow. Where the PT is censored, the uncensored APTT carries the trend information and should be followed explicitly, as it was here, falling from 133.4 to 41.0 seconds between days 1 and 15. Where available, direct measurement of the activities of factors II, VII, IX and X is preferable to a saturated global test, as was done in the paediatric series in which factor activities were measured in every patient.[8] And where neither is available, a censored value must be treated as an unknown rather than as a maximum, because the assumption that ">6" means "about 6" systematically underestimates severity.

The first discharge was premature, and the data say so

The pivotal event in this patient's course was not the poisoning but the first discharge, marked on the timeline in Figure 2 at the junction between the first admission and the 20-day interval at home. On day 28 her INR was 5.32, more than four times the upper limit of normal, on a PT of 64.8 seconds and an APTT of 44.1 seconds, as recorded in the final row of Table 2 and shown at the right-hand end of Figure 3A. She was discharged because she had stopped bleeding and because a month of treatment had been completed, and she relapsed 20 days later with a haemoglobin concentration that fell to 4.0 g/dL within 48 hours of readmission. The retrospective inference is uncomfortable but hard to avoid: this was not a new bleeding event superimposed on a

corrected haemostatic system but the uninterrupted continuation of a coagulopathy that had never been corrected.

There is external evidence that the biochemical endpoint matters independently of the clinical one. In a multicentre United Kingdom study of 1,771 patients with major bleeding on vitamin K antagonists, a post-intervention INR above 1.3 was associated with 3.2-fold higher odds of death within 30 days compared with an INR at or below 1.3, and higher doses of vitamin K were more likely to achieve correction than lower ones.[28] That study concerned acute reversal rather than chronic superwarfarin poisoning, and its outcome measure was mortality rather than relapse, so it cannot be transposed directly; but it establishes that residual INR elevation after treatment is a marker of unfinished business rather than an acceptable resting state. In superwarfarin poisoning specifically the expected treatment duration is measured in months, not weeks: in the paediatric series the mean treatment course was 2.82 ± 1.85 months and extended to 10 months,[8] and in a family cluster of three members with brodifacoum poisoning, therapy was continued for approximately five months.[29] Judged against that benchmark a 28-day course terminated at an INR of 5.32 was always likely to fail.

The same principle explains why adherence to prolonged oral therapy after discharge has been singled out as a specific hazard in this poisoning, to the point of prompting an explicit call for action on non-adherence with long-term oral vitamin K_1 regimens.[30] In our patient adherence was not the problem, since the second relapse occurred while she was taking 10 mg every eight hours; but the dose that was adequate to keep her from bleeding was plainly inadequate to keep her INR normal, which is the same failure viewed from the other side.

Rebound on day 10: antidote supply against toxicant reservoir

The most striking single observation in this report is the rebound during the second admission, when the INR rose from 2.92 on day 8 to 14.40 on day 9 and 21.60 on day 10 despite continuous parenteral vitamin K_1 (Figure 3B; Table 3). Two mechanisms plausibly contribute. The first is the pharmacokinetics of the toxicant. Brodifacoum is sequestered in the liver and undergoes enterohepatic recirculation, so that plasma concentrations decline slowly and, when modelled as first-order rather than zero-order clearance, imply substantially longer required treatment durations; on that basis Nosal and colleagues recommended that plasma concentrations be monitored simultaneously with the INR during follow-up, and that concentrations be re-checked after therapy is stopped to confirm that they remain below the 10 ng/mL threshold that the same group has proposed as safe.[31,4] A compartment that continues to release inhibitor into the portal circulation will periodically overwhelm a fixed antidotal dose. The second, and it is the simpler of the two, concerns the pharmacokinetics of the antidote rather than of the toxicant. Vitamin K_1 has a plasma half-life of a few hours, and the correction achieved on day 6 followed a single 50 mg intravenous infusion; between day 6 and day 8 the patient received 10 mg every eight hours, roughly a sixth of that daily amount. On this reading the rebound represents the antidote receding against an unchanged inhibitor burden rather than the inhibitor burden advancing against an unchanged antidote. The two explanations are not mutually exclusive, and nothing in the available data separates them: serial plasma rodenticide concentrations, the only measurement that could have done so, were not obtainable. We therefore state both, and note that the practical conclusion is identical under either — the dose delivered between days 6 and 8 was inadequate, and the two 50 mg infusions given on day 6 and again on day 10 mark the points at which supply finally exceeded demand.

