Further, the presence of methaemoglobinaemia should be considered clinically in cases of atypical suspected psychostimulant overdose so that effective and appropriate treatment with reducing agents such as methylene blue can be promptly instituted.

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Johan Duflou

National Drug and Alcohol Research Centre, University of New South Wales, Sydney, NSW, Australia; Sydney Medical School, University of Sydney, NSW, Australia

Contact Prof Johan Duflou.
E-mail: jduflou@forensicmedicine.com.au

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MALT lymphoma with IgM paraprotein and bone marrow involvement mimicking Waldenström macroglobulinaemia

Sir,

Waldenström macroglobulinaemia (WM) is defined in the World Health Organization (WHO) classification as lymphoplasmacytic lymphoma (LPL) associated with serum monoclonal immunoglobulin (Ig) M and involvement of the bone marrow by lymphoma.[1,2] Extranodal marginal zone lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma) is defined as a low-grade lymphoma composed of a heterogeneous population of small B cells. Patients with MALT lymphoma have an indolent clinical course, have bone marrow involvement in less than half of patients, and rarely have a serum IgM paraprotein. Here, we report two patients with MALT lymphoma who had advanced tumour stage and features of WM, including high levels of serum monoclonal IgM and bone marrow involvement by lymphoma.

Case 1 was 64-year-old woman with a history of chronic hepatitis B and C who presented with thyroid-associated orbitopathy—proptosis, swelling of eyelids and blurred vision (OU)—for 3 years. She went to the rheumatology outpatient department for dry mouth. Laboratory examination revealed a high serum IgM level (5740 mg/dL; normal range 40–230 mg/dL) and lymphocytosis (15,500/μL). Furthermore, a serum protein electrophoresis (SPEP) and immunofixation electrophoresis (IFE) showed a monoclonal IgM with λ light chain restriction (Fig. 1A). Computed tomography (CT) showed enlarged lymph nodes in the bilateral axillary, retrocaval, aortocaval and peritoneal regions and diffuse subcutaneous nodules over the chest and abdomen. No hyperviscosity-related symptoms were noted. Incisional biopsy of the right eyelid yielded a diagnosis of MALT lymphoma. Additional pathological examination of the bone marrow and left back subcutis showed involvement by lymphoma. The patient received three cycles of cyclophosphamide, vincristine and prednisone, and six additional cycles of therapy supplemented with rituximab for 6 months. The eyelid swelling improved greatly. Imaging studies showed regression of lymphomatous lesions. The serum IgM level decreased to 132 mg/dL. Follow-up bone marrow aspiration and biopsy were negative for lymphoma. At last clinical follow-up, the patient had been clinically stable for one year after completion of chemotherapy. Pathologically, the right eyelid specimen showed a dense infiltrate of lymphoid cells in a diffuse pattern (Fig. 1B). The tumour cells exhibited predominantly small irregular nuclei with inconspicuous nucleoli and pale (monocytoid) cytoplasm (Fig. 1B, inset). Small numbers of large lymphoid cells (centroblast-like) and plasma cells with Dutcher bodies were present (Fig. 1C). Immunohistochemical analysis showed that the neoplastic cells were positive for CD20 (Fig. 1D) and MNDA (Fig. 1E), but negative for CD5, CD10, and cyclin D1. The plasma cells were positive for monotypic, cytoplasmic λ light chain (Supplementary Fig. 1A,B, Appendix A). Karyotypic analysis of bone marrow aspirate showed 48,XX,+3,+12,t(14;18)(q32;q21.1)[2] (Fig. 1F), consistent with a diagnosis of MALT lymphoma. Paraffin-embedded eyelid tissue was analysed for *MYD88* L265P and *CXCR4* mutations using droplet digital polymerase chain reaction (ddPCR; Bio-Rad Laboratories, USA) which showed negative results (Supplementary Fig. 2, Appendix A).

