Results The majority of the 11876 participants included in the analysis were White, employed, and median age was 33 years. In 2013, overall and undiagnosed HIV prevalence was 13.6% (106/782) and 3.2% (25/782) respectively. Overall undiagnosed fraction remained unchanged: 34% (45/131) in 2000 and 24% (25/106) in 2013. Undiagnosed fraction among sexual health clinic non-attenders in last year remained unchanged: 62% (23/37) in 2000; 59% (10/17) in 2013. HIV testing in the last year increased: 26% (263997) to 60% (467777); among undiagnosed HIV+ men, it increased from 28.6% (10/35) to 66.7% (16/24). Compared to men aged >45, men aged 15–25 (AOR: 7:47, 95% CI: 1:56–35:74); compared to sexual health clinic attenders in the last year, non-sexual health clinic attenders (AOR: 4:39, 95% CI: 1:90–10:16) were more likely to have undiagnosed HIV.

Conclusions HIV testing has increased yet undiagnosed HIV remains unchanged. Strategies to increase HIV testing among young MSM and in non-sexual health clinics should be developed and evaluated.

Declaration of interest statement AMJ has been a Governor of the Wellcome Trust since 2011. The other authors declare that they have no conflicts of interest.

O21 - HIV and co-morbidity

O21.1 PLATELET DERIVED SOLUBLE GLYCOPROTEIN VI DECREASES PRIOR TO CORONARY EVENT IN HIV POSITIVE PATIENTS

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Introduction Platelets play a key role in coronary artery disease (CAD). Glycoprotein VI (GPVI) is a platelet specific collagen receptor which is shed when activated. Soluble (s) GPVI is associated with CAD in the general population and lower levels have been found in patients taking abacavir. This trial was performed to determine if sGPVI was predictive of CAD in HIV.

Methods 24 HIV+ subjects with CAD (HIV+ cases) with stored plasma available in the 12 months before CAD diagnosis were age and sex matched 1:2 with 46 HIV+ subjects without CAD (HIV+ controls). 41 HIV negative controls (healthy controls) were used as comparators. HIV+ patients had two samples analysed; 12 and 1 month before CAD diagnosis; healthy controls had a single sample analysed (202 samples in total). sGPVI was determined by ELISA.

Results Of the combined HIV+ subjects 63% (90%) were male; mean 51 years; 92.8% taking antiretrovirals. HIV+ subjects (combined HIV+ cases and HIV+ controls) were more likely to smoke (34% [30.6%] vs 3 [7.3%], p < 0.001) than healthy controls. HIV+ cases were hypertensive (13 [54.1%] vs 5 [10.8%], p < 0.001) and had a family history of CAD (12 [52.1%] vs 9 [25.0%], p = 0.033) at higher rates than HIV+ controls. sGPVI was higher in HIV+ subjects (combined) than healthy controls (123.2 ng/ml [SD 59.5] vs 9 [25.0%], p = 0.033) at higher rates than HIV+ controls. sGPVI was higher in HIV+ subjects (combined) then healthy controls (129.9 ng/ml [SD 59.5] vs 84.4 ng/ml [SD 46.1], p < 0.001). 12 months before event there was no difference in sGPVI between HIV+ cases and HIV+ controls (129.9 ng/ml [SD 59.5] vs 84.4 ng/ml [SD 46.1], p < 0.001). 1 month before event sGPVI was significantly lower in HIV+ cases (111.1 ng/ml [SD 45.0] vs 143.9 ng/ml [SD 56.1], p = 0.016).

Conclusion HIV+ subjects have higher sGPVI than healthy controls; sGPVI is lower prior to CAD event in HIV+. sGPVI may play an important role in promoting CAD in HIV.

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