Acute Lung, Heart, Liver, and Pancreatic Involvements with Hyponatremia and Retinochoroiditis in a 33-Year-Old French Guianan Patient

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Description of Case

A 33-year-old man living in Cayenne, French Guiana, was admitted in the Bichat hospital in Paris with a 3-week history of fever associated with dyspnea and confusion. A chest X-ray previously made in French Guiana showed an alveolar infiltrate of the middle lobe. Amoxicillin and then doxycycline were administered, but did not alleviate the symptoms. Physical examination revealed a loss of 4 kilograms in the last month, confusion, hypotension (98/63 mmHg), tachycardia (126 beats per minute), a red but painless left eye, congestive heart failure, fine crackles in the right lung field, and a one-centimetre-wide left axillary node.

Haematological blood tests revealed a haemoglobin level of 137 g/l with signs of haemolysis (elevated lactic dehydrogenase, low haptoglobin), a total white blood cell count of 11.1×10⁹/L (69% granulocytes, 21% lymphocytes), and thrombocytosis (392×10⁹/L platelets). The blood film showed an altered left ventricular ejection fraction. Physical examination revealed signs of mononucleosis syndrome. Blood chemistry revealed hyponatremia, mild hepatitis, pancreatitis, and elevated cardiac markers. Table 1 summarizes biological data.

Electrocardiogram showed sinus tachycardia. Transthoracic echocardiography revealed an altered left ventricular ejection fraction (35% estimation with Simpson’s method), global hypokinesia, but no signs of endocarditis. Abdominal computed tomography (CT) scanner was normal.

What Further Investigations Would You Perform to Make Etiological Diagnosis?

There are many diagnoses that can lead to subacute fever, hepatitis, pancreatitis, and pneumonia in a patient coming from the Amazonian region. Blood cultures, thick and thin smears for malaria, and intradermal purified protein derivative test for tuberculosis were negative. Mycobacterium tuberculosis was not detected on gastric aspirates. Urines were sterile, and antigen detection for Streptococcus pneumoniae and Legionella pneumophila was negative. HIV, hepatitis viruses (A, B, and C), Coxiella burnetii, Rickettsia conorii, Salmonella typhi and paratyphi, Brucella sp., Dengue fever, and Mumps virus serologies were also negative. Chlamydia pneumoniae and Mycoplasma pneumoniae serologies were positive with the presence of both IgM and IgG. Epstein-Barr virus (EBV)-IgM and IgG anti-VCA antibodies were also found, but detection of EBV and Cytomegalovirus (CMV) DNA by polymerase chain reaction (PCR) was negative. Since the patient had severe cardiac dysfunction, Chagas’ disease was also looked for, but antibodies against Trypanosoma cruzi were absent. Cerebrospinal fluid analysis revealed 8 cells/mm³, hypoglycorrhachia (2.3 mmol/L with venous glycemia 5.5 mmol/L), hyperproteinorachia (0.54 g/L), sterile culture, and negative pneumococcal and cryptococcal antigen detection. Toxoplasma gondii, Herpes simplex virus (type-1 and -2), EBV, and Varicella-zoster virus DNA were not detected by PCR in the cerebrospinal fluid.

What Finally Led to Diagnosis?

The patient reported the consumption of semi-rare game (Brazilian Tapir, Taphirus terrestris, locally known as Matpouri) 3 weeks before the beginning of symptoms, pieces of which were still kept frozen.

Ophthalmologic examination showed a corneal ulcer associated with multiple foci of retinochoroiditis in the left eye, which is suggestive of toxoplasmosis.

Systematic exploration of the causes of mononucleosis syndrome led (besides HIV, EBV, and CMV) serologies to T. gondii serology, which was positive with the presence of IgG (63 IU/ml), high levels of IgM (index of 42.7, Vidia Biomerieux), and IgA (12/12 with ISAGA Biomerieux test). The avidity of anti-toxoplasmosis IgG was low (4% Vidia Biomerieux). These data suggested recent toxoplasmosis infection. Table 1 summarizes serological data.

How Can Hyponatremia and Hemolysis Be Explained?

Since blood osmolality was low (259 mosmol/L) and urine osmolality was high (508 mosmol/L), the syndrome of inappropriate antidiuretic hormone secretion (SIADH) was suspected. Cortisol response to synacthen test and level of serum thyroid-stimulating hormone were normal. No medication causing hyponatremia as a side effect was taken. Brain magnetic resonance imaging was normal,

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and full-body CT scan did not suspect cancer. SIADH diagnosis was confirmed and related to pneumonia. There were no schizocytes on the blood film. Direct Coombs’ test and hemoglobin electrophoresis were negative or normal. Pyruvate kinase deficiency and Mankowski-Chauffard disease were also looked for but excluded. Glutathione stability test revealed mild g6pd deficiency (7% g6pd activity). The patient, though living in French Guiana, was of Chinese descent, and g6pd deficiency is frequent in Asia. Amoxicillin and doxycycline are suitable for g6pd patients, and we can imagine that sepsis favored haemolysis.