Case 2 was a 66-year-old woman who had a history of gastric ulcer with *Helicobacter pylori* infection and second degree atrioventricular block treated by permanent pacemaker implantation. She experienced intermittent shortness of breath and epigastric discomfort on hunger for a long time, but did not pay much attention to the symptoms. During follow-up in the cardiovascular outpatient department, chest radiograph revealed consolidation of bilateral lung fields. The patient did not have fever or cough. Laboratory evaluation showed atypical lymphocytes in the peripheral blood smear and a high serum IgM level, 3,220 mg/dL. SPEP and IFE showed a monoclonal IgM with κ light chain restriction (Fig. 2A). The patient then underwent bronchoscopy and biopsy showed MALT lymphoma. Chest and abdominal CT scans showed lymphoma involving mediastinal and retroperitoneal lymph nodes, bilateral lungs and the stomach as well as splenomegaly. Bone marrow examination and gastric biopsy also revealed MALT lymphoma. The patient received three cycles of cyclophosphamide, vincristine and prednisone, and six additional cycles of therapy supplemented with rituximab, as well as triple therapy for *H. pylori* eradication. After 7 months, the patient's serum IgM level decreased to 742 mg/dL. Imaging studies showed obvious regression of lymphoma lesions as well as resolution of the splenomegaly.

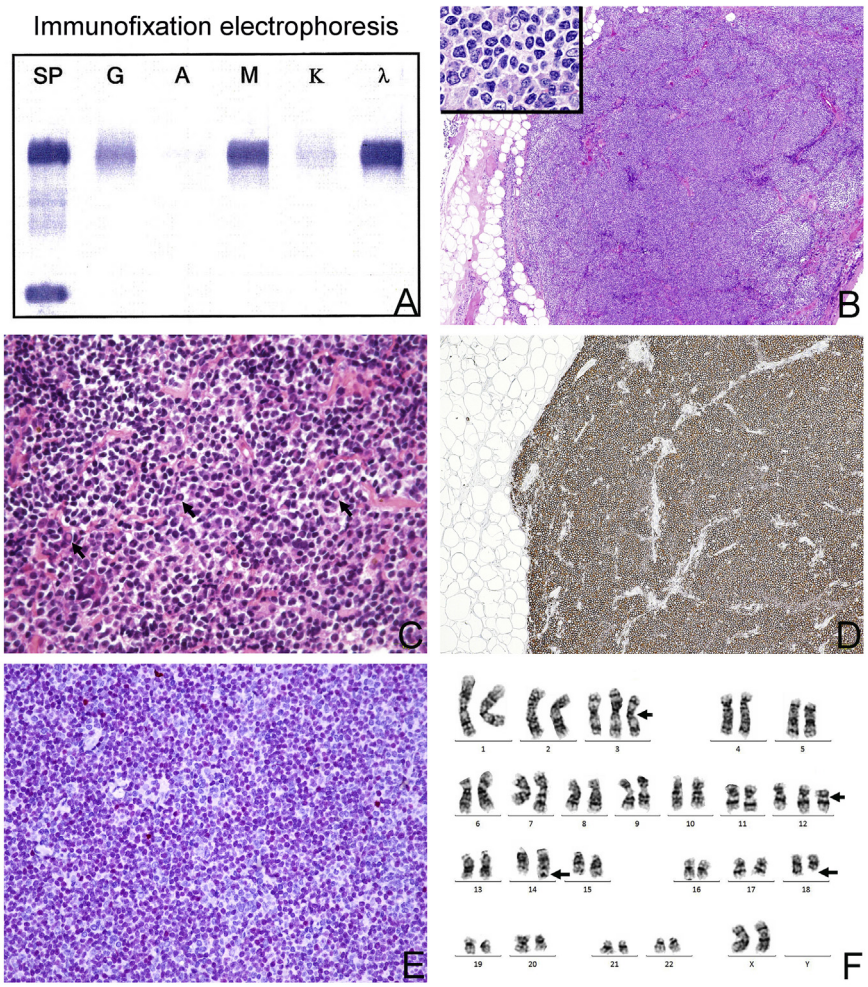

Fig. 1 Clinical and pathological features of Case 1. (A) Immunofixation electrophoresis of serum yields an IgM-λ monoclonal gammopathy. (B) Histology of the eyelid biopsy shows dense infiltration of neoplastic lymphoid cells in a diffuse pattern (H&E). The clear lymphoma cells are evident at right lower field. At higher magnification (inset), the lymphoma cells are mainly small with irregular and angulated nuclei admixed with some immunoblasts or centroblasts. (C) Plasmacytoid cells with Dutcher bodies are discerned (arrow, H&E). (D,E) Immunohistochemically, the tumour cells are positive for CD20 (D) and MNDA (E). (F) Karyotyping from bone marrow tissue yielded trisomy 3 and trisomy 12 (short arrows), and t(14;18)(q32;q21.1) (long arrows).

