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ABSTRACTS

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choose the best therapeutic strategy. HFP is a complex of symptoms caused by biogenic amines, mainly histamine, contained in seafood. The diagnosis is quite difficult as the symptoms of this particular condition are similar the symptoms of a normal allergic reaction. *Methods:* We collected 10 cases (3 male and 7 female) of HFP and 50 non-HFP patients (35 female and 15 male). On examination seven of them had a diffuse erythematous rash over the face, arms, legs and torso, flushing, diarrhoea, vomiting, tachycardia, itching and hypotension. Four patients presented with dizziness, nausea and abdominal pain. Laboratory examinations were carried out, including kidney and liver function, clotting system study and tryptase serum levels in samples obtained before any therapeutic intervention, by fluoroenzymeimmunoassay method (Phadia ImmunoCAP™). *Results:* As expected, tryptase serum levels of most of the 50 patients with allergic or anaphylactic disorders were highly increased above normal value, whilst all the tryptase serum levels of the 10 patients with HFP were within the normal range. *Conclusion:* An early diagnosis and treatment is the correct pathway to an effective therapy: a level of serum tryptase within the normal range in patients with clinical signs of histamine mediated reaction and anamnesis compatible with HFP is a useful indicator to support the diagnosis.

303. Tiger Snake (*Notechis scutatus*) Bite with Late Developing Myolysis and Thrombotic Stroke

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Objective: To report a very unusual case of tiger snake (*Elapidae*; *Notechis scutatus*) bite with aberrant venom detection results, delayed myotoxicity and thrombotic stroke. *Case report:* A 63 year old female was bitten above the ankle by a witnessed snake, which was killed and later identified as an adult tiger snake. A PIB (pressure immobilisation bandage) was applied as first aid and she presented at hospital 1 hour post-bite. Bite site venom detection was positive for death adder. She complained of severe local pain, with local bruising and swelling, but no systemic signs of envenoming and normal coagulation, CK, so no antivenom given. She had a recent history of cough and basal crackles on examination. Only LFTs were initially abnormal, and these improved over several days. Coagulation remained normal, except elevated d-dimer on day 2. CK rose from day 3, peaking on day 10 (17852 U/l). Chlamydia serology positive (day 2 & 10) but antibody titre negative. CXR showed bibasal collapse. On day 5 the patient developed headache, vomiting and on day 10 worsening, when CT head showed a R parietal lobe lesion consistent with venous thrombosis. The cause was not apparent, and late envenoming was considered unlikely, so antivenom was not given. She improved and underwent rehabilitation and at 3 months has no residual symptoms or signs. There are no previously reported cases of thrombotic stroke following Australian snakebite, but it is unclear if envenoming was causative in this case, given the late onset. *Conclusion:* Despite evidence of only mild systemic envenoming, consistent with a tiger snake, but with erroneous venom detection, this patient developed a severe adverse event, linkage to envenoming being uncertain. However, thrombotic strokes are now being described in isolated cases for other snake species, indicating this may be a rare complication for snakebite, not just restricted to the two known thrombotic species from the Caribbean, *Bothrops lanceolatus* and *B. caribbaeus.*

304. Cholinergic Symptoms Due to Brown Fly Agaric Poisoning

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Objective: The Finnish Poisons Information Service (FPIC) annually receives about a dozen inquiries concerning brown fly agaric mushroom, *Amanita regalis*. In most cases it has been confused with the edible parasol mushroom, *Macrolepiota procera*. Brown fly agaric is often considered just as a brown variation of fly agaric, *Amanita muscaria*. Also according to current sources of information it contains muscimol and ibotenic acid. However, in a few poisonings reported in Finland and in Germany, the symptoms were not characteristic of the mushrooms containing ibotenic acid and muscimol. In those cases symptoms began usually 1–2 hours after ingestion and the patients had gastrointestinal symptoms, unconsciousness, convulsions and cholinergic symptoms.[1,2] We now report a new case of poisoning due to brown fly agaric demonstrating cholinergic symptoms. *Case report:* In August 2009 a 62-year-old man ate several brown fly agarics believing he had eaten parasol mushrooms. Soon after ingestion he developed gastrointestinal symptoms and confusion. Four hours after ingestion and admission to the hospital the patient was sweating profusely, had muscle jerks and agitation. Activated charcoal, atropine and diazepam were given. About 6 hours later the patient was unconscious, but responded to painful stimuli with trembling and twitching. Nine hours after ingestion, the patient had apneic periods and the muscle twitching continued. Flumazenil was given, because he had received slightly excessive doses of diazepam. Flumazenil relieved both muscle tension and breathing. Symptoms gradually resolved and the patient was discharged 22 hours after the ingestion of the mushrooms. *Conclusion:* Our case provides further support that brown fly agaric mushroom ingestion causes cholinergic symptoms different from the typical ones seen after ingestion of mushrooms containing muscimol and ibotenic acid. *References:* 1. Elonen E, Tarssanen L, Härkönen M. Poisoning with Brown Fly Agaric, Amanita Regalis. Acta Med Scand 1979; 205:121–3. 2. Hentschel H, Volkmann H, Grummt B, et al. Poisoning with Amanita regalis (Brown Fly Agaric). Clin Toxicol 2004; 42:512.

