Recurrence of acute suppurative thyroiditis in a young man.

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Acute thyroiditis, also referred to as suppurative or infectious thyroiditis, is a rare thyroid disorder usually caused by bacteria, mycobacteria, fungi or protozoa invasion of the thyroid gland[1]. This condition is usually caused by infection deriving from infected tissue that surrounds the thyroid gland or from an internal fistula from the remnants of the fourth pharyngeal pouch. In some instances, it is provoked by metastatic seeding of bacteraemia or direct trauma caused by fine needle aspiration or by persistent thyroglossal duct. Penetrating oropharyngeal injuries resulting from swallowed foreign bodies provide an additional channel of infection into a relatively resistant thyroid gland. Recurrent acute suppurative throat infections are frequently associated with branchial fistulas; they have usually been reported in young patients and, rarely, in adults[2].

H.M., a 31-year-old man from Albania, was admitted to a Department of Medicine of the Careggi University Hospital of Florence (Italy), complaining of fever, painful swelling of the front part of the throat and severe dysphagia for 1 week. Similar symptoms had recurred every 6 months during the last 2 years. The patient had been previously treated with unspecified drugs.

At the time of admission, body temperature was 39°C; the patient was unable to eat because of painful dysphagia and swallowing was difficult as well. On physical examination, the front of the throat, mainly on the right side, was hard and painful. The skin was hot and erythematous; the patient was unable to put his neck backwards. The pharynx was red and the tonsils were hypertrophic. No symptoms or signs of hyperthyroidism or of hypothyroidism were present. Assuming that the patient had an acute thyroid gland infection, oral treatment with amoxicillin/clavulanic acid (2 g daily) and with aspirin (600 mg daily) was immediately started. Blood tests revealed neutrophilic leucocytosis, elevated erythrocyte sedimentation rate and markedly increased acute-phase proteins, such as ferritin, α-macroglobulins and fibrinogen (Table 1).

The patient's routine screening tests for hepatitis B virus detected low levels of HBsAg, anti-HBe and anti-HBc in the serum; HbeAg, anti-HbsAg, IgM anti-HbcAg were negative; cold agglutinins were weakly positive and serum alanine aminotransferase was 179 IU/l. No other viral or bacterial infections were found; the other blood tests and enzyme values were normal. Free T3, T4 and thyroid-stimulating hormone were in the normal range; thyroid antibodies (antiperoxidase and antithyroglobulin) were negative, whereas thyroglobulin serum levels were significantly increased (Table 2). Urine test revealed the presence of proteins (70 mg/dl) and increased urobilinogen levels (> 2).

Throat ultrasonography revealed an enlarged thyroid gland with abnormalities in the internal structure; the left thyroid lobe was enlarged and showed inhomogeneous and thickened texture and peripheral vascularisation (Fig. 1). The right middle-lower lobe showed a large mass that extended towards the isthmus with a textureless area and high-reflecting pieces inside, findings consistent with suppurative thyroiditis. The soft tissue that covered the thyroid gland appeared abnormal as well and right-sided lateral-cervical

Table 1. Blood tests on admission and at discharge.

	Admission	Discharge
ESR (mm/h)	105	36
Fibrinogen (mg/dl)	650	260
α-Macroglobulins (g/dl)	1.68	0.52
Ferritin (µg/l)	452	253
White blood cell count (/mm^3)	18.200	7000
Neutrophils (%)	81	59
Platelets (/mm^3)	648.000	202.000

ESR, erythrocyte sedimentation rate.

Table 2. Thyroid hormones and thyroid antibodies on admission.

FT3	3.6 pmol/l (normal range 3.5-6.4 pmol/l)
FT4	18.4 pmol/l (normal range 0.3-19.4 pmol/l)
TSH	0.57 mU/l (normal range 0.25-3.5 mU/l)
Antiperoxidase antibodies	< 20 U/ml (normal range <40 U/ml)
Antithyroglobulin antibodies	< 10 U/ml (normal range <35 U/ml)
Thyroglobulin	141.9 ng/ml (normal range <48 ng/ml)

TSH, thyroid-stimulating hormone.