The patient was clinically stable without recurrence at last follow-up, 11 months after completion of chemotherapy. The gastric biopsy specimen showed an atypical small lymphocytic proliferation with plasmacytic differentiation and scattered large cells (Fig. 2B,C). Immunohistochemical analysis showed that the atypical cells were positive for CD20 (Fig. 2D), CD43, and MNDA (Fig. 2E), and negative for CD5, CD10, CD23 and cyclin D1. The plasma cells were positive for monotypic, cytoplasmic κ light chain (Supplementary Fig. 1C,D, Appendix A). The bronchus biopsy and bone marrow specimens showed involvement by lymphoma. Karyotypic analysis of bone marrow aspirate showed 46,XX,t(11;18)(q21;q21)[4], consistent with MALT lymphoma (Fig. 2F). Examination of paraffin-embedded gastric tissue for *MYD88* L265P and *CXCR4* mutations by ddPCR showed no evidence of mutations (Supplementary Fig. 2, Appendix A).

MALT lymphoma is defined in the WHO classification as an extranodal lymphoma composed of morphologically heterogeneous small B cells including centrocyte-like cells, monocytoid cells, and scattered immunoblast- and centroblast-like cells.[2] The stomach is the most commonly involved site. Other common sites of MALT lymphoma include the ocular adnexae, skin, lungs, salivary glands, breasts, and thyroid gland. Chromosomal translocations, such as t(11;18)(q21;q21), t(14;18)(q32;q21), t(3;14)(p14.1;q32), t(1;14)(p22;q32) occur in 30−40% of MALT lymphomas. These translocations show a strong predilection for anatomical sites. For example, t(11;18)(q21;q21) is mainly found in lung, gastric and intestinal MALT lymphomas; t(14;18)(q32;q21) is usually detected in orbital, salivary gland, and skin MALT lymphomas.[3] An additional subset of cases of MALT lymphoma carry trisomy 3 or trisomy 18.[3] Clinical features reminiscent of WM, including bone marrow involvement and serum monoclonal IgM paraprotein, are rarely observed in patients with MALT lymphoma.

WM is defined in the WHO classification as LPL with bone marrow involvement and an IgM monoclonal gammopathy of any concentration.[1,2] However, rare reports in the literature have described WM caused by other small B-cell lymphomas, such as extranodal marginal zone lymphoma.[4,5] In older literature, WM was regarded as a biological syndrome that could occur in association with different types of low-grade B-cell lymphoma.[6]

In the English literature, we found an additional eight cases of MALT lymphoma with WHO-defined WM features

