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unknown pregnancy at time of insertion as potential causes of contraceptive failure. The only explanation for contraceptive failure in the present case would appear to be the hepatic enzyme-inducing effect of the antiretroviral therapy, since the patient was not on any other medication apart from a Becotide inhaler for her asthma.

Efavirenz is the only component of the patient's antiretroviral regimen known to have a liver enzymeinducing effect. The nucleoside reverse transcriptase inhibitors are metabolised via a different route and thus would be unlikely to compete for the same metabolic enzymes and elimination pathways.

Efavirenz has a high affinity for binding to plasma proteins, displays a prolonged plasma half-life, is metabolised via cytochrome P450 2B6 and 3A4, and induces CYP450 activity during chronic administration.⁶ Other compounds that are substrates of CYP3A4 such as progesterone, anticonvulsants and anti-tuberculosis agents may have decreased plasma concentrations when coadministered with efavirenz. Dosage adjustment is therefore necessary with the co-administered drug. Efavirenz is also known to increase the plasma concentration of ethinylestradiol, the clinical significance of which is not known.⁷

Conclusions

HIV-seropositive women continue to be sexually active after diagnosis. All such women should be counselled regarding proper use and possible side effects of their chosen method(s) of contraception.

The importance of using barrier methods in addition to their primary choice of contraception cannot be overemphasised - even in those women with HIV-positive partners – in order to reduce the potential for transmission of drug-resistant virus. Condoms should be promoted and provided free of charge, since their correct and consistent use during sexual intercourse decreases the risk of transmitting HIV to the uninfected partner by up to 96% in addition to providing protection against other sexually transmitted infections and unplanned pregnancies.8

All such women wishing to use hormonal contraceptive methods should also be given condoms and counselled as to their use, especially since protease inhibitors, rifamycins and non-nucleoside reverse transcriptase inhibitors may decrease the effectiveness of hormonal contraceptives.^{7,9,10}

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CLINICAL CONUNDRUM CORRESPONDENTS

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ERRATUM

'Migraine and use of combined hormonal contraceptives: a clinical review'.

E Anne MacGregor, J Fam Plann Reprod Health Care 2007: 32(3): 159-169

Readers should be aware that the text heading in Box 1 on page 166 was incorrectly printed as 1.1 Migraine without aura, when it fact the heading should have been 1.2 Migraine with aura. The correct version of Box 1 is reproduced below.

Box 1: International Headache Society (IHS) diagnostic criteria for typical aura with migraine headache

Typical aura consisting of visual and/or sensory and/or speech symptoms. Gradual development, duration no longer than 1 hour, a mix of positive and negative features and complete reversibility characterise the aura, which is associated with a headache fulfilling the criteria for '1.1 Migraine without aura'.

1.2 Migraine with aura

Diagnostic criteria:

- At least two attacks fulfilling criteria B-D
- Aura consisting of at least one of the following, but no motor weakness
 - Fully reversible visual symptoms including positive features (e.g. flickering lights, spots or lines) and/or negative features (i.e. loss of vision)
 - Fully reversible sensory symptoms including positive features (i.e. pins and needles) and/or negative features (i.e. numbness)
 - 3. Fully reversible dysphasic speech disturbance
- At least two of the following:
 - 1. Homonymous visual symptoms1 and/or unilateral sensory symptoms
 - 2. At least one aura symptom develops gradually over ≥5 minutes and/or different aura symptoms occur in succession over ≥5 minutes
 - 3. Each symptom lasts ≥5 and <60 minutes
- Headache fulfilling criteria B–D for '1.1 Migraine without aura' begins during the aura or follows aura within 60
- Not attributed to another disorder