Whichever mechanism dominates, the therapeutic implication is the same and is worth stating plainly: in superwarfarin poisoning a falling INR during treatment does not demonstrate that the toxicant has been eliminated, only that the antidote is currently winning. This reframes the entire management problem as one of estimating and outlasting a reservoir whose size is usually unknown. It also explains the renewed interest in interventions that shrink the reservoir itself rather than compensating for it. In a retrospective analysis of 115 patients with rodenticide poisoning, of whom 24 received haemoperfusion, the rate of blood toxin clearance per unit time appeared faster in the haemoperfusion group, and in the subgroup poisoned with brodifacoum haemoperfusion significantly reduced the duration of vitamin K_1 therapy.[32] That study is retrospective and its groups were markedly unequal in size, so the finding is hypothesis-generating rather than practice-changing, and haemoperfusion was not available to, or considered for, the patient described here. The direction of effect is nevertheless consistent with the reservoir model, and a patient such as ours — young, with a documented rebound and facing months of therapy — is exactly the patient in whom the question deserves to be asked prospectively.

Fresh frozen plasma, prothrombin complex concentrate and the choice we made

This patient received large volumes of fresh frozen plasma: five units per day initially, with further 5- and 10-unit episodes on at least six subsequent occasions across the two admissions (Tables 2 and 3). Plasma was the only factor concentrate available, and in that context the decision was correct. It is nonetheless important to record what the comparative evidence shows, because it explains the transient nature of each correction seen in Figure 3A. In a retrospective analysis of 370 patients drawn from two prospective randomised phase 3b trials, 85.9 % of patients receiving four-factor prothrombin complex concentrate achieved an INR of 1.5 or less 30 minutes after infusion, compared with only 39.1 % of those receiving plasma; and, critically, after plasma administration the mean activity levels of factors II and X remained below 50 % at every time point assessed within three hours, irrespective of the post-infusion INR.[33] Plasma, in other words, may move the number without restoring the factor activity the number is supposed to represent. A comparative study in patients with gastrointestinal bleeding on vitamin K antagonists reached conclusions consistent with this.[34] Where concentrate is available, the PROPER3 randomised trial found effective haemostasis in 87.3 % of patients given a fixed 1,000 IU dose against 89.9 % given weight- and INR-adjusted dosing, with shorter door-to-needle times in the fixed-dose arm, although non-inferiority was not formally demonstrated.[35] The corresponding harms should also be stated. Repeated large-volume plasma transfusion in a young woman carries the risks of transfusion-associated circulatory overload, transfusion-related acute lung injury, allergic reactions and alloimmunisation, the last of which has particular consequences for subsequent pregnancies. It is also worth restating the standard principle that plasma is not indicated for a prolonged clotting time in the absence of bleeding or of a planned invasive procedure; several of the transfusion episodes recorded in Table 2 were given for a laboratory value rather than for a bleeding event. Taken together these data suggest that if prothrombin complex concentrate had been available, a smaller number of more effective corrections might have been achieved, with less volume load in a young anaemic woman who was, unknown to everyone, in the first trimester of pregnancy. What no factor product

does is address the underlying inhibition; that remains the exclusive job of vitamin K_1.