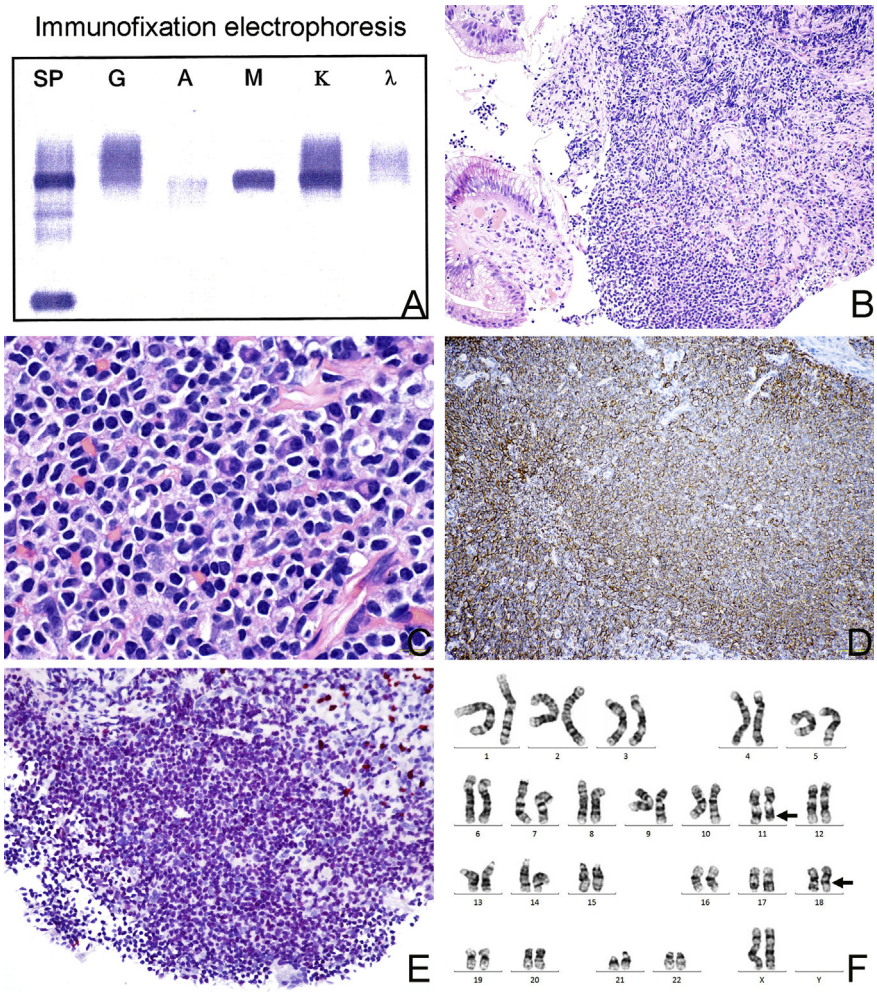

Fig. 2 Clinical and pathological features of Case 2. (A) Immunofixation electrophoresis of serum yields an IgM-κ monoclonal gammopathy. (B) Histology of the gastric biopsy shows dense infiltration of small atypical lymphoid cells (H&E). (C) At higher magnification (H&E), the lymphoma cells show small, angulated nuclei with scant cytoplasm intermixed with some plasma cells. (D,E) Immunohistochemically, the tumour cells are positive for CD20 (D) and MNDA (E). (F) Karyotyping from bone marrow tissue yielded t(11;18)(q21;q21) (arrows).

similar to the two cases we report (Table 1).[4,7–10] All 10 patients had lymphomatous involvement of the bone marrow and an IgM monoclonal gammopathy. The mean patient age was 65.1 years (range 31–80 years) with an equal male-to-female ratio. Association with autoimmunity and presentation with hyperviscosity syndrome were uncommon. Three patients (30%) had history of autoimmune disease, including rheumatoid arthritis, polymyositis, temporal arteritis and thyroid-associated orbitopathy. Two patients (20%) experienced symptoms of hyperviscosity syndrome. In comparison with patients with genuine WM, the frequency of hyperviscosity syndrome was similar (20% versus 10–30% in WM),[11] and the level of IgM paraprotein was mostly above 3000 mg/dL (range 569–8800, mean 3776 mg/dL). The primary sites of lymphoma in the 10 patients included the gastrointestinal tract (n=4), and salivary glands (n=2), with one case each in the nasopharynx, kidney, lung and eye. Regarding the IgM gammopathy, κ and λ light chains were evenly distributed (n=5 each). All 10 patients with MALT lymphoma and features of WM followed an indolent clinical course and were clinically stable with partial or complete remission until the last clinical follow-up (Table 1). These observations suggest that features of WM in patients with MALT lymphoma do not adversely impact the prognosis. Furthermore, a decrease in serum IgM levels could be an indicator of treatment response.

As is acknowledged in the WHO classification, the current definition of WM, although clinically useful, lacks sufficient pathological specificity. For the differential diagnosis of LPL/WM versus MALT lymphoma with WM-like features, we suggest that the results of genetic studies and, in particular MYD88 status, are very helpful. Although MYD88 mutation is not specific for LPL/WM and is well described in a subset of diffuse large B-cell lymphomas and less commonly other B-cell lymphomas, MYD88 mutation is rare in MALT lymphoma. Therefore, the negative results in this case supported the diagnosis of MALT lymphoma with WM-like features in this context. The karyotype of LPL/WM is not specific, but *MYD88* L265P mutations are very common.[12,13] In contrast, a subset of MALT lymphomas is associated with chromosomal translocations or trisomy of chromosome 3 or 18,[3] unlike LPL/WM. Therefore, these genetic features are useful to distinguish MALT lymphoma from WM.