305. Multiple Splenic Infarctions After Viper Envenomation

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Objective: To describe a case of viper envenomation where a progressive increase of D-dimer levels was correlated to multiple splenic infarctions. *Case report:* A 66-year-old man was admitted to the ED ten hours after a viper-bite on his right hand: a painful oedema was present up to the elbow and the patient suffered several episodes of diarrhoea. The next day the oedema appeared unchanged while laboratory tests revealed an increase of D-dimer (3,916 ng/mL), WBC (20,700/mm^3), AST (106 IU/L) and CPK (1247 IU/L), so treatment with low weight heparin (LWH) was started; the patient remained clinically stable and an echo-colour-Doppler was negative for signs of thrombosis. The following day D-dimer was 20,000 ng/mL and started decreasing during the following 24 hours with levels that ranged between 10,000 and 14,000 ng/mL until discharge. From day 4 the oedema progressively improved; in the meanwhile LWH was suspended because the patient developed a mild haemolytic anaemia (haemoglobin 11.8g/dL). On day 8 WBC and platelets decreased to 14,000 and 76,000/mm^3 respectively; D-dimer was 13,000 ng/mL. On day 9, an abdominal echography showed a spleen enlargement (up to 16 cm) with non-homogeneous areas. A contrast CT-scan was immediately performed, that revealed areas of hypodensity due to multiple infarcts occupying most of the parenchyma; no signs of thrombosis were detected. Considering the stable levels of haemoglobin and platelets, LWH was re-started at low dosage. The areas of infarction slightly reduced during the following 5 days, so the patient did not undergo splenectomy and was discharged on day 21 with a program of follow-up abdominal echographies. At 6 months from the viper-bite parameters had normalized and non-homogeneous areas were still present in 50% of the spleen parenchyma; D-dimer was 14,000 ng/mL. *Conclusions:* Considering the limited severity and the stability of clinical conditions during the first 24 hours, Fab-fragments were not administered. The progressive increase of D-dimer suggested looking for possible thrombosis despite improvement of general condition. In this case D-dimer has been an important marker for identification of severe systemic effects of viper venom and permitted identification of rare complications such as splenic infarction.

306. Familial Tetrodotoxin Poisoning in French Guiana

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Introduction: Tetrodotoxin poisoning is a rare event outside Japan where this marine poisoning is well known. The authors describe a case of collective poisoning in French Guiana where porcupinefish and pufferfish are usually not considered to be edible. *Case series:* Two adults and a 2-year-old child had a meal composed of 3 unidentified fresh fish given by a fisherman. They removed the skin and internal organs, washed, grilled and then ate the flesh. The first adult (case 1) ate one fish, the other adult ate half a fish, and the child ate only a spoonful. Case 1: A 54-year-old man was admitted to an emergency unit at H10 with slurred speech and paresis of the lower limbs. Cardio-respiratory arrest occurred suddenly several minutes later, causing anoxic complications. This patient died on D47 (D2 serum tetrodotoxin concentration: 18.5 ng/mL). Case 2: A 34-year-old man presented at H16 with oral dysaesthesia and dizziness which had appeared several minutes after the meal. Muscle weakness, ataxia and slurred speech were also reported. At H35, the clinical features included dilated pupils, hypersalivation, diminished reflexes and sinus bradycardia (55 bpm) and respiratory depression requiring mechanical ventilation. All symptoms progressively resolved after H60 and extubation was possible on D4. Case 3: The child was admitted at H16 with ataxia. Hypersalivation, diarrhoea, muscle weakness, paraesthesia and a vestibular syndrome were observed. Progressive recovery was reported from D3 and was complete on D4. *Discussion:* Tetrodotoxin poisoning was proposed on the basis of the clinical history and clinical features of the 3 patients and was confirmed by blood assay in case 1. Several fish species in French Guiana can cause tetrodotoxin poisoning; tetrodotoxin is mainly found in the liver, ovaries, intestine, and skin of the fish. In the cases reported here, all these parts had been removed before cooking. However, tetrodotoxin is also present in lower concentrations in the flesh and the large amounts ingested by the 2 adults probably account for their severe poisoning. The interval between the meal and onset of respiratory paresis/paralysis was unusually prolonged. Such a delayed onset has been previously reported in only 2 cases.

307. A Bittersweet Symphony

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Objective: To describe an unusual cyanide poisoning due to ingestion of large quantities of seeds containing amygdalin which caused typical symptoms linked to inhibition of cellular respiration. *Case report:* A young woman of 35 years, mentally disturbed, was admitted to our ED. The parents said they had found her in the living room, surrounded by apricots from which she had extracted the core, eating the kernels. According to anamnesis, it seems that the woman had swallowed 40 to 60 kernels 30 minutes before arrival at ED.

