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## Case Report

# Dilemma in management of menorrhagia with cerebral stroke

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### Introduction

Most cerebral infarctions are caused by either thrombosis of the cerebral vessels or emboli from a proximal source like heart or pelvic vessels or due to cerebral hemorrhage. Severe anemia can lower oxygen carrying capacity and lead to hypoxia, which in turn leads to global brain hypoperfusion.

We report a case of 48 year old woman with previously undiagnosed hypertension and diabetes who had an episode of severe menorrhagia followed by development of hemiparesis. We managed the patient by doing a total abdominal hysterectomy followed by anticoagulant therapy. The patient has recovered completely and is symptom free presently.

### **Case History**

A 48 year old, multiparous woman presented with right sided hemiparesis and menorrhagia of three days duration on 9th May 2006. The patient had been diagnosed an year earlier to have had a fibroid. She was

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days she had developed excessive bleeding per vaginum along with weakness of the right upper and lower limbs. Neurological examination revealed right sided

not on regular treatment for menorrhagia. In the prior 3

hemiparesis. On pelvic examination the uterus was firm, irregularly enlarged and corresponding to 14 weeks size.

Coagulation profile, kidney and liver function tests were within normal limits. Contrast enhanced computer tomography of the head showed a subacute ischemic infarct in the left parietal area. A diagnosis of the right sided hemiparesis due to ischemia was made.

After consultation with the neurologist, she was managed with antihypertensives and cerebral perfusion agents - citicholine and piracetam. There was a need for starting thrombolytic therapy for improving the cerebral perfusion, this was not started due to menorahagia. Hormonal treatment could not be given as it could aggravate thrombosis. Hence only hemostatic agents could be given for the management of menorrhagia. As vaginal bleeding persisted and the patient's general condition especially did not improve, she was taken up for an emergency hysterectomy. Routine elective surgery is delayed, until the brain has had time to recover from infarction. However in view of continuing vaginal bleeding and no improvement in the neurological status, an emergency hysterectomy was decided upon. Surgery was performed under spinal anesthesia after

informed consent on 10<sup>th</sup> May 2006. Total abdominal hysterectomy with bilateral salpingo-oophorectomy was done. Histopathological examination revealed adenomyosis with chronic cervicitis.

Postoperatively aspirin was started on the third day. The patient steadily improved and was discharged on the eighth postoperative day. Follow up after 8 months showed partial recovery of hemiparesis.

### **Discussion**

Hypertension and diabetes are two conditions, which increase the risk of cerebrovascular accidents due to increased tendency for atherogenesis and hemorrhage<sup>1</sup>. Aggressive management of cerebrovascular diseases necessitates thrombolytic and anticoagulant therapy.

Severe menorrhagia can lead to a state of hypovolemia leading to cerebral hypoperfusion and aggravation of the cerebral ischemia. Ueda et al<sup>2</sup> have described a case of multiple bilateral border zone infarcts in cerebral and cerebellar hemispheres in a woman with anemia due to excessive uterine bleeding. Schapiro and Thorp<sup>3</sup> reported a case of leiomyoma induced menorrhagia leading to myocardial infarction where hysterectomy was done to stop further hemorrhage 3. Thrombolytic and anticoagulant therapy can aggravate the uterine bleeding<sup>4</sup>. Hormonal treatment in the form of high doses of progesterone can control the menorrhagia but the thrombogenic ill effects of the above prohibit their use in a case of stroke 5. A levonorgestrel intrauterine device can be used in a patient with oral contraceptive pill related cerebral venous thrombosis and simultaneous vaginal bleeding 6. GnRH analogues can also be used in

dysfunctional uterine bleeding. But they cause an initial flare effect and beneficial effects are seen after 3 weeks<sup>7</sup>.

Ideally the surgery should have been delayed till the brain recovered from the effects of infarction but continuing uterine bleeding worsened the problem of cerebral hypoperfusion. This was coupled with the inability to start thrombolytic therapy due to the risk of aggravating menorrhagia. The hemodynamic stability attained after the hysterectomy probably hastened the patient's neurological recovery.

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