In conclusion, MALT lymphoma may show WM-like features, including bone marrow involvement and the presence of IgM paraprotein. The morphology and immunophenotype of MALT lymphoma and LPL can be indistinguishable. However, conventional cytogenetic analysis and testing for *MYD88*

Table 1 Clinicopathological findings of patients with MALT lymphoma and WM-like features

Case	Age/sex	Autoimmune disease	Hyperviscosity syndrome	Tumour primary site	Paraprotein (mg/dL)	Plasmacytic differentiation	Cytogenetics	Treatment	Prognosis (time)	Reference
1	31/M	Not mentioned	Not mentioned	Stomach	IgM-κ 2900	Yes	Trisomy 3	C/T+SCT	Alive, 36 months	Allez *et al.*[7]
2	71/M	Not mentioned	Not mentioned	Duodenum	IgM-λ 3100	Yes	NA	C/T	Alive, 18 months	Allez *et al.*[7]
3	50/M	Not mentioned	Altered mental status, visual changes, chest pain	Nasopharynx	IgM-κ 8800	Yes	NA	C/T	Alive, unknown	Valdez *et al.*[4]
4	60/F	Not mentioned	Not mentioned	Labial salivary gland	IgM-κ 3200	Yes	NA	C/T	Alive, unknown	Valdez *et al.*[4]
5	79/M	Not mentioned	Not mentioned	Stomach	IgM-λ 5100	Yes	NA	C/T	Alive, unknown	Valdez *et al.*[4]
6	75/F	Rheumatoid arthritis, temporal arteritis	Peripheral neuropathy	Submandibular gland	IgM-λ 3600	Yes	NA	C/T+R	Alive, 6 months	Mikolaenko[9]
7	75/F	Rheumatoid arthritis, polymyositis	Not mentioned	Kidney	IgM-κ 569	Yes	NA	C/T	Alive, 8 months	Chi *et al.*[8]
8	80/M	Not mentioned	Not mentioned	Lung	IgM-λ 1526	Yes	+3,add(9)(p13),+12,der(14)t(14;18)(q32;q21),del(16)(q22),−18,+mar	C/T+R	Alive, 28 months	Akasaka *et al.*[10]
9	64/F	Thyroid-associated orbitopathy	No	Eye	IgM-λ 5740	Yes	+3, +12, t(14;18)(q32;q21.1),	C/T+R	Alive, 18 months	Our case
10	66/F	No	No	Stomach, lung	IgM-κ 3220	Yes	t(11;18)(q21;q21)	C/T+R	Alive, 18 months	Our case

C/T, systemic chemotherapy; MALT, extranodal marginal zone lymphoma of mucosa-associated lymphoid tissue; NA, not available; R, rituximab immunotherapy; SCT, autologous blood stem cell transplantation; WM, Waldenström macroglobulinaemia.

L265P and *CXCR4* mutations are helpful to distinguish these entities and establish the correct diagnosis.

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APPENDIX A. SUPPLEMENTARY DATA

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Shao-Chang Wang[1], Wan-Ting Huang[1], Ming-Chun Ma[2], L. Jeffrey Medeiros[3], Kung-Chao Chang[4]

[1]Department of Pathology and Laboratory Medicine, Chang Gung Memorial Hospital-Kaohsiung Medical Center, Chang Gung University College of Medicine, Kaohsiung, Taiwan; [2]Division of Hematology/Oncology, Department of Internal Medicine, Chang Gung Memorial Hospital-Kaohsiung Medical Center, Chang Gung University College of Medicine, Kaohsiung, Taiwan; [3]Department of Hematopathology, The University of Texas MD Anderson Cancer Center, Houston, TX, USA; [4]Department of Pathology, National Cheng Kung University Hospital, College of Medicine, National Cheng Kung University, Tainan, Taiwan

Contact Kung-Chao Chang, MD, PhD.
E-mail: changkc@mail.ncku.edu.tw

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Audit of stool testing performed in a microbiology laboratory: will using fixed predefined clinical criteria for stool testing of *Giardia* and *Cryptosporidium* lead to missed cases?

Sir,

Giardia lamblia (intestinalis or duodenalis) and *Cryptosporidium* are protozoan parasites which frequently cause diarrhoeal illness. Both have been associated with frequent outbreaks in community and institutionalised settings.[1,2] Several modalities for detection of these parasites are available, ranging from microscopy, antigen assays, enzyme immunoassays (EIA) and multiplex polymerase chain reaction (PCR). Faecal microscopy is time-consuming, operator-dependent and insensitive for detection of these parasites in low burden setting. Faecal testing using other modalities like antigen assay, EIA and multiplex PCR are able to detect these parasites and are incorporated depending on the laboratory size and workflow.

