A 58-year-old realtor presented to her community hospital with an acute left-sided facial droop in December, 2015. She reported that in the past 2 months her children had noticed (and teased her for) malapropisms (video 1), but she had no other symptoms and had continued to manage complicated sales. She did not smoke and consumed alcohol occasionally. Her body-mass index was 23 kg/m². On examination she had a left lower facial weakness and made occasional paraphasic errors during speech. Her platelet count was 58 × 10⁹ per L, white cell count 15·2 × 10⁹ per L (normal 4·50–11·0 × 10⁹ per L), lactate dehydrogenase was 507 IU/L (normal 110–210 IU/L), and C-reactive protein was 30·50 mg/L (normal <5·0 mg/L). MRI of the brain showed new acute bihemispheric infarcts (figure) and a transoesophageal echocardiogram identified two mobile echo-densities on the atrial surface of the mitral valve leaflets (video 2).

On admission she was alert but had difficulty following instructions, expressive speech difficulties, and sensory neglect on the left side. Her platelets were 58 × 10⁹ per L, white cell count 15·2 × 10⁹ per L (normal 4·50–11·0 × 10⁹ per L), lactate dehydrogenase was 507 IU/L (normal 110–210 IU/L), and C-reactive protein was 30·50 mg/L (normal <5·0 mg/L). MRI of the brain showed new acute bihemispheric infarcts (figure) and a transoesophageal echocardiogram identified two mobile echo-densities on the atrial surface of the mitral valve leaflets (video 2).

CT scan showed a slightly enlarged right ovary (6 cm × 3·3 cm × 3·6 cm) and mild fat-stranding of the omentum. On PET/CT there was FDG uptake (standard uptake value [SUV] 7·3) in the right ovary and a small focal FDG uptake (SUV 4·88) in the left ovary; there were no other abnormalities. Her CA125 level was 1667 IU/mL (<4·9 µg/L). A CT-directed biopsy sample showed high-grade serous carcinoma, probably of ovarian or tubal origin. Despite anticoagulation treatment she continued to have cerebral and systemic embolic events, with additional strokes and infarctions affecting both kidneys and the spleen (figure) so despite her low platelet count (28–36 × 10⁹ per L) we started dose-dense chemotherapy (carboplatin area under the curve [AUC] 2 and paclitaxel 80 mg/m² once a week) because the patient was too unwell to receive a full dose every 3 weeks, which is the standard of care. After one cycle platelets had risen to 218 × 10⁹ per L, the CA125 had reduced to 555 IU/mL; she had no further embolic events and we were able to discontinue treatment.

2 weeks later she returned to the same hospital with acute onset confusion and disorientation. Her platelets had dropped to 66 × 10⁹ per L. Repeat CT showed no further changes but her symptoms continued to worsen, and 48 h later she was transferred to our institution for further assessment.

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operate successfully, leaving no residual disease (appendix). Final histopathology revealed high-grade serous carcinoma originating in the fimbria of the right fallopian tube (figure), with metastasis to the right ovary, and focal migration to the left ovary and supracolic and infracolic omentum. Lymph nodes were negative. She was treated with intraperitoneal and intravenous chemotherapy postoperatively.1 At follow-up in April, 2017, her CA125 was 6 IU/mL, cardiac and haematological function were normal, and she had regained her driver’s licence although she still had expressive dysphasia (video 2).

Our patient first presented with malapropism, which is word substitution often with unintended amusing effect (named after Mrs Malaprop, a character in Sheridan’s 1775 play The Rivals), caused by infarcts in the right insular cortex, with limited damage to Wernicke’s area because comprehension and writing were initially spared (video). Procoagulant cytokines released by the high-grade serous carcinoma activated platelets and the clotting cascade and caused damage to her mitral valve endothelium, resulting in non-bacterial thrombotic endocarditis with emboli from friable vegetations of the mitral valves. She had no abdominal or gynaecological symptoms. Although this presentation is rare, cancer of the fallopian tube often presents in unusual ways. Indeed it could be said that if cancer is the emperor of all maladies, the fallopian tube is the empress of subterfuge. High-grade serous carcinoma is the emperor of all maladies, the fallopian tube is presents in unusual ways. Indeed it could be said that if cancer is the emperor of all maladies, the fallopian tube is the empress of subterfuge. High-grade serous carcinoma originates in the sinuous tubal fimbriae can capitalise on its access to the peritoneal cavity, metastasising to the ovaries, abdominal organs, and diaphragm, while the primary tumour lesion remains inconspicuous. In retrospective studies2 of cancers originally thought to be of ovarian or peritoneal origin, meticulous re-examination of the fallopian tubes identified them as the primary site in 40–70% of cases. In a prospective study3 of women with non-specific symptoms, when high-grade serous carcinoma was identified early, 75% were found to have cancers that originated in the fallopian tube and spread to the abdomen with normal ovaries, which underscores the futility of focusing on the ovaries for early detection of high-grade serous carcinoma. Many groups4 are investing in DNA-tagging technology to aid early diagnosis by identifying mutations associated with high-grade serous carcinoma in pap smears from the genital tract (eg, NCT02288676). However, this approach will only work if mutations used in the diagnostic panel, identified by The Cancer Genome Atlas (TCGA), are appropriate for early diagnosis; 95% of high-grade serous carcinomas sequenced in TCGA were found to be advanced stage III/IV.5 In our patient, who was diagnosed in very early stage III disease, the mutations found were not those identified in TCGA (appendix). The question remains whether this case is unusual, or whether the mutations associated with advanced high-grade serous carcinoma are not associated with early disease. Identifying driver mutations of early high-grade serous carcinoma might take time, given that most high-grade serous carcinomas are diagnosed at an advanced stage. Adding serial CA125 to the battery of tests usually ordered for persistent or unexplained symptoms in postmenopausal women over the age of 50 years might increase the proportion of high-grade serous carcinoma diagnosed early enough for complete resection and potential cure.

Contributors
LG, KJ, XZ, TL, EE, GA, JA, LF, DM, CM, AS, and KN cared for the patient. JR, TR, and CM did the gene sequencing. All authors contributed to writing of the report. Written consent to publication was obtained.

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