with concomitant fracture; although some authors recommend one attempt at closed reduction, it is likely that such patients will require open reduction with fixation of the fracture, so early consultation with an orthopaedic surgeon is advisable [7, 8]. If closed reduction appears successful, the arm should be immobilised and post-reduction radiographs should be obtained to verify placement and identify any new fractures.

Patients with posterior shoulder dislocations should be seen by an orthopaedic surgeon, either in the ED or within 5–7 days after discharge, and the patient should remain in a shoulder immobiliser until this evaluation. Some patients may require early surgical intervention, while others may be treated with immobilisation [9]. Rotator cuff exercises or physical therapy can be useful in preventing recurrence of dislocation, especially in those with seizure disorders who are at risk of future dislocations during seizures [10].

We report a case of bilateral posterior shoulder dislocations that were identified and successfully reduced in the ED. Posterior shoulder dislocations occur rarely but are often missed on initial presentation, resulting in ongoing patient discomfort, long-term morbidity and elevated health care costs. Posterior shoulder dislocations should be considered in post-ictal patients with shoulder pain or an abnormally appearing shoulder. ED physicians may attempt to reduce the dislocation if there is no concomitant fracture, but early consultation with orthopaedic surgery is often advisable.

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Amino acid sequence homologies between HCV polyprotein and thyroid antigens

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Recent evidence in the literature suggests that molecular mimicry between viral and self antigens may be involved in the pathogenesis of autoimmune thyroid diseases in patients with chronic hepatitis C virus (HCV) infections [1–3].
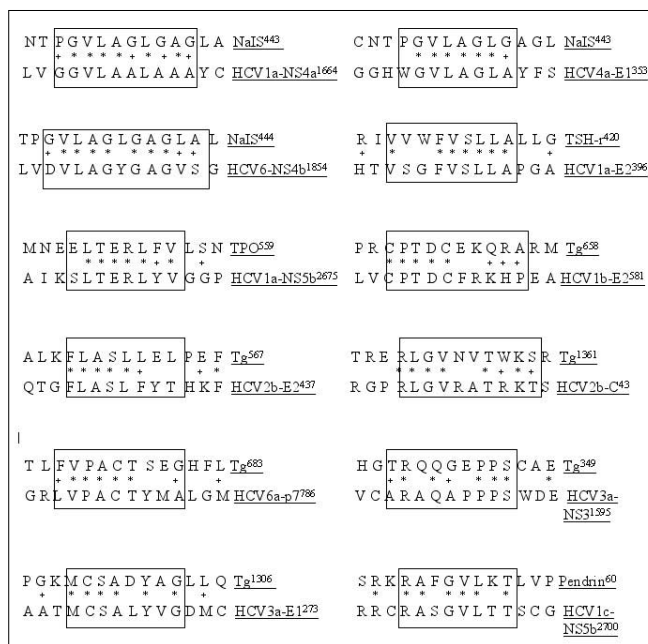Chronic HCV infection has been reported to be associated with thyroid autoimmunity and thyroid function disorders with a mean incidence of 10% and 3%, respectively [4, 5]. Alfa-IFN therapy may exacerbate or induce underlying latent thyroid disorders, increasing the incidence of thyroid autoimmunity and thyroid function disorders to 20% and 11%, respectively [4, 5].

In keeping with the tenets of the clonal selection theory of acquired immunity, an infectious agent may circumvent the deletion of anti-self lymphocytes activating clones with receptors sufficiently degenerated to respond to mimicking epitopes and host antigens [6].

A minimum of five to six amino acids are necessary to induce an immune response, and the probability of 20 amino acids occurring in six identical residues between two proteins is 20^6 (for each peptide, irrespective of the sequence) or 1 in 128 000 000 [7].

We performed the comparison between the amino acid sequence of the HCV polyprotein and five tissue-specific antigens of human thyroid, available in the database on www.ncbi.nlm.nih.gov/pubmed.

In particular, we examined the following HCV genotypes (with the respective NCBI sequence identification number): HCV1a (GI:130455), HCV1b (GI:130469), HCV1c (GI:385131), HCV2a (GI:130466), HCV2b (GI:130468),

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NT  P G V L A G L G A G  LA   NaIS⁴⁴³          CNT  P G V L A G L G  AGL   NaIS⁴⁴³
    * * * * * + * + *                               * * * * * * +
LV  G G V L A A L A A A  YC   HCV1a-NS4a¹⁶⁶⁴    GGH  W G V L A G L A  YFS   HCV4a-E1³⁵³


TP  G V L A G L G A G L A  L  NaIS⁴⁴⁴          R I  V V W F V S L L A  LLG  TSH-r⁴²⁰
    * * * * * * * * * +                         +   * * * * * * +
LV  D V L A G Y G A G V S  G  HCV6-NS4b¹⁸⁵⁴     HT  V S G F V S L L A  PGA  HCV1a-E2³⁹⁶


MNE E L T E R L F V  LSN  TPO⁵⁵⁹               PR  C P T D C E K Q R A  RM  Tg⁶⁵⁸
    * * * * * * + *                                * * * * *     + + +
AIK S L T E R L Y V  GGP  HCV1a-NS5b²⁶⁷⁵       LV