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A case of 'crouching' triglyceride and 'hidden' cholesterol

C. M. MAK, R. W. C. PANG, and S. TAM
Division of Clinical Biochemistry, Queen Mary Hospital, Hong Kong SAR, P. R. China

Fasting specimens are recommended for full lipid profile testing by the third report of the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP III) because of the well-known errors in the Friedewald estimation of low-density lipoprotein cholesterol (calculated LDL-C) for non-fasting samples. The report remains highly focused on LDL-C management and includes the definition of target values; however, non-high-density lipoprotein cholesterol (non-HDL-C) may play an important role as a secondary therapeutic goal for initiation of LDL-lowering.[1]

Recently, a case of ischaemic heart disease (IHD) complicated with acute pulmonary oedema (APO) was encountered. The fasting plasma lipid profile results for the 70-year-old female patient showed total cholesterol 5.2 mmol/L (reference value <5.2), triglyceride 5.8 mmol/L (<1.7) and HDL-C 0.19 mmol/L (>1.0). Intriguingly, because LDL-C calculation is invalid when TG level is >4.5 mmol/L, LDL-C was measured by a direct surfactant method (homogeneous enzymatic, Hitachi 912, Roche Diagnostics) and was found to be very low (0.2 mmol/L). Apolipoprotein B100 (apo B) was, however, 1.20 g/L (0.41–1.07) and apo A1 was 0.73 g/L (1.20–2.00).

Correspondence to: Dr Chloe M. Mak
Division of Clinical Biochemistry, Queen Mary Hospital,
102 Pokfulam Road, Hong Kong
Email: makm@ha.org.hk

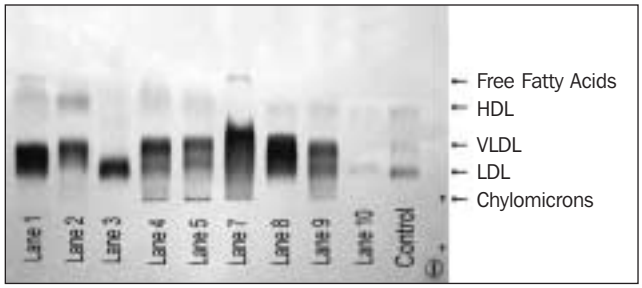

Fig. 1. Lipoprotein electrophoresis of the patient's plasma sample. Lane 3 shows a dense and distinct β-lipoprotein band, which is compatible with lipoprotein(a).

The sample appeared clear. Lipoprotein electrophoresis of the plasma sample revealed a dense and distinct β-lipoprotein band; however, the pre-β fraction was absent and there was no chylomicron at the origin. The faint α band was consistent with the low plasma HDL-C level (Fig. 1, Lane 3). Lipoprotein(a) (Lp[a]) was measured and found to be 505 mg/L (Immage immunochemistry system, Beckman Coulter).

The patient was on warfarin but not on any lipid-lowering drugs. Although the presence of other classes of triglyceride-rich lipoproteins could not be excluded, the lipoprotein pattern appeared unremarkable. Presumably, the presence of a high level of endogenous free glycerol (measured as triglycerides by a non-blank method) in patients under stress (IHD and APO) may account for the high plasma triglyceride result in this patient. Of concern, however, was the extremely low LDL-C concentration by a direct surfactant method.

The patient showed no evidence of obstructive liver disease or cholestasis, so the presence of LPX (which lacks apo B) or LPY (a triglyceride-rich lipoprotein) becomes unlikely. So, where had all the cholesterol gone?

Lipoprotein(a) is a cholesterol-rich lipoprotein that resembles LDL in size and lipid composition, yet contains apo(a), a structural glycoprotein that is covalently attached, via a disulphide linkage, to the apo B moiety of the LDL-like particle. The concentration of Lp(a), measured as its protein moiety of apo(a), by an immunonephelometric assay (Beckman Coulter Array system) was found to be very high in this sample. Although a method for directly determining Lp(a) cholesterol using lectin-bound plasma technology[2] was not available at the time of testing, following the approach proposed by Scanu,[3] the estimated cholesterol content of the Lp(a) protein based on the known chemical composition of Lp(a) was 3.2 mmol/L.

Unfortunately, Lp(a) is not included as part of most routine lipid profiles. Although its omission is inconsequential in individuals with low plasma Lp(a) levels,

it becomes increasingly significant when the levels are elevated. It is important to determine the proportion of cholesterol derived from LDL and Lp(a) independently because it is well established that many cholesterol-lowering agents (e.g., statins) are effective in lowering plasma LDL-C but have little or no effect on Lp(a). □

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Molecular typing of *Dientamoeba fragilis*

J. J. WINDSOR*, C. G. CLARK† and L. MACFARLANE*.

**National Public Health Service for Wales Aberystwyth, Bronglais Hospital, Aberystwyth, Ceredigion; and †Department of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine, London, UK*

Dientamoeba fragilis is a human intestinal protozoan parasite that was described originally by Jepps and Dobell in 1918.[1] Initially it was thought to be an amoeba but subsequent studies showed that it shared antigens with the flagellates Histomonas and Trichomonas.[2,3] Transmission electron microscopy (TEM) studies confirmed many trichomonad-like aspects of *D. fragilis* ultrastructure and accordingly it was reclassified to reflect this.[4] However, it was not until Silberman *et al*.[5] sequenced the small subunit ribosomal RNA (rRNA) gene of *D. fragilis* that it was shown conclusively to be a trichomonad, albeit one without a flagellum.

