delivery. She denied any previous history of deep vein thrombosis. On admission, she had a temperature of 38 °C, and blood tests revealed white cell count 13 300/μl, neutrophilia 79.3 %, haemoglobin 149 g/l, platelets 302 000/μl and D-dimer 3800 ng/ml[1]. Initially, appendicitis or endometritis was suspected, and intravenous cefmetazole was given. On post-admission day 2, contrast-enhanced CT was performed and showed right ovarian vein thrombosis and thrombus extending into the inferior vena cava (Fig. 1). Enhancement of the right uterine wall was weakened compared with the contralateral uterine wall (Fig. 1). Following the diagnosis of OVT, the patient was treated with low-molecular-weight heparin for 2 weeks. The patient defervesced on post-admission day 5, and antibiotic was discontinued after another week.

The aetiology and incidence of OVT remain unknown. Anamnesis showed that this patient had an uncomplicated antepartum course and vaginal delivery. Hypercoagulable state and changes in fibrinolysis and coagulation predispose postpartum women to the development of OVT [1,3], which is in accordance with Virchow's triad. In most cases, OVT arises in the right ovarian vein for the following reasons: dextrotorsion of the uterus during pregnancy which causes compression of the ovarian vein; immediate collapse of the ovarian vein after delivery; incompetent valves in the right ovarian vein that induce blood flow stasis; and the right ovarian vein is longer than the left ovarian vein [1]. Due to the anterograde direction of blood flow in the right ovarian vein, thrombus in the right ovarian vein can extend into the inferior vena cava.

OVT can be managed effectively with anticoagulation and antibiotic therapy [2], but prompt diagnosis is required. Fever and abdominal pain are the most common symptoms. Laboratory tests showing leukocytosis and an elevated D-dimer level can also be used as indicators of OVT. However, due to the non-specific symptoms, OVT is difficult to distinguish from appendicitis and endometritis. Although some authors have concluded that a diagnosis can be made if a patient's fever does not respond to antibiotics after 48 h [3,4], definite diagnosis relies on imaging, particularly contrast-enhanced CT.

In 1980, Zerhouni et al. described three criteria for CT diagnosis of venous thrombosis: enlargement of the thrombosed vein, a low-density lumen and a sharply defined wall. CT was first used to diagnose OVT in 1981 [5], and it remains the gold standard for diagnosis; no additional diagnostic criteria have been reported. However, in this case, the ipsilateral uterine wall was affected by OVT on CT. To the best of our knowledge, this is the first report of an effect of ovarian vein thrombosis on the uterine wall. We also found that the ovarian vein forms an anastomosis with the uterine venous plexus on CT.

[1] ml

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Declaration of Competing Interest

The authors report no declarations of interest.

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Incidental *SOS1* variant identified by non-invasive prenatal screening: Prenatal diagnosis and family clinical reassessment

Dear Editor, *we report on* a 34-year-old woman undergoing non-invasive prenatal screening (NIPS) for aneuploidies and a list of monogenic diseases. NIPS evidenced high risk for a Likely Pathogenic variant (NM_005633.3:c.1433C > G; NP_005624.2 :p.Pro478Arg) in the *SOS1* (SOS RAS/RAC guanine nucleotide exchange factor 1, MIM*182530) gene, associated with Noonan Syndrome 4 (NS 4, MIM#610733).

Noonan Syndrome (NS) is an autosomal dominant genetic disorder, characterized by a wide spectrum of features in variable combination and different severity degrees [1]. This syndrome is classified in the group of RASopathies due to germline mutations in the Ras/mitogen-activated protein kinase (MAPK) pathway [2]. Despite *SOS1* is the second major gene causing NS [3], the first one being the *PTPN11* (Protein-Tyrosine Phosphatase, Nonreceptor-Type 11, MIM*176876) gene [4], in literature cases with incidental prenatal detection of a *SOS1* gene pathogenic variant and its interpretation are not described.