MULTIPLE SPLENIC INFARCTIONS AFTER VIPER ENVENOMATION

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Objective

D-dimer is a useful marker for monitoring of systemic symptoms secondary to Italian viper bites. The inoculated toxins seem to induce primary hyperfibrinolysis and consequent increase of D-dimer serum concentrations.
We describe a case of viper envenomation where a progressive increase of D-dimer levels was correlated to multiple splenic infarctions.

Case report

A 66-year-old man was admitted to the ED ten hours after a viper-bite on his right hand: a painful oedema was present up to the elbow and the patient referred several episodes of diarrhoea. The next day the oedema appeared unchanged while laboratory tests revealed an increase of D-dimer (3,916 ng/ml; normal dosage < 500 ng/ml), WBC (20,700/mm3), AST (106 IU/L) and CPK (1247 IU/L), so treatment with low weight heparin (LWH) was started; the patient remained clinically stable and an echo-colour-Doppler was negative for signs of thrombosis.
The following day D-dimer was 20,000 ng/ml and started decreasing during the following 24 hours with levels that ranged between 10,000 and 14,000 ng/ml until discharge.
From day 4 the oedema progressively improved; in the meanwhile LWH was suspended because the patient developed a mild haemolytic anaemia (haemoglobin 11.8g/dL). On day 8 WBC and platelets decreased to 14,000 and 76,000/mm3 respectively; D-dimer was 13,000 ng/ml. On day 9, an abdominal echography showed a spleen enlargement (up to 16 cm) with non-homogeneous areas. A contrast CT-scan was immediately performed, that revealed areas of hypodensity due to multiple infarcts occupying most of the parenchyma; no signs of thrombosis of the major splenic vessels were detected (Figure 1).
Considering the stable levels of haemoglobin and platelets, LWH was re-started at low dosage. The areas of infarction slightly reduced during the following 5 days, so the patient didn't undergo splenectomy and was discharged on day 21 with a program of follow-up of abdominal echographies.
Six months after the viper-bite diameters had normalized and non-homogeneous areas were still present in 50% of the spleen parenchyma; D-dimer was 14,000 ng/ml.

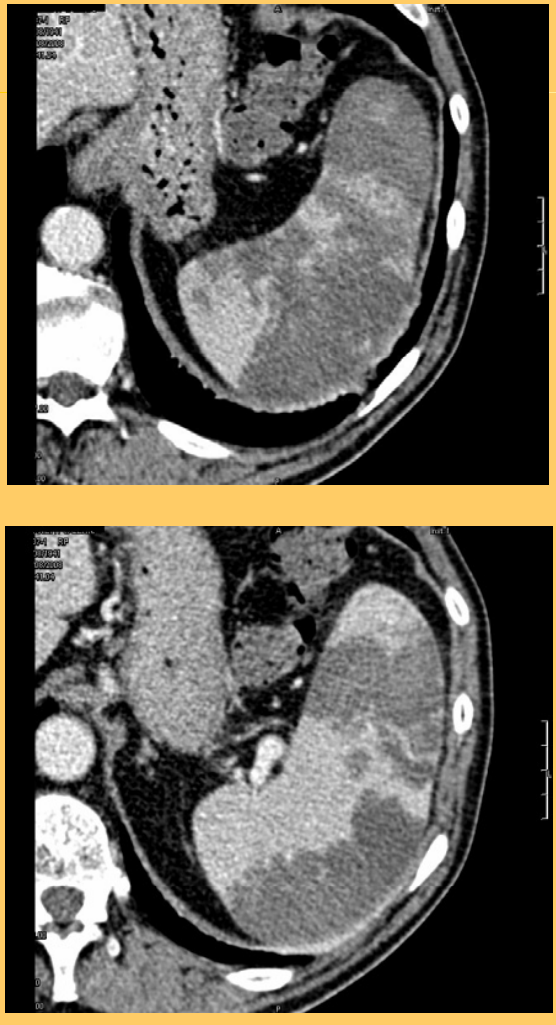

Figure 1: (CT-scan) spleen multiple infarctions

Conclusions

Considering the early resolution of initial systemic symtoms and the stability of clinical conditions during the first 24 hours, Fab-fragments were not administered even though the clinical picture could have been classified as a bite of moderate severity according to the Grading Severity Score (GSS 2) (1,2). Nevertheless the progressive increase of D-dimer suggested to look for possible thrombosis despite improvement of general conditions. Rare case reports of thrombosis and infarction away from the site of envenomation are reported in the literature (3,4,5). In this case D-dimer has been an important marker for identification of a severe systemic effect of viper venom and permitted to identify an unusual complication such as multiple splenic infarctions.

References
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