D. fragilis remains a little-studied parasite despite numerous reports linking it with gastrointestinal symptoms.[6–9] This may be explained in part by the difficulties associated with the diagnosis of *D. fragilis* infection, and certainly many laboratories in the UK do not use suitable methodologies for its detection.[10] *D. fragilis* does not have a known cyst stage, and, as the nuclei are not visible in saline or iodine preparations, the trophozoite is difficult to detect in direct microscopy.[11] Permanently-stained smears or faecal culture techniques are the recommended detection methods for *D. fragilis*.[12]

D. fragilis has a worldwide distribution and is one of the most common intestinal protozoa when suitable methods are employed.[12] The numbers of *D. fragilis* detected and reported to the Communicable Disease Surveillance Centre (CDSC; www.hpa.org.uk/cdr/) in England and Wales remain relatively low, although this is likely to be a gross underestimation of the true prevalence. In a previous report, using a sensitive culture method, *D. fragilis* was detected in 2.6% (25/976) of unselected specimens.[13] One of the advantages of using the culture method is that isolates can be typed subsequently using molecular methods.

Johnson and Clark[14] analysed *D. fragilis* ribosomal genes using a polymerase chain reaction (PCR)–restriction fragment length polymorphism (RFLP) technique (riboprinting). They found evidence for two genetically distinct variants; however, the number of isolates investigated was small (n=12). The degree of divergence (about 2%) between these variants is comparable to that between the pathogenic *Entamoeba histolytica* and the non-pathogenic *E. dispar* (which are morphologically indistinguishable). In view of this it seems plausible that the existence of genetic variants might be associated with the variable symptomatology of infection.

In the present study, *D. fragilis* isolates are typed using riboprinting to investigate further the degree of genetic diversity in a larger sample size, in order to add to the limited data available.

All faecal specimens sent to NPHS Aberystwyth were cultured routinely for intestinal protozoa using Robinson's culture method.[15] Over an 18-month period, 43 positive *D. fragilis* cultures were obtained. All were confirmed as *D. fragilis* using a trichrome stain after fixation with Schaudinn's fixative. Cells were pelleted and lysed in

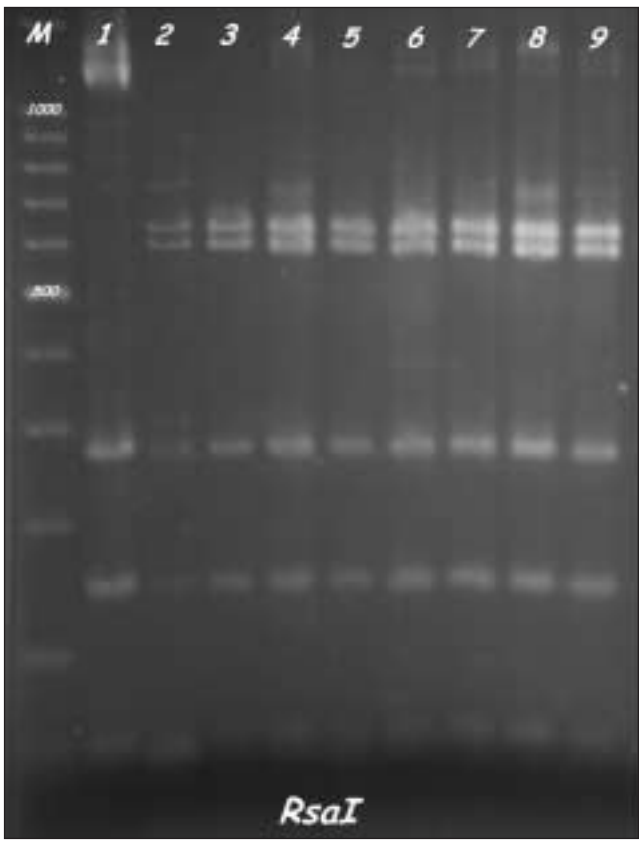

Fig. 1. *Rsa*I digest patterns of *D. fragilis* PCR-amplified SSU-rDNA. M: 100-bp molecular weight marker. Lane 1: genotype 2 (Bi/pa). Lanes 2–9: all genotype 1 (NPHS Aberystwyth isolates).

Correspondence to: Jeff Windsor
NPHS Aberystwyth, Bronglais Hospital, Caradoc Road, Aberystwyth, Ceredigion SY23 3HF
Email: jeff.windsor@nphs.wales.nhs.uk