**How Would You Manage the Patient?**

When *C. pneumonia* and *M. pneumoniae* serologies came back positive, macrolide therapy (roxithromycine 300 mg/day) was started. However, physicians doubted that these serological data could explain the whole symptoms. Three weeks after the first serologies, *C. pneumonia* and *M. pneumoniae* antibodies’ rates did not rise, pointing out a cross-serological reactivity with *T. gondii* antibodies. The patient was in a critical condition and despite mild g6pd deficiency, gold standard anti-toxoplasmosis treatment [1] was promptly started after the serological diagnosis of recent *T. gondii* infection. The patient’s condition improved with pyrimethamine (100 mg/day for the first 2 days followed by 50 mg/day), sulfadiazine (6 g/day), and folic acid (25 mg/day). There was no increased haemolysis with sulfadiazine, but this drug was stopped and replaced by clindamycin (2.4 g/day) due to hepatotoxicity. Anti-*Toxoplasma* drugs were given for a total of 6 weeks.

A salt-free diet, furosemide, and increased doses of perindopril and bisoprolol were also administered. Heart rate diminished, congestive heart failure symptoms disappeared, and brain natriuretic peptide and troponin levels normalized. Periodic transthoracic echocardiogram evaluations were performed and LVEF measures reached 45% at the patient’s discharge.

When furosemide was stopped, and thanks to fluid deprivation, natremia normalized. The patient recovered his full intellectual abilities.

With anti-*Toxoplasma* treatment and Vitamin A ointment, cornea ulcer and chorioretinitis healed without visual aftereffect.

Lipase rose to 17N but decreased under antiparasitic therapy.

**Additional Molecular and Parasitological Investigations**

A quantitative PCR-based assay detected the 200- to 300-fold repetitive 529 bp DNA fragment of *T. gondii* in a blood sample collected 3 days after admission. This blood sample was also inoculated into mice. Four weeks post-inoculation, all mice tested positive for *T. gondii* infection and showed signs of disease due to a virulent *T. gondii* strain. All surviving inoculated mice were sacrificed and numerous *T. gondii* cysts were microscopically observed in the brain samples. Cell cultures were inoculated with trypsinized cysts of *T. gondii*, and the strain was named BRC TgH41001.

Genotyping analysis with 15-microsatellite markers [2] of the BRC TgH41001 strain was performed on DNA extracted from cysts collected at first isolation in mice. In each pair, one primer was 5’-end labelled with fluoroscein (6-FAM, HEX, or NED) to allow sizing of PCR products.

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**Table 1. Laboratory findings.**

| Biological Characteristics | Day 1  | Day 6  | Day 19 | Day 41 | Day 60 | Normal Values |
|----------------------------|-------|-------|-------|-------|-------|---------------|
| Natremia (mmol/L)          | 127   | 125   | 133   | 140   | 140   | 135−145       |
| Blood osmolality (mosmol/L)| 259   | na\(^1\) | na   | na   | na   | 300−310       |
| Urine osmolarity (mosmol/l)| 508   | na   | na   | na   | na   | 500−100       |
| Thyroid stimulating hormone (mIU/L) | 1.9 | na | na | na | na | 0.3−5.0 |
| Cortisol\(^2\) (nmol/L)    | 598   | na   | na   | na   | na   | 138−745       |
| Aspartate aminotransferase (IU/L) | 118 | 83  | 233  | 55   | 46   | <40          |
| Alanine aminotransferase (IU/L) | 97  | 86  | 189  | 46   | 45   | <40          |
| Gamma-glutamyl transpeptidase (IU/L) | 127 | 76 | 245  | 27   | 27   | <58          |
| Alkaline phosphatase (IU/L) | 66    | 51   | 119  | 34   | na   | <34          |
| Total bilirubin (micromol/L) | 17    | 13   | 9    | 10   | na   | <17          |
| Troponin (microg/L)       | 1,590 | 0.36 | na   | 0.05 | na   | <0.6         |
| Brain natriuretic peptide (ng/L) | 1,510 | 274 | na   | 258  | na   | <100         |
| Lipase (IU/L)             | 503   | 1,142 | 771  | 283  | 0    | <60          |
| Lactic dehydrogenase (IU/L)| 1,978 | na   | na   | na   | na   | <470         |
| Haptoglobin (g/L)         | 0.1   | na   | na   | na   | na   | 0.64−1.7     |
| Creatinine phosphokinase (IU/L) | 340 | na | na | na | na | 10−200       |
| Anti-Toxoplasma IgG (IU/mL)\(^3\) | 68  | 147  | 199  | 785  | 294  | positive if >4 IU/mL |
| Anti-Toxoplasma IgM (index)\(^4\) | 42.7 | 45   | 38   | 45   | 45   | positive if >0.75 |
| Anti-Toxoplasma IgG Avidity index\(^4\) | 0.04 | na | na | na | na | recent infection if <0.3 |

**Notes:**

\(^1\) Not available.