Vitamin K_1 dosing, route and duration

The dose escalation in this patient — from 2 mg every eight hours intramuscularly, to 10 mg every eight hours, to two 50 mg intravenous infusions, and then to a titrated oral regimen documented across 16 clinic visits — is a practical illustration of the principle that dosing in this condition is empirical and driven by response. Several points from the literature bear on the choices made. The intramuscular route used during the first admission should be avoided outright in a patient whose PT is beyond the analytical ceiling of the laboratory, because every injection into muscle is an iatrogenic bleed into a compartment that cannot tamponade itself; the oral and intravenous routes are preferred, and in a patient with this degree of coagulopathy the intramuscular route has no advantage that could offset the risk. No injection-site haematoma was documented in this patient, although the absence of documentation is not the same as the absence of the event. The starting dose deserves the same scrutiny. Two milligrams every eight hours is a replacement dose for dietary vitamin K deficiency, not an antidotal dose for a compound designed to saturate vitamin K epoxide reductase for months, and it lies an order of magnitude below the maintenance requirements reported in the series cited here. The patient's own later requirement for two 50 mg intravenous infusions and for an oral regimen charted at up to 90 mg demonstrates the gap retrospectively. This is not a criticism that could reasonably have been made prospectively, because the size of the toxicant burden was unmeasurable at the time; it is, in our opinion and not on the basis of any comparative study, since none exists for this poisoning, an argument for starting high and titrating downwards rather than the reverse whenever the size of the reservoir cannot be quantified. The oral route is well supported: in a family of three with brodifacoum poisoning, prolonged intravenous therapy was successfully replaced by the injectable formulation of vitamin K_1 given by mouth for approximately five months, an approach adopted because oral tablets were not commercially available and one that is directly applicable to resource-constrained settings such as ours.[29] Reports of massive vitamin K antagonist overdose similarly emphasise sustained high-dose antidotal therapy with prolonged observation.[36] Two further case reports document the same trajectory of protracted, individually titrated therapy over months.[37]

The duration in our patient — approximately eight months of continuous oral vitamin K_1 — sits comfortably within the published range and towards its upper end, consistent with the mean of 2.82 ± 1.85 months and maximum of 10 months in the paediatric series.[8] The decision to stop was made on clinical and biochemical grounds alone. The methodologically superior approach would have been to confirm that the plasma rodenticide concentration had fallen below 10 ng/mL before withdrawal and to re-check it afterwards, as has been explicitly recommended.[31] In the absence of that assay, the two post-withdrawal observations at the fifteenth and sixteenth clinic visits — INRs of 1.31 and 1.16 with no antidote on board — are the only available evidence that the reservoir had genuinely been exhausted, and they are the reason the outcome can be described as a cure rather than as a suppression. A final consideration is that vitamin K_1 replacement in a patient whose vitamin K-dependent anticoagulant proteins C and S are also decarboxylated restores procoagulant and anticoagulant pathways simultaneously; the theoretical concern that overshoot might produce thrombosis has been raised in the context of concurrent anticoagulation during vitamin K_1 therapy for rodenticide poisoning.[38] No thrombotic event occurred in our patient.

Pregnancy: an uncoupled second compartment

The obstetric dimension of this case is where it departs most sharply from the existing literature. The pregnancy was discovered by an ultrasound examination ordered to exclude intraperitoneal haemorrhage, at 11 weeks and 3 days of gestation; the gestational sac and fetus are shown in Figure 4A and the confirmatory colour and pulsed-wave Doppler study of the fetal heart in Figure 4B. It is worth reconstructing the arithmetic explicitly, because the conclusion is not obvious. Counting backwards from the dating scan, which was performed on the third day of the second admission, the interval to the first presentation was 20 days of interval at home plus 27 days of the first admission plus 2 days, that is 49 days; the patient had been bruising for 14 days before that presentation and had ingested the rodenticide 3 days earlier still, so the ingestion preceded the scan by approximately 66 days. A gestational age of 11 weeks and 3 days corresponds to 80 days from the last menstrual period and therefore to conception approximately 66 days before the scan. Both inputs are soft: crown–rump length dating at this gestation carries a confidence interval of roughly five to seven days, and the 17-day interval rests on a distressed adolescent's recollection that her bruising began two weeks before presentation and that she had swallowed the rodenticide three days before that. Within those margins the ingestion and conception coincide. We are deliberate about what this does and does not establish. It does not establish exposure from the precise moment of fertilisation, for which the data are not accurate enough. What it does establish, and what the argument in fact requires, is that the exposure preceded or coincided with conception and therefore spanned the whole of embryogenesis and both maternal coagulopathic episodes.