Most microbiology laboratories across Australia have individualised stool testing protocols with some routinely testing for *Giardia/Cryptosporidium* on all specimens with community-onset of diarrhoeal illness while others testing only when there is clinical information/history available. The usual history that prompts such testing is that of persistent or chronic diarrhoea (symptoms $\geq$2 weeks), recent overseas travel and diarrhoea in an immunosuppressed patient, patient from a refugee health clinic or at the request of the local public health unit (these are 'predefined clinical criteria' for our microbiology laboratory).[3] Variations in testing exist as some laboratories perform a broad range multiplex PCR upfront on stool specimens, some perform EIA or antigen-based tests and others faecal parasite multiplex PCR. For the laboratories that do not perform multiplex PCR on all stool specimens, testing for *Giardia/Cryptosporidium* is done at the clinician's request and/or if the predefined clinical criteria are included on the pathology request form. The above testing strategies were found to be widespread after consultation with the representatives from the microbiology laboratories within NSW Health and a few other larger laboratories across Australia.

Electronic ordering is now standard in most Australian hospitals. Figure 1 shows the sequential appearance of the 'pop-up' boxes during the electronic ordering of stool tests. While the 'clinical history' field is crucial, this field (middle box in Fig. 1) can be bypassed with a single character computer entry and other useful information like immunosuppression, recent overseas travel, and onset are not mandatory fields. For laboratories performing *Giardia/Cryptosporidium* testing based on predefined clinical history/criteria, the absence of this information creates difficulty for the laboratory to determine appropriate tests.

We had the opportunity to audit the stool testing in our microbiology laboratory as the faecal testing for *Giardia* and *Cryptosporidium* species was performed on all specimens [except cases of hospital-onset of diarrhoea (diarrhoea $\geq$72 hours after admission)] over a 4-year period (2014–2017). Clinical details of the cases with *Giardia* and *Cryptosporidium* were obtained from electronic medical records (eMR). Ethics approval was obtained as a quality improvement audit. The microbiology laboratory uses Giardia/Cryptosporidium Quik Chek assay (Abbott Diagnostics, USA) for detection. The manufacturer reports the sensitivity and specificity as 97.6% and 100%, respectively. In literature, this test performs better than conventional stool microscopy; however, multiplex PCR has shown even better results with specificity.[4,5] The cost of consumables/test for this assay is AU$10.70.

Over a 4-year period (2014–2017), 7162 faecal specimens were examined in our laboratory. Of these, 35 were positive (non-duplicate) for *Giardia lamblia* and 19 for *Cryptosporidium* (total *n*=54). This gave a 'pick-up' rate for this test of 0.75%. Clinical records were available for 52 of 54 patients (two patients were excluded from analysis). Based on the pathology request forms and electronic orderable only 30% (16/52) of these infections would have been detected. These 16 patients had a history of chronic diarrhoea or had a 'travel history'. Therefore, we reviewed clinical records of all available patients with positive results (*n*=52) to determine further history, treatment given, and outcomes including resolution of symptoms and/or recurrence of an episode. Characteristics and findings of these patients are summarised in Table 1.

Based on information gathered from the eMR audit, parasite testing should have been performed on 25/52 (48%) patients if appropriate history and information were available to the laboratory at the time of performing the test. These additional cases (*n*=9) were patients with chronic diarrhoea (duration >2 weeks), history of travel and immunosuppression, of which four patients represented with recurrent symptoms and were eventually treated. While it is difficult to gauge the clinical impact of performing such testing (and missing the diagnosis) on a retrospective audit, overall, we found that six patients (12%, including the four patients mentioned above) represented back to the emergency department with ongoing symptoms after an initial presentation to the hospital. Definitive therapy with metronidazole and nitazoxanide was prescribed in 12/52 (23%) patients after results of these tests were available to the clinicians, which would not have happened if the *Giardia/Cryptosporidium* testing was carried out. Data on resolution could be obtained on only 32 patients due to the retrospective nature of the audit. All 32/52 (61%) patients showed complete resolution of symptoms. Empirical therapy with metronidazole was only given in 13/52 (25%) patients after presentation with their symptoms as the initial presenting symptoms were thought to be either viral or non-infective.

We also retrospectively extracted information concerning stool consistency from the laboratory database to determine if this could provide a useful laboratory marker to direct testing. The retrospective review of stool consistency showed that 7.4% (530/7162) of all stools and 11% (6/54) of positive stools were categorised as 'formed'. This indicates that if formed stools are used as an exclusion criterion one would still have to test 92.6% of all stools and 11% of positives would be missed. Currie *et al.* and Lindo *et al.* also found that