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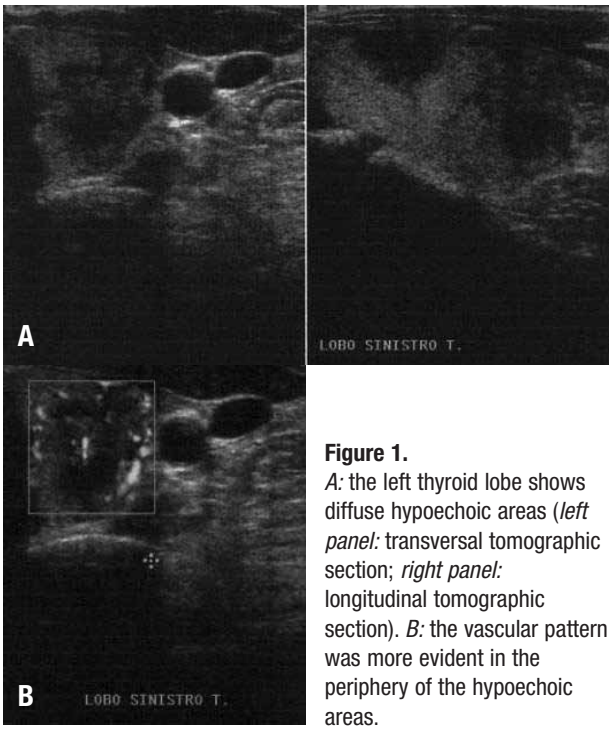

Figure 1.
A: the left thyroid lobe shows diffuse hypoechoic areas (*left panel:* transversal tomographic section; *right panel:* longitudinal tomographic section). *B:* the vascular pattern was more evident in the periphery of the hypoechoic areas.

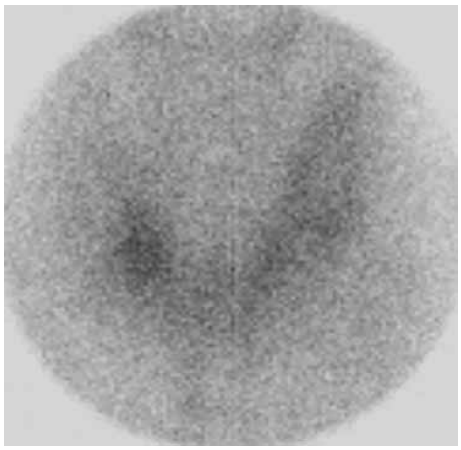

Figure 2. Thyroid scan with ^{99m}Tc shows a very low-uptake level.

reactive lymph nodes of 1 cm in diameter were found. Thyroid ^{99m}Tc scan showed only a very low uptake in the whole gland (Fig. 2).

On the sixth day after admission, symptoms markedly decreased; a few days later, acute inflammatory signs disappeared, throat swelling decreased, the patient was apyretic and started eating normally. Acute phase reactants, alanine aminotransferase, erythrocyte sedimentation rate levels and neutrophil count were also reduced.

The diagnosis of acute (suppurative) thyroiditis is quite difficult on clinical grounds, because of the relative rarity of the disease. However, the simultaneous presence of symptoms (pain in the region of the thyroid gland and dysphagia) and signs (lymphadenopathy, redness and tenderness of the front of the throat) of severe local inflammation may help the physician to suspect this condition.

Systemic signs like fever and/or malaise frequently occur. Usually, no signs of hyperthyroidism or of hypothyroidism are present: the evidence of the patient's thyrotoxic status steers the physician into a different diagnosis, such as a subacute thyroiditis. However, subacute thyroiditis shows normal or near-normal leucocyte count, transient elevation of antithyroid antibodies in low titres and abnormal T3 and T4 serum levels, either elevated or reduced depending on the phase of the disease. Moreover, subacute granulomatous thyroiditis usually disappears in 10-12 weeks; conversely, our patient showed some episodes of thyroiditis over a long period of time (> 28 months). There was no such evidence in the observed patient.