In order to confirm the NIPS findings, an amniocentesis was offered to the woman, but she declined the invasive procedure. Segregation analisis was performed on DNA extracted from both parents periferal blood samples through Sanger sequencing. The variant was detected in the father of the fetus, indirectly confirming the NIPS result. To better characterize the molecular diagnosis, a clinical examination of the father was required. He showed subtle signs compatible with RASopathy. He presented short stature (he was 164 cm tall), hypertelorism, downslanted

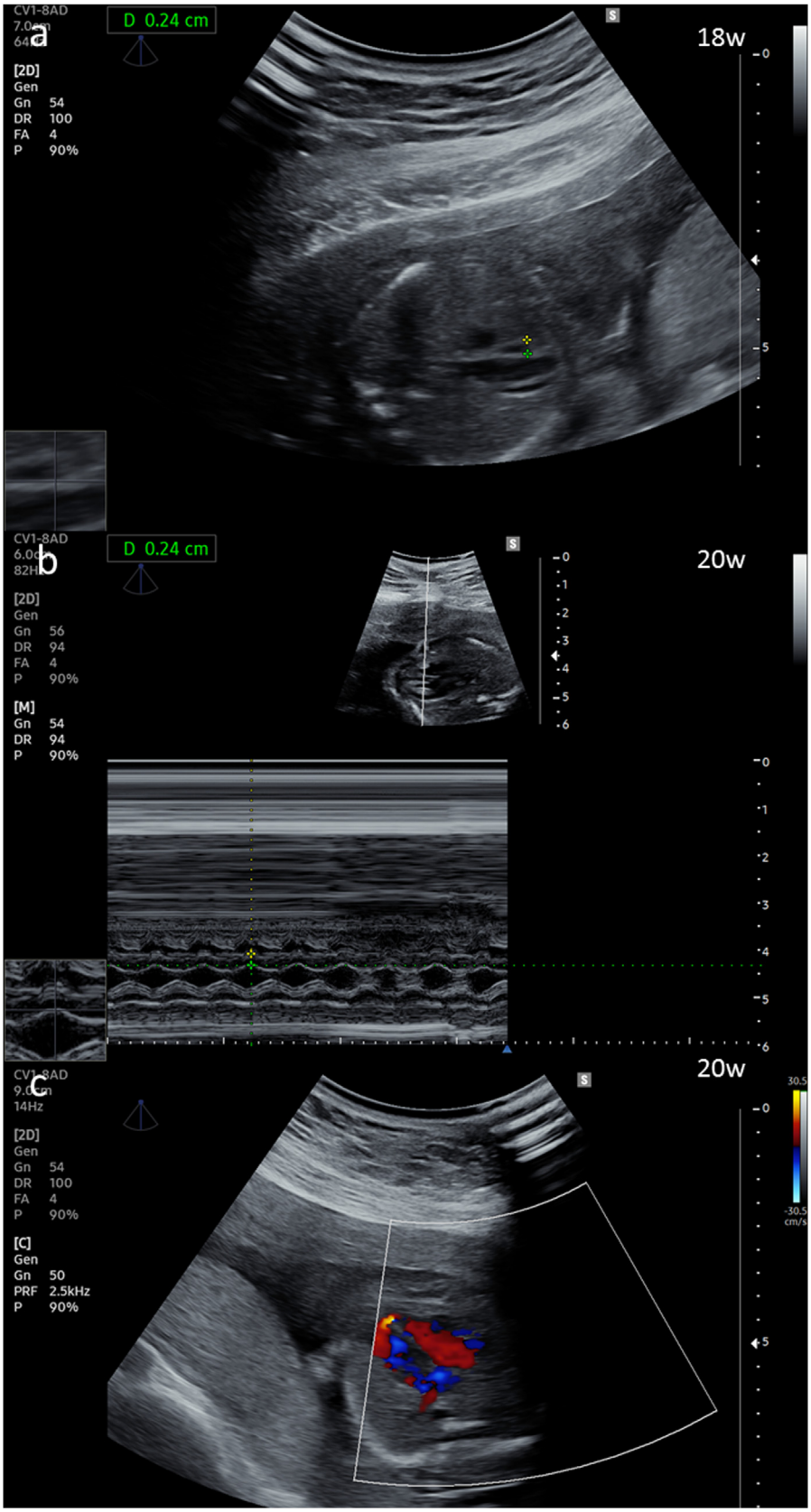

Fig. 1. Prenatal echocardiography. a. Two-dimensional measurement of interventricular septum at 18 w. b. M-mode measurement of interventricular septum at 20 w. c. Small apical ventricular septal defect detected at 20 w.

palpebral fissures and low-set posteriorly rotated ears. He was also diagnosed with attention deficit hyperactivity disorder.

The woman underwent fetal echocardiography at 18 ad 20 weeks of gestational age. Interventricular septal dimensions were at the upper limit of normal range (Fig. 1a, b) and a small colored spot was noted in its apical part, suggesting a muscular ventricular septal defect (Fig. 1c). During the 3rd trimester polyhydramnios was noted. These ultrasound findings supported the molecular diagnosis of NS, which can prenatally presents with thickness of interventricular septum, valvular dysplasias and polyhydramnios [5].

The baby was born at 40w3d by Cesarean delivery. Neonatal weight was 4.310 g (92th centile), lenght was 50 cm (12th centile), and occipitofrontal circumference measured 35.5 cm (43th centile). He presented dysmorphic features comprising high forehead, hypertelorism, ptosis, epicantal fold, low-set and posteriorly rotated ears and short neck, reminiscent of RASopathies, which became more evident during the first weeks of life.

Transthoracic echocardiography, performed two days after birth in order to better characterize the prenatal findings, showed dysplastic mitral valve, with redundant leaflets and elongated chordae. Also the aortic valve was dysplastic, with thickening of the leaflets without stenosis or regurgitation. The previously detected muscular ventricular septal defect measured 2.5−3 mm and showed a moderate left-to-right shunt.