The single closely comparable report describes a woman who bled at 34 weeks and 3 days of gestation and was found to have brodifacoum poisoning; fetal ultrasonography showed extensive intracranial haemorrhage and intrauterine demise followed, while the mother's coagulation profile was successfully corrected with intravenous and oral vitamin K_1 titrated against serum brodifacoum concentrations.[24] The parallel with our patient is exact in the feature that matters: maternal biochemical rescue and fetal survival were dissociated. The mechanism is not mysterious. Coumarin-type vitamin K antagonists cross the placenta, and the fetal liver is immature in its capacity to carboxylate clotting factors, so that fetal haemostasis is disproportionately vulnerable at any given maternal exposure. The consequences of therapeutic warfarin illustrate the range: fetal intracranial haemorrhage has been documented at 35 weeks in a woman whose own INR was maintained between 1.5 and 2.5 throughout pregnancy — that is, in a mother who was not over-anticoagulated by any conventional standard,[22] and first-trimester exposure is associated with the characteristic facial and skeletal features of coumarin embryopathy.[23] A superwarfarin, being both more potent and vastly more persistent than warfarin, should be expected to reproduce these effects at lower and more sustained exposure.

Two features of this case sharpen the obstetric reading before the implications are drawn. The first is that the notes record a diagnosis of threatened abortion without describing vaginal bleeding, which is the finding that ordinarily defines it. In a patient whose INR was above the analytical ceiling this matters in both directions: had vaginal bleeding been present it might have represented the coagulopathy rather than a threatened pregnancy loss, which would have argued for haemostatic rather than obstetric management; and if it was absent, the label was applied on the basis of the maternal

condition rather than of any specific obstetric finding. We cannot resolve which occurred, and we reproduce the diagnosis as it was made. The second is that we do not know whether a pregnancy test was performed at the first presentation. If none was requested, the miss was procedural and the remedy is a protocol. If one was requested at approximately 4 weeks and 3 days of gestation and returned negative, the miss was biological, and the lesson becomes the different and arguably more useful one that a single early negative test does not exclude pregnancy in a patient who will remain under observation for weeks.

Two clinical implications follow. The first is diagnostic discipline: a pregnancy test should be performed, and repeated if initially negative, in every woman of reproductive age presenting with unexplained coagulopathy, before rather than after the search for a bleeding source. Had that been done at the first presentation, the pregnancy would have been identified at approximately six weeks of gestation, obstetric input would have been available from the outset, and the choice of vitamin K_1 route and dose could have been made with the fetus in view. The relevance of a structured approach to early-pregnancy bleeding is underlined by work comparing predictive models in women presenting to emergency departments with first-trimester bleeding.[39] The second implication is prognostic honesty. Vitamin K_1 itself has not been associated with teratogenicity and its administration to the mother is appropriate; it was given here for a maternal indication, and no fetal benefit should be inferred from it. There is no maternal laboratory value that reports fetal haemostatic competence, and the maternal INR should not be used to reassure about the fetus. In our patient the pregnancy was lost at 18 weeks while maternal coagulation was steadily improving under outpatient vitamin K_1 therapy — that is, at the point in the illness at which maternal haemostasis was closer to normal than it had been at any time since the ingestion. Whether the loss was caused by fetal haemorrhage, by placental insufficiency, by the profound maternal anaemia, or by a combination of these, cannot be determined because neither fetal imaging for intracranial haemorrhage nor histopathological examination of the products of conception was performed. Of these candidate mechanisms, maternal anaemia is the only one for which there is direct documented evidence in this patient: the haemoglobin concentration was 6.0 g/dL before the pregnancy was known and fell to 4.0 g/dL after it had been recognised, and a haemoglobin of 4.0 g/dL in the late first trimester is a profound insult to placental oxygen delivery in its own right, independently of any haemostatic effect on the fetus. That omission is the most important limitation of this report, and it is one that a prospectively aware team would not repeat.