Suppurative thyroiditis is most commonly found in young or very young patients (ranging from 3 to 14 years, median 6 years) due to an embryonic disorder such as pyriform sinus fistulae or remnant of the fourth branchial arch. The disorder seems to be equally present in males and females (male to female ratio is about 1.5:1.2)[3]. The left lobe of the thyroid seems to be preferentially affected in the majority of patients (90%), whereas the right lobe is affected in only about 10% of patients; some cases are bilateral. Infection recurrence often develops in the same site of the gland. Only 15 adult patients out of 110 reported cases have been described in the literature since 1933[2].

Recurrence is due to the presence of a pyriform sinus-thyroid gland fistula, mainly (90%) during childhood (during the first decade of life) and more rarely in the adult age (8%)[4-6]. The fistula seems to be related to the ultimobranchial body of the fourth branchial pouch[7].

Diagnosis of acute suppurative thyroiditis relies on laboratory findings of significant leucocytosis and normal thyroid function status. It has been suggested that fine needle aspiration and culture of the aspirate is the first diagnostic step when a suppurative thyroid disorder is suspected. Since our patient had such a rapid clinical response after starting nonsteroidal anti-inflammatory and antibiotic drugs, it was not deemed necessary to perform fine needle aspiration, because the patient who was feeling well refused the test. Thus, we were unable to identify the bacterial agents.

However, the clinical status, the ultrasound examination of the thyroid and the overall tests seem to confirm the clinical diagnosis. Thyroid scan with ^{99m}Tc was not clear because of the low-uptake level, probably due to the extension (both thyroid lobes were affected) and severity of inflammation. We suspected the presence of a pyriform sinus fistula because of the recurrence of the

episodes. However, in order to identify the fistulous tract, it is important to wait for a clinical quiescent stage, so that oedema occurring during the suppurative phase does not obstruct the fistula orifice.

For these reasons, in our patient, we performed direct laryngoscopy, barium swallow examination and computed tomographic scan several weeks after the acute episode. Nevertheless, we were unable to identify the fistula orifice. We performed a second computed tomographic scan using air as contrast agent, during a well performed Valsalva manoeuvre[8]; however, also with this test, we did not find any anatomical alterations accounting for the recurrent infection. At the same time, we did not find any other cause of bacterial inflammation. Dental and echocardiographic examinations were completely normal and penetrating oropharyngeal injuries were excluded. However, penetrating lesions are not usually linked to recurrent infective acute episodes, in contrast to what observed in our subject. When suppurative recurrent thyroiditis is due to the presence of a pyriform sinus fistula, final therapy is the excision of the sinus tract, so as to prevent recurrence, after incision and drainage of the intrathyroidal abscess. Large-spectrum antibiotic and nonsteroidal anti-inflammatory drugs, commonly used in view of definitive surgery, may sometimes help physicians to obtain a well being status, until a definitive diagnosis of the fistula is made.

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Peripheral arteriopathy of the lower limbs in a patient with severe congenital thrombocytopenia

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When a blood vessel is injured, platelets adhere to the exposed subendothelium, are activated and secrete their granule contents, including some platelet agonists that, by interacting with specific platelet receptors, contribute to the recruitment of additional platelets to form aggregates. In addition, platelets play a role in the coagulation mechanisms, providing the necessary surface of procoagulant phospholipids. Congenital or acquired abnormalities of platelet number or function are associated with an increased risk of bleeding, thus confirming the important role played by platelets in haemostasis[1]. Typically, patients with platelet disorders have mucocutaneous bleedings of variable severity, and excessive haemorrhage after surgery or trauma. No information is available on a possible association between thrombocytopenia and cardiovascular disease.

A 57-year-old male patient was referred to our Thrombosis Centre after the discharge from a medical department where a diagnosis of severe peripheral arteriopathy with Leriche syndrome had been made. He reported the onset of intermittent claudication of the lower limbs 1 year before with rapid worsening of symptoms with a claudication distance of < 100 m. Ultrasound and angiographic tests revealed that, beyond