Both molecular and ultrasound prenatal findings were clinically confirmed after birth through genetic assessment, also allowing to predict the recurrence risk of 50 % for each following pregnancy of the couple and to plan an appropriate follow-up for the child.

This report highlights the difficulties raised by NIPS results for monogenic conditions and the implications of variant interpretation in prenatal setting. In this context, RASopathies represent a specific challenge due to the broad phenotypic spectrum and inter- or intra-familiar variability, which needs a multidisciplinary approach and expertise. A deep knowledge of the clinical, prenatal and dysmorphic features of this spectrum is required to properly rise clinical suspicion and guide prenatal management and genetic counseling.

Statement of ethics

The parents gave written informed consent, approved by Ethical Committee of Sapienza University of Rome.

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Declaration of Competing Interest

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Abdominal wall necrosis from surgical incisions letter to editor – Brief communication

Dear Editor,

We found that understanding of the blood supply to the abdominal wall is essential when operating on a previously incised abdomen.

We report a case of a 32-year old female with lower abdominal wall necrosis secondary to parallel transverse surgical incisions.

The patient had a past medical history of two lower segment Caesarean sections (LSCS) and an abdominoplasty over a five-year period through the same lower abdominal incision. Five years after the last open abdominal procedure, she was admitted for her third elective LSCS. The Pfannenstiel incision was performed inferior and parallel to the old abdominal scar.

Ecchymosis to the soft tissue in between the two scars was noticed day one postoperative. The patient was readmitted to the postnatal unit six days later via general practitioner referral for “black and blistering” soft tissue between the scars, measuring 3 × 12 cm (Fig. 1).

Plastic surgery opinion was sought, which determined abdominal wall necrosis from interruption of the blood supply. The necrotic abdominal wall tissue was debrided and directly closed to form a single scar by the obstetric surgeons, with good wound healing observed on follow up.