Psychiatric, social and public health dimensions

The exposure in this patient was deliberate self-poisoning in an 18-year-old, and the diagnosis was made only after psychiatric co-management had been established. Deliberate ingestion accounted for 31 of 88 published cases in the pooled analysis,[1] and the risk of re-exposure is a continuing feature of the illness for as long as therapy lasts — which in this condition means many months of outpatient contact. That extended contact is an opportunity as much as a burden. It permits sustained psychiatric follow-up, and it is also the interval during which non-adherence with oral vitamin K_1 becomes the principal preventable cause of relapse.[30] The household and environmental dimension should not be neglected either; poison centre data show that rodenticide enquiries frequently involve children and domestic animals in the same household,[2] and cluster investigations have identified additional exposed household members who were themselves asymptomatic at the time of detection.[21]

Limitations

Several limitations constrain the inferences that can be drawn. No rodenticide assay was performed, so the identity of the compound is unknown and the diagnosis rests on the exposure history and on a compatible, self-consistent clinical and biochemical course; against this, the durability of the coagulopathy over eight months is itself difficult to explain by any agent other than a second-generation anticoagulant rodenticide. Individual vitamin K-dependent factor activities and mixing studies were not measured, so the mechanism was inferred from the global tests rather than demonstrated directly. The PT and INR were censored at the analytical ceiling on eight occasions, so the true peak severity is unknown, and the assay platform and thromboplastin reagent, on which that ceiling depends, are not recorded. The extreme values of day 10 of the second admission — a PT of 191.5 seconds and an INR of 21.60 — were single measurements that were not repeated, and the record does not state whether the sample was drawn from a peripheral vein or from the line through which plasma and vitamin K_1 were being infused; sampling from an infusing line is a recognised cause of spuriously prolonged clotting times and cannot be excluded here, although both the clinical deterioration and the subsequent response to escalation are consistent with the values being real. Absolute leukocyte and platelet counts were not recorded in a form that permits their inclusion here, and fibrinogen and D-dimer were not measured at all, so the exclusion of a consumptive coagulopathy rests on the platelet count and on the time course rather than on those assays. The cumulative volume of plasma transfused cannot be reconstructed, because the record documents "fresh frozen plasma five units per day" without an end date alongside the discrete episodes shown in Table 2. The patient's rhesus D status is not recorded and there is no documentation of whether anti-D immunoglobulin was administered after the second-trimester loss; both are of direct consequence for any subsequent pregnancy and both are also relevant to the alloimmunisation risk created by the transfusion burden. The obstetric record is similarly incomplete: no interval fetal sonography was performed between the dating scan and the loss, no obstetric management of the threatened abortion is documented, and the management of the incomplete abortion itself is not described. The calendar intervals between outpatient visits were not recorded, so the visit axis in Figure 3C is ordinal only. Continued psychiatric follow-up, contraceptive counselling and pre-pregnancy counselling after the loss are likewise undocumented. The recorded outpatient vitamin K_1 doses are reproduced exactly as documented in the clinic record, where the discharge prescription was 30 mg every eight hours; readers should therefore interpret the values in Table 4 and Figure 3C as the doses as charted rather than as unambiguously daily totals. The date of the pregnancy loss was not recorded against a numbered clinic visit; because 18 weeks of gestation lies 6 weeks and 4 days beyond the dating scan, the event must have occurred approximately five weeks after the second discharge, and it has therefore not been anchored to a visit number in Table 4 or Figure 3C, whose horizontal axis is ordinal rather than linear in time. Finally, and most importantly, no fetal imaging for intracranial haemorrhage and no examination of the products of conception were undertaken, so the mechanism of the pregnancy loss is not established. This is a single case, and no causal claim about the fetal outcome can be made from it; the observations are offered as hypothesis-generating.