\(^2\) 8:00 am measurement.

\(^3\) Vidas Biomerieux.

\(^4\) Vidas Biomerieux.

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with an automatic sequencer. PCR was carried out in a 25-μL-reaction mixture consisting of 12.5 μL of 2X QIAGEN Multiplex PCR Master Mix (Qiaegen) and 5 pmol each primer. Cycling conditions were 15 min at 95°C, 30 s at 94°C, 3 min at 61°C, 30 s at 72°C (35 cycles), and 30 min at 60°C. PCR products were diluted in desionised formamid with a dye-labeled size standard (ROX 500, Applied Biosystems) and electrophoresed at 3100, Applied Biosystems). The sizes of the alleles in base pairs (bp) were estimated using GeneMapper analysis software (version 4.0, Applied Biosystems).

Total DNA was extracted on eight samples of the frozen Mailpouri meat and submitted to a quantitative PCR-based assay targeting the 200- to 300-fold dilution of the frozen Maı̈pouri meat and samples of the frozen Maı̈pouri meat testing for Toxoplasma infection (Daniel Ajzenberg, personal communication).

| Type | Isolate | Origin | Host | TUB2 | W35 | Tgm-A | B18 | B17 | M33 | IV.1 | XL1 | M48 | M102 | N60 | N82 | AA | N61 | N83 |
|------|---------|--------|------|------|------|-------|-----|-----|-----|-----|-----|-----|------|-----|-----|----|-----|-----|
| Atypical | BRC TgH41001 | French Guiana | Human | 291 | 246 | 203 | 158 | 346 | 169 | 272 | 356 | 229 | 172 | 142 | 105 | 261 | 105 | 316 |
| I | CT1 | USA | Cow | 291 | 248 | 209 | 160 | 342 | 169 | 274 | 358 | 209 | 168 | 145 | 119 | 265 | 87 | 306 |
| II | PTG | USA | Sheep | 289 | 242 | 207 | 158 | 336 | 169 | 274 | 358 | 215 | 174 | 142 | 111 | 265 | 91 | 310 |
| III | Ctg | USA | Cat | 289 | 242 | 205 | 160 | 336 | 169 | 274 | 354 | 219 | 174 | 151 | 119 | 259 | 79 | 332 |
| Atypical | COUGAR | Canada | Cougar | 289 | 242 | 205 | 158 | 336 | 169 | 274 | 354 | 219 | 174 | 151 | 119 | 259 | 79 | 332 |
| Atypical | TgCatBr1 | Brazil | Cat | 289 | 242 | 205 | 160 | 342 | 165 | 278 | 358 | 233 | 164 | 147 | 111 | 316 | 89 | 308 |
| Atypical | TgCatBr3 | Brazil | Cat | 289 | 242 | 205 | 160 | 348 | 165 | 278 | 356 | 213 | 190 | 142 | 111 | 263 | 113 | 312 |
| Atypical | TgCatBr5 | Brazil | Cat | 291 | 242 | 205 | 160 | 362 | 165 | 278 | 356 | 237 | 174 | 140 | 111 | 265 | 89 | 314 |
| Atypical | RUB | French Guiana | Human | 289 | 242 | 205 | 170 | 360 | 167 | 274 | 356 | 223 | 190 | 142 | 109 | 259 | 85 | 312 |
| Atypical | VAND | French Guiana | Human | 291 | 242 | 203 | 162 | 344 | 167 | 276 | 356 | 217 | 170 | 142 | 113 | 277 | 91 | 308 |
| Atypical | BRC TgH18001 | French Guiana | Human | 289 | 246 | 203 | 160 | 344 | 167 | 272 | 356 | 229 | 176 | 142 | 113 | 263 | 85 | 312 |
| Atypical | BRC TgH18002 | French Guiana | Human | 289 | 246 | 203 | 160 | 337 | 165 | 274 | 356 | 209 | 172 | 136 | 111 | 251 | 109 | 310 |
| Atypical | BRC TgH18003 | French Guiana | Human | 291 | 242 | 203 | 160 | 339 | 165 | 272 | 358 | 221 | 174 | 138 | 107 | 277 | 95 | 312 |

1 PTG is a clone of the ME49 strain; CTG is also known as CEP or C strain; COUGAR is also known as TgGcCa1 or COUG strain; BRC TgH18001 is also known as GUY-DOS or GUY-2001-DOS strain; BRC TgH18002 is also known as GUY-KOE or GUY-2002-KOE strain; BRC TgH18003 is also known as GUY-MAT or GUY-2002-MAT.

