patients who had SCLC-LEMS with a matched group of patients who had SCLC only.

Survival data were available for 15 SCLC-LEMS patients seen in a neuromuscular clinic since 1987. All had biopsy-proven SCLC, typical clinical and electromyographic features of LEMS, and raised titres of anti-P/Q-type voltage-gated calcium channel antibodies detectable by RIA.1 Hospital records were available for SCLC patients who had no neurological illness and who had participated in randomised SCLC treatment trials between 1988 and 1997 at the Middlesex and University College Hospitals, London, UK. We matched each of the 15 SCLC-LEMS patients with five or six SCLC-only patients (n=81) for sex, age at SCLC diagnosis, tumour extent (limited or extensive), and treatment (chemotherapy or radiotherapy). Computer matching was done by medical staff masked to the SCLC-LEMS patients’ survival data.

Three SCLC-LEMS patients had extensive disease at tumour diagnosis; four did not receive specific tumour treatment because they died soon after diagnosis. With one exception, symptoms of LEMS predated the diagnosis of SCLC (range 0·3–4·7 years). At the time of the study, four SCLC-LEMS patients were alive with no evidence of tumour recurrence after a median of 6 years (range 1·5–8·5) since tumour detection and treatment.

Kaplan-Meier survival estimates (figure) show a significantly shorter median survival time from the diagnosis of SCLC in SCLC-only patients (17·3 months, p=0·048, Log-rank test). Factors contributing to this better survival rate in SCLC-LEMS might be a slower rate of growth in tumours since tumour detection and treatment.

Proportions of CRP changes are shown in the figure. CRP concentrations were 1·55 (range 0·25–8·85) mg/L and 1·10 (range 0·20–3·8) mg/L. Because CRP more frequently decreased than increased during statin treatment and 1·41 mmol/L (0·69), LDL cholesterol 4·96 mmol/L (0·75), serum cholesterol 7·08 mmol/L (0·79) HDL cholesterol 4·0 mmol/L were randomised to receive either 20 mg atorvastatin or 20 simvastatin once daily in a double blind fashion. CRP was measured with an enzyme-immunoassay (Medix Biochemica, Espoo, Finland) from two baseline samples and after 4 months of treatment. The advantage of this CRP assay is that its sensitivity is 0·3 mg/L as compared with the sensitivity of 10 mg/L of older immunoturbidimetric methods.

Baseline lipids were (means of two determinations [SD]): serum cholesterol 7·08 mmol/L (0·79) HDL cholesterol 1·41 mmol/L (0·69), LDL cholesterol 4·96 mmol/L (0·75), triglycerides 1·66 mmol/L (0·60). Medians of baseline CRP concentrations (mean of two determinations) and CRP during statin treatment were 1·58 (range 0·20–18·5) mg/L, and 1·10 (range 0·20–38·0) mg/L. Because CRP more frequently decreased than increased during statin treatment the sign test for paired comparisons was statistically significant (p=0·012; Wilcoxon signed rank test p=0·090). If CRP values of 10 mg/L or more (reflecting possible interventional infection [n=12]) were excluded, the medians of CRP concentrations were 1·55 (range 0·25–8·85) mg/L and 1·00 (range 0·20–6·80) mg/L during baseline and statin treatment, (sign test p=0·005, Wilcoxon signed rank test p=0·012). Proportions of CRP changes are shown in the figure.

To our knowledge this is the first study to show an effect of statins on CRP, probably because a sensitive measurement was used. These results are preliminary since the double blind study continues and type of statin as well as in-trial lipid values in individual patients are currently unknown. Thus, we do not know long-term effects, the possible relation between lipid lowering and CRP change,
increases of CRP have predicted the development of myocardial infarction. Furthermore, pravastatin has been reported to be especially effective in reducing risk of recurrent myocardial infarction in patients with signs of inflammation at baseline.1

and whether the two statins have different effects. However, the results are of clinical interest because even small increases of CRP have predicted the development of myocardial infarction.2 Furthermore, pravastatin has been reported to be especially effective in reducing risk of recurrent myocardial infarction in patients with signs of inflammation at baseline.1


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Two de-novo balanced autosomal translocations after intracytoplasmic sperm injection

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Intracytoplasmic sperm injection (ICSI) pregnancies have an increased risk of sex chromosome abnormalities, but not of de-novo autosomal rearrangements.3,4 Only one case of trisomy 21 has been reported in a woman aged 41 years.1

We report the prenatal diagnosis of a fetus with two de-novo balanced chromosomal translocations, 45, XX, t (2;6) (p;q), –14; –21; t (14q;21), in an ICSI pregnancy.

The patient, a woman aged 32 years, underwent amniocentesis at 17 weeks’ gestation after genetic counselling. Parental karyotypes were normal, which shows de-novo origin of the two chromosomal rearrangements in the fetus. Ultrasonography at 24 weeks showed no malformations, but fetal measurements were 2 weeks smaller than expected.

Complex chromosome rearrangements (CCR) are rare—only ten cases have been diagnosed prenatally. Congenital anomalies were present in three cases, and mental retardation of delayed development in two cases. Only three fetuses developed normally. Four pregnancies were terminated.3,4 On the basis of these data and suspected

HIV-1 replication in patients with undetectable plasma virus receiving HAART

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With highly active antiretroviral therapy (HAART) it is expected that, for most previously treated patients, plasma HIV-1 will decline to levels below the detection limit of currently available assays.1 It has been claimed that HIV-1 replication is completely inhibited, although replication-competent HIV-1 can be isolated in most patients by activating resting, presumably latently infected, CD4 cells.2,3 Undetectable viraemia may not reflect absence of HIV-1 replication, but rather the limitations of the assays used. We examined whether ongoing viral replication could be shown in patients with sustained plasma HIV-1 levels of less than 50 copies/mL. Of 138 patients receiving HAART in whom plasma levels of HIV-1 were below the detection limit of commercially available assays (<400–<500 HIV copies/mL) for at least 6 months, only 26 had <50 HIV copies/mL with sensitive RT-PCR assays. These 26 patients (all male; mean duration of HAART, 19 months; mean duration of <400–<500 HIV copies/mL, 17 months; table) were studied to evaluate viral replication in cellular compartments. All the patients were receiving lamivudine in combination with either zidovudine or stavudine, and all but one were receiving protease inhibitors. Three were receiving nevirapine. Half the patients were receiving intermittent interleukin-2, in addition to their antiretroviral therapy, as part of other protocols.1

Peripheral blood mononuclear cells (PBMCs) from these patients were examined for evidence of ongoing viral replication with highly sensitive nested RT-PCR techniques. In all but two patients, cell-associated HIV-1 RNA could be detected with gag-specific primers (sensitivity, 10 copies/million cells). In all cases, a control with no reverse transcriptase control was negative. In the remaining two