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the origin of renal arteries, the abdominal aorta was dilated and completely occluded by a thrombus. Iliac arteries were reperfused by mammalian and epigastric arteries. No other obstruction was detected upon examination of the lower limb arteries, whereas the internal carotid arteries showed stenosis with a lumen reduction of 50% in the right carotid and of 65% in the left carotid artery. Physical examination revealed normal findings, in particular no spleen enlargement was detected. The patient was a heavy smoker (20 cigarettes/day) and he had stopped smoking only a few months before the onset of lower limb claudication. He had a normal body mass index (24.3 kg/m^2), was normotensive and normo-cholesterolaemic (total cholesterol 177 mg/dl, low-density lipoprotein [LDL] cholesterol 105 mg/dl). He reported good health notwithstanding he was affected by severe thrombocytopenia (platelet count 1-3 x 10^9/l). Thrombocytopenia was discovered at the age of 3, when the first blood cell count was performed. All the subsequent platelet counts demonstrated values < 20 x 10^9/l, and in recent years persistently < 10 x 10^9/l. Despite severe thrombocytopenia, he never had relevant haemorrhagic events: he reported occasional mucocutaneous bleedings in childhood, which became less frequent in the adult age. There were only two clinically relevant events: one at the age of 14, when he had smallpox with haemorrhagic skin lesions, and the other at the age of 3, when he had a small macular retinal haemorrhage. Tooth extractions were done without any excessive bleeding, but he never required surgery. Family history was negative for bleeding diathesis and his parents and sister had normal platelet counts. He led a normal life, got married and had a daughter with normal platelet count, and he had decided to withdraw any further clinical control. However, the recent onset of arteriopathy required medical attention and the detection of severe thrombocytopenia (1-3 x 10^9/l) led to the hypothesis of autoimmune thrombocytopenic purpura. On this basis, he received immunosuppressive therapy with prednisone 1 mg/kg and intravenous immunoglobulins, but both treatments did not have any effect on the platelet count.

Standard morphological examination of blood smears confirmed the diagnosis of severe thrombocytopenia, and evidenced an increased platelet size with several giant platelets. Apart from increased size, platelet morphology was normal. No alterations in leucocyte and erythrocyte morphology were observable, with the only exception of a mild red blood cell anisopoikilocytosis. Bone marrow aspirate and biopsy showed normocellularity and no cytomorphological abnormalities of haemopoietic precursors. In particular, megakaryocytes presented normal number, size and morphology, with well-developed platelet fields.

On the basis of the patient's medical history and haematological picture, a diagnosis of congenital thrombocytopenia was considered. According to the diagnostic algorithm recently published by the Italian "Gruppo di Studio delle Piastrine"[2], history and physical examination did not identify any of the clinical features associated with the decreased platelet count in syndromic forms of inherited thrombocytopenia. Further examination allowed excluding the diagnosis of any described form of non-syndromic inherited macrothrombocytopenia with giant platelets. In fact, flow cytometry showed that membrane glycoproteins Ib/V/IX were all normally expressed, thus excluding Bernard-Soulier syndrome. Macrothrombocytopenia related to mutation of the *MYH9* gene was ruled out by immunofluorescence studies, which demonstrated a normal distribution of NMMHC-IIA within the cytoplasm of polymorphonuclear granulocytes. On May-Grunwald-Giemsa-stained blood smears, platelets showed a normal content in azurophilic granules, which were organised in granulomeres with normal structure, thus excluding the diagnosis of grey platelet syndrome. The absence of anaemia, signs of haemolysis and/or thalassemia allowed ruling out the diagnosis of X-linked thrombocytopenia with thalassemia or dyserythropoietic anaemia with thrombocytopenia due to mutation of the gene for transcription factor GATA-1. Based on these findings, all known forms of genetic thrombocytopenia have been excluded.

We then performed tests for acquired and congenital thrombophilia. Serum levels of antithrombin, protein C, and protein S were within the normal range. The search for factor V Leiden mutation and for G20210A prothrombin gene mutation was negative. No lupus anticoagulant or anticardiolipin antibodies or anti-beta2GP1 antibodies were detected. Homocysteine serum levels were 14.1 μmol/l (normal value < 19 μmol/l) and homocysteine metabolism after methionine loading was normal as well as the serum levels of factor VIII. On the contrary, lipoprotein(a) serum levels were higher (572 mg/l, normal value < 300 mg/l) and D-dimer plasma levels resulted 2-fold higher than normal.