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**Why Was the Patient’s State So Severe?**

One of the evolutionary strengths of *T. gondii* is its ability to adapt its reproductive behaviour in different environments. In North America and Western Europe, it benefits from ancient agricultural habits of breeding of a limited number of species, which provide a stable environment. The latter is ideal for asexual reproduction, which provide a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. The latter is ideal for asexual reproduction, which provides a stable environment. 

On the contrary, the Amazonian rainforest ecosystem is a hotbed of diversity. In order to survive in such an environment, *T. gondii* uses preferentially sexual reproduction in wild felids' gastrointestinal tract to generate atypical strains with highly divergent genotypes [5]. The report of an atypical *T. gondii* strain isolated in a free-living jaguar in French Guiana supports the existence of a *T. gondii* wildlife cycle and then genetic diversity [6]. Since 1998, several severe (and sometimes lethal) cases of acute toxoplasmosis in immunocompetent subjects involving such strains have been reported in French Guiana [7–10]. They disseminate via parasitaemia to multiple organs and especially to the lungs [10].

So far, there are only epidemiological and clinical data [10] supporting the fact that atypical Guianan strains are more virulent in immunocompetent patients than other *T. gondii* strains. Their high genetic diversity may provide them with enhanced invasive abilities, or it could interfere with the Th1 host immune response, as suggested by experiments in mouse models [11–13]. However, evidence has also been given indicating interindividual variability after infection to a same Guianan strain in an outbreak [14]. Biochemical mechanisms of virulence have not yet been studied in...
humans, but experimentally, ROP proteins seem to be essential for invasion and maintenance of the parasitophorous vacuole membrane. It has been shown that the overexpression of ROP18 or the transfection of the ROP18 gene into a nonpathogenic T. gondii strain enhances mortality in a mouse model [11–13]. Further studies, such as the ROP18 gene sequencing of atypical Guianan strains, would be of high scientific value.

Source of Infection

There is no formal evidence that the Maïpouri meat was the source of infection. Nonetheless, many strong arguments support this view. First, previous studies [9] demonstrated that consumption of game from the Amazonian forest was strongly associated with the risk of developing 10 to 20 days later severe toxoplasmosis. Second, we have proved that there was T. gondii DNA in the Maïpouri sample. Third, the patient ate undercooked meat, which makes highly likely the fact that he ingested living T. gondii cysts. Finally, and despite thorough questioning, we could not identify any other potential source of T. gondii infection. The patient was living in an urban area, without any pets, and has never travelled to the Amazonian rainforest. He drank only boiled water and did not report any other raw-meat consumption in the month before the beginning of symptoms besides the Maïpouri steak. To our knowledge, evidence has never been published demonstrating that a piece of meat was the source of human Toxoplasma infection by matching the parasite’s genotype from the patient’s biological sample and from a piece of meat likely to be the source of infection.

Among the five people who shared the Maïpouri dish, only two ate undercooked meat; the patient and his father. The latter remained asymptomatic, but he refused to perform a test to check his serological status against toxoplasmosis. Only hypotheses can be made to explain why he remained healthy. Either his steak was free of T. gondii cyst or most probably, thanks to his age, he was already immunized against toxoplasmosis.

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Key Learning Points

- Atypical and highly virulent strains circulating in the Amazonian rainforest ecosystem
- To date only described in French Guiana but likely underreported in other countries of the Amazonian area
- Lung involvement is frequent and lethal cases have been described
- Specific anti-toxoplasmosis treatment should be promptly started immediately after serological results, without waiting for positive PCR or parasitic isolation

Conclusion

In medical practice, T. gondii is mainly a health concern in pregnant women and in immunosuppressed patients. In immunocompetent patients, acute toxoplasmosis is generally asymptomatic or associated with benign symptoms such as prolonged fever, polyadenopathy, and myalgia. Severe complications, such as pneumonia, myocarditis, or meningo-encephalitis, are infrequent. Eye involvement is likely underestimated, and the burden of ocular toxoplasmosis seems to be extremely high in certain tropical areas of South America [15].

To date, more than a hundred cases of acute toxoplasmosis in immunocompetent patients (adults and children) due to atypical strains of T. gondii have been reported in French Guiana (M. Demar, personal data). Approximately 40 have been published. It is highly likely that these atypical and virulent strains of T. gondii also exist in other Amazonian countries than French Guiana, but clinical cases are probably underreported.

Physicians should systematically consider acute toxoplasmosis as a possible diagnosis for any infectious syndrome with visceral (especially lung) involvement in patients who have recently travelled to or lived in the Amazonian area. Since lethal cases have been reported and since treatment associating pyrimethamine and sulfadiazine is efficient, such treatment should be promptly started immediately after serological results, without waiting for positive PCR or parasitic isolation.