What this case adds

Three propositions emerge that are not, in this combination, available from the existing literature. First, in superwarfarin poisoning the endpoint of inpatient therapy should be a

normal, and ideally a vitamin K_1-independent, INR rather than the cessation of visible bleeding; this patient was discharged at an INR of 5.32 and relapsed within three weeks. Second, a falling INR during treatment does not establish that the toxicant reservoir is exhausted, as the rise from 2.92 to 21.60 over 48 hours during continuous parenteral antidotal therapy demonstrates; the only unambiguous evidence of cure in this patient came from the two normal values recorded after vitamin K_1 had been stopped. Third, pregnancy constitutes a second, uncoupled compartment whose haemostatic status is not reported by any maternal test, so that a pregnancy test belongs in the first set of investigations for unexplained coagulopathy in any woman of reproductive age, and maternal biochemical recovery must never be presented to a family as fetal reassurance.

4. Conclusion

Superwarfarin intoxication is a serious clinical condition that requires immediate treatment and prolonged monitoring, and its non-specific presentation frequently delays diagnosis. This report describes an 18-year-old woman in whom deliberate rodenticide ingestion produced a vitamin K-dependent coagulopathy severe enough to exceed the analytical limits of the laboratory, which relapsed after an apparently adequate 28-day inpatient course, rebounded to an INR of 21.6 during continuous parenteral antidotal therapy, and required approximately eight months of INR-guided oral vitamin K_1 before it could safely be withdrawn. An unrecognised intrauterine pregnancy of 11 weeks and 3 days was discovered incidentally during the investigation of suspected intraperitoneal bleeding and was lost as an incomplete abortion at 18 weeks despite progressive restoration of maternal haemostasis.

Three lessons are offered. Superwarfarin poisoning should be considered in any patient with an unexplained, severe and isolated prolongation of the PT and APTT in the presence of a normal platelet count and normal hepatic function, and the history should be taken more than once and in a setting that permits disclosure. Treatment should be directed at the hepatic toxicant reservoir rather than at the INR of the day, which means high-dose vitamin K_1 continued for months and discharge criteria anchored to a normalised rather than a merely improved coagulation profile. Where a quantitative plasma rodenticide assay exists, confirmation that concentrations have fallen below the accepted safe threshold is the ideal criterion for stopping therapy. Where it does not — which is the situation of most clinicians who will read this report — the practical alternative used here is to withdraw the antidote and then document a normal coagulation profile on at least two subsequent occasions off treatment, which is the only evidence this patient's course provides that the reservoir was genuinely exhausted. In women of reproductive age a pregnancy test belongs among the first investigations and should be repeated if the first is negative and the patient remains under observation, because the fetus is a compartment that no maternal coagulation test can report on, and maternal biochemical recovery cannot be equated with fetal safety. Aftercare should be planned from the outset and should include continued psychiatric follow-up, explicit contraceptive counselling for as long as a fetotoxic hepatic reservoir persists, determination of rhesus status with anti-D prophylaxis where indicated — noting that the rhesus-negative phenotype is uncommon in this population and that anti-D immunoglobulin is not routinely stocked in many centres, so that both the test and the product need to be planned for rather than assumed — and pre-pregnancy counselling before any subsequent conception. Wider availability of quantitative rodenticide assays and of prothrombin complex concentrate, together with structured psychiatric and adherence support during the many months of outpatient therapy this condition demands, would materially improve the care of patients such as the one described here.

5. Declarations

Ethical approval. This case report was prepared in accordance with the ethical standards of the institutional research committee and with the 1964 Declaration of Helsinki and its later amendments. Institutional approval for the publication of anonymised case material was obtained.

Informed consent. Written informed consent for the publication of this case report and of the accompanying clinical and sonographic images was obtained from the patient. All direct and indirect identifiers, including those embedded in the ultrasound images, have been removed.

Data availability. All data supporting the findings of this report are contained within the article. Further anonymised details are available from the corresponding author on reasonable request.

Conflict of interest. The authors declare that they have no competing interests.

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Author contributions. Both authors participated in the clinical care of the patient, contributed to the acquisition and interpretation of the data, drafted and critically revised the manuscript, and approved the final version.

Use of generative artificial intelligence. All clinical data were extracted from the primary medical record by the authors, every reference was verified against its digital object identifier, and the authors reviewed, corrected and approved the entire manuscript and take full responsibility for its content.

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