Treatment with simvastatin 20 mg/day was started and, after 6 months, total cholesterol reached 145 mg/dl and LDL cholesterol 75 mg/dl. Treatment with losartan 50 mg/day was also started, aimed at giving further protection against the occurrence of stroke.

After 1 year of treatment with losartan and simvastatin, the ultrasound examination of the carotid arteries demonstrated a stability of the stenosis and the patient was asymptomatic for cardiac or cerebral ischaemia. In addition, the length of walk without pain was increased (> 250 m) with a significantly improved quality of life and no occurrence of bleeding events.

Inherited thrombocytopenias are rare disorders that are usually classified according to both platelet size and the presence (syndromic) or the absence (non-syndromic) of clinical features other than those deriving from platelet defects[3]. However, more than one third of patients remains without a definite diagnosis despite accurate investigation, since their disease does not meet the diagnostic criteria for any of the described forms[4]. This indicates that several genetic platelet disorders are still waiting to be characterised and that one of them probably affected the patient described in this paper. He had severe atherosclerosis of the abdominal aorta and of the carotid arteries despite severe thrombocytopenia. To the best of our knowledge, this association has not been previously described. Surgical treatment of arteriopathy was excluded due to thrombocytopenia. For the same reason, no antithrombotic drugs, which represent a cornerstone in the treatment of arterial disease, could be used. The preventive strategy in this high-risk patient with a clear contraindication to antithrombotic drugs should be optimised by different ways. Risk factors for cardiovascular disease should be modified in order to reduce rapid progression of the disease, in particular by decreasing cholesterol serum levels. Although the patient had normal cholesterol values at baseline, a treatment with simvastatin was started and LDL cholesterol was reduced to 75 mg/dl, according to recent evidence suggesting that a reduction in LDL cholesterol serum levels < 70 mg/dl should be obtained in high-risk patients[5,6]. Statin therapy has been demonstrated to improve the clinical outcome of atherosclerosis independently of a reduction in serum cholesterol levels. Statins induce a decrease in the levels of inflammatory biomarkers that are related to the beneficial effects of these drugs on the progression of atherosclerosis[7]. In addition, recent evidence suggests that the angiotensin II receptor antagonist losartan may offer advantages beyond blood pressure lowering in hypertensive patients[8]. A reduction in arterial stiffness and the inhibition of the deleterious effects of angiotensin II on endothelial function are some of the mechanisms proposed to explain the gain in stroke protection obtained in patients treated with these drugs. In keeping with this evidence, we started treatment with losartan to obtain a possible further reduction in the risk of stroke.

The other interesting issue in the history of this patient is the absence of clinically relevant bleeding events. In fact, despite he led a normal life until 57 years, no serious bleeding episodes occurred, suggesting that the bleeding risk is not related to the extremely low platelet count, although the reason for this behaviour remains unknown. It is well known from platelet kinetic studies that approximately one third of the platelet mass is stored in the spleen, and this percentage raises up to 90% in patients with hypersplenism[9]. However, this patient had normal sized spleen and the total platelet mass should therefore be estimated very low. The lifelong severe reduction in platelet count did not protect the patient against the development of serious thrombotic disease, suggesting that a more than 10-fold lower platelet count is sufficient for thrombus formation on atherosclerotic plaques.

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A case of transposition of the great arteries in a female infant of a HIV-1-infected woman. Potential teratogenic effects of antiretroviral drugs

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Highly active antiretroviral therapy (HAART) has diminished human immunodeficiency virus-1 (HIV-1)-related morbidity and mortality[1]. As the rate of sexual transmission is increasing[1], the growing proportion of newly HIV-1-infected women is accounting for the increased risk of mother-to-child HIV-1 transmission[1]. Although perinatal zidovudine (ZDV), elective cesarean section and alternatives to breast feeding are reducing vertical transmission rates, foetuses may still be exposed in utero to adverse effects of maternal HIV-1 infection mainly related to the plasma viral load[1-3]. Vertically transmitted HIV-1 infection and uterine environment are associated with postnatal cardiovascular abnormalities, such as reduced left ventricular size[2,4]. Moreover, HAART in pregnancy raises concerns of potential teratogenic and long-term metabolic effects on the child[1,3,5]. In rodents, ventricular septal defects have been associated with the use of delavirdine[1]. We report a case of transposition of the great arteries in a female infant born to a HIV-1-infected woman treated with HAART.

A 34-year-old naïve-treatment HIV-1-infected woman became pregnant in October 2002. She was treated with ZDV, lamivudine (3TC) and nevirapine after the 14th week of gestation. At the beginning of pregnancy, maternal $CD4^+$ T-cell count and plasma HIV-RNA load were 543 cells/μl and 9100 copies/ml, respectively. In July 2003, after the 38th week of gestation, she underwent an elective cesarean section and gave birth to a female infant who had cyanosis and tachydyspnoea. Echocardiography and cardiac angiography suggested the diagnosis of transposition of the great arteries (Fig. 1), which was surgically treated. At present, the child is in good health and the search for anti-HIV antibodies is negative.

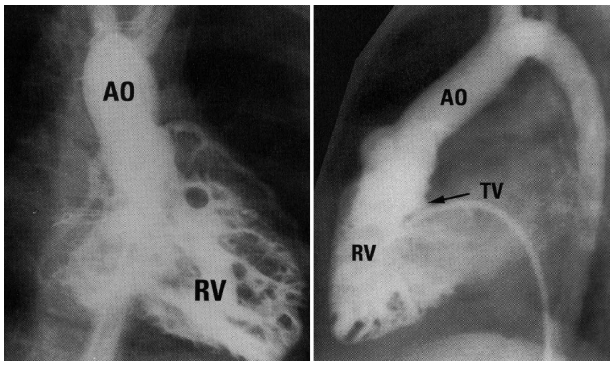

Figure 1. Digital angiography of transposition of the great arteries in the reported case. *Left panel:* anterior-posterior view; *right panel:* lateral projection. AO, aorta; RV, right ventricle; TV, tricuspid valve.

The incidence of transposition of the great arteries in the general population is about 5%[6]. The present knowledge concerning the pathogenesis of transposition of the great arteries in animal models and in humans includes genetic, teratogenic and dysmetabolic factors[7-10]. In the present paper we report the case of transposition of the great arteries in a child born to a HIV-1-infected woman. A limited number of analogous cases have been previously reported[11]. One of the consequences of the development of improved therapies for the treatment of HIV infection has been the emergence of diseases not directly associated with opportunistic infections. These have included myocarditis, dilated cardiomyopathy and neonatal cardiovascular abnormalities[2,4] such as cyanotic lesions, secundum atrial septal defect, ventricular septal defect, and patent ductus arteriosus. A number of aetiologic agents have been proposed to be responsible for the initiation of cardiovascular diseases, such as HIV-1 itself, fungi, parasites or other viruses[12,13]. Finally, it has been suggested that the uterine environment could play an important role in congenital cardiovascular abnormalities[13]. In the case here reported, the uterine environment should have played a minor role in the pathogenesis of the cardiovascular malformation. In fact, although the mother was HIV-positive, the child is HIV-negative and no other infection has been detected both in the mother and in the newborn. In contrast, we wonder whether the HAART regimens might have played a role in the development of the congenital cardiovascular abnormality.

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Combination regimens including three or more antiretrovirals, often referred to as HAART, have a dramatic impact on morbidity and mortality of HIV-infected subjects. Even if the potential risk of antiretroviral drugs used in pregnancy is difficult to assess, it is generally accepted that the use of HAART is associated with a low rate of teratogenic effects and pregnant women are currently treated with HAART after the 14th week of gestation. Of note, the administration of oral ZDV to the mother starting at 14-34 weeks of gestation, followed by intravenous infusion during labour, and to the newborn for 6 weeks after birth, reduced mother-to-child transmission by 67%[5,14-16]. However, septal defect has been reported as a cardiovascular abnormality occurring in children born to HIV-positive mothers[2,4] and an increased frequency of premature birth, low birth weight, low Apgar scores or stillbirth, but not of congenital anomalies, has been associated with HAART treatment during pregnancy[5,17-19]. Therefore, the administration of antiretroviral drugs during pregnancy should be recognised as a potential risk for the foetus. The Food and Drug Administration (FDA) has classified antiretroviral drugs in four categories on the basis of their possible teratogenic risk as follows: (A) adequate and well-controlled studies involving pregnant women failed to demonstrate a risk to the foetus during the first trimester of pregnancy; (B) reproduction studies in animals but not conducted in pregnant women failed to demonstrate a risk to the foetus; (C) safety in human pregnancy has not been determined, studies in animals outweighs the potential risk to the foetus; (D) there is positive evidence of human foetal risk in the form of data regarding adverse reactions from investigational or marketing experiences, but the potential benefits from the use of the drug in pregnant women may be acceptable despite its potential risk.

It has been reported that the use of ZDV or ZDV/3TC in pregnancy could induce anaemia and lactic acidosis which favours muscle and neurologic abnormalities, while the use of protease inhibitors has been associated with low birth weight, prematurity, glucose intolerance, and diabetes[1]. We wonder whether the congenital cardiovascular abnormalities detected in the case here reported could be related to the mitochondrial toxicity of ZDV and/or 3TC taken during pregnancy. Of note, ZDV, 3TC and nevirapine are classified in FDA category "C" which includes drugs with proven foetal toxicity in animals[1]. However, this hypothesis has been ruled out on the basis of available literature data[20]. Finally, we also wonder what the teratogenic role of circulating HIV-1 virus could be although it is known that HAART regimens reduce the rate of mother-to-child HIV transmission to less than 2%. In conclusion, as at present it is not possible to definitely ascertain the real risk of HIV-1 infection and/or HAART treatment on foetal development, a careful management of pregnant HIV-1-infected women is mandatory in order to obtain an early diagnosis of congenital cardiac malformations.

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Food poisoning by *Datura stramonium:* an unusual case report

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Datura stramonium is a common weed along roadsides, in cornfields and pastures, and in waste areas. It comes from the family of *Solanaceae*, the potato family. The plant is native from Asia, but can also be found in the West Indies, Canada, the United States and Europe. Although exposure is sometimes unintentional by gardeners or farmers, its toxic effects are seen most commonly in adolescents, who intentionally misuse it for its hallucinogenic and euphoric effects[1,2]. *D. stramonium* contains a variety of tropine alkaloids and produces atropine-like effects. The seeds of *D. stramonium* contain hyoscyamine, scopolamine, and atropine, all substances with anticholinergic properties that competitively antagonise acetylcholine muscarinic receptors[3]; this predominantly occurs at peripheral (e.g., heart, salivary glands, sweat glands, gastrointestinal tract) postganglionic parasympathetic muscarinic receptors. Anticholinergic substances minimally compete with acetylcholine at other sites (e.g., autonomic ganglia). Central nervous system symptoms result from central cortical and subcortical muscarinic receptor antagonism[4]; grading of symptoms is related to the drug's ability to cross the blood-brain barrier. We here describe a case of a female admitted to hospital showing symptoms mimicking a cerebral ischaemic attack and later attributed to *D. stramonium* poisoning.

A 53-year-old female patient was admitted to our emergency department for acute loss of consciousness. A witness reported that the patient had fallen on her back after lunch. She had been unconscious and had awoken with agitation, confusion, hallucinations and combative behaviour. Her relatives had called the general practitioner who suggested hospital admission due to suspicion of a cerebral ischaemic attack. The first neurological examination, which was performed 1.5 h after sudden symptom onset, revealed mydriasis, hyperreflexia, disorientation and aphasia. The patient scored 9 on the Glasgow coma scale (eye opening = 2; verbal response = 3; motor response = 4). Family history revealed only idiopathic hypertension and cerebral concussion occurred in the last 4 months with fracture of the left eye-socket. This accident caused chronic postural dizziness.

On differential diagnosis, we found cerebral ischaemic attack, haemorrhagic stroke, hepatic encephalopathy, hypo- or hyperglycaemia, active use of drug or central nervous system substances.

Vital signs, such as cardiopulmonary function, body temperature, and blood oxygen saturation were normal (only heart rate was 110 bpm). Laboratory tests did not reveal electrolyte disorders, kidney or hepatic failure, hypo- or hyperglycaemia. Patient characteristics (a 53-year-old housewife), a female with idiopathic hypertension and a history of a recent cerebral concussion suggested a diagnosis which seemed to exclude a case of chronic drug abuse; therefore, at first we performed determination of blood alcohol, barbiturate and benzodiazepine concentration only. Blood alcohol concentration was 0.03%, barbiturate and benzodiazepines were absent.

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Computed tomography scan of the brain was performed to detect brainstem infarction or supratentorial mass lesions. There were no pathological findings.

The second neurological examination was performed 3 h later. Vital signs were stable. Some hours later, she was sleeping in her bed. Over the next hours, the patient's neurological signs gradually subsided and the following morning she was sitting in her bed collaborating, properly oriented (Glasgow coma score = 15), with complete amnesia about the events of the previous afternoon. In history, when asked what she had done the previous day, the patient referred she had decided to prepare some pumpkin blossom fritters using some leaves of an exuberant shrub grown in her garden. We asked her husband to bring us some leaves in the afternoon. That shrub was *D. stramonium*. Forty-eight hours after, the patient was discharged in good clinical conditions, without neurological deficits except for amnesia regarding the acute toxic episode.

Coma and neurological disorders from exogenous poisons or drugs are a common diagnostic problem, not only because of the variety of clinical symptoms but also because of incomplete medical histories and misguided efforts by families and friends to conceal facts. Even in case of suspecting a particular toxic agent, results of a chemical analysis may arrive too late. Therefore, an accurate and immediate diagnosis mostly depends on clinical findings[5].

The typical anticholinergic syndrome (caused by the inhibition of cholinergic neurotransmission at the muscarinic receptor level) shows the following features: agitation, confusion, hallucinations and combative behaviour, decreased bowel sounds, functional ileus, urinary retention, hypertension, trembling, and myoclonic muscular twitching, mydriasis, dry mouth, and tachycardia[6,7]. Laboratory evaluation is usually considered unnecessary since treatment is based on clinical evaluation. Toxicological screening is not considered helpful for management, although some anticholinergics such as atropine and hyoscyamine may be detected in urine. Urine comprehensive drug screening by gas-chromatography-mass spectrometry can reveal scopolamine and atropine[8]. Since atropine and hyoscyamine are optical isomers, they cannot be distinguished from one another by their mass spectra.

Elevated aspartate amino transferase, lactate dehydrogenase, bilirubin, and prothrombin time have been reported, possibly secondary to muscle breakdown from seizures, hypertonia and hyperthermia. Electroencephalographic changes include prominent lambda activity, increased slow wave activity and a bizarre high-voltage pattern[9].

The antidote for anticholinergic toxicity is physostigmine salicylate[10]. Physostigmine is the only reversible acetylcholinesterase inhibitor capable of directly antagonising the signs and symptoms of anticholinergic toxicity in the central nervous system; it is an uncharged tertiary amine that efficiently crosses the blood brain barrier. Physostigmine can reverse the central effects of coma, seizures, severe dyskinesias, hallucinations, agitation, and respiratory distress[11]. The most common adverse effects from physostigmine are peripheral cholinergic manifestations (e.g., vomiting, diarrhoea, abdominal cramps, diaphoresis). Physostigmine may also produce seizures, a complication frequently reported when administered to individuals with tricyclic antidepressant poisoning. Rarely, physostigmine may produce bradyasystole when administered to patients with severe tricyclic antidepressant poisoning. Sinus tachycardia[12] is common and does not require treatment in stable patients; however, tachycardia often resolves with crystalloid infusions, control of agitation (e.g., benzodiazepines), and fever control (e.g., fluids, antipyretics, active cooling measures). In this case it is often useful administering a trial dose of physostigmine[13] over 2-5 min in patients with narrow QRS supraventricular tachydysrhythmias resulting in haemodynamic deterioration or ischaemic pain. Ventricular arrhythmias can be treated with lidocaine. Following initial stabilisation, gastrointestinal decontamination[14] is usually necessary after anticholinergic poisoning by ingestion: for the vast majority of patients, single-dose activated charcoal (1 g/kg with or without a cathartic) orally or by nasogastric tube is sufficient. Activated charcoal is effective over some 24 h after ingestion, owing to diminished gastric motility, and gastric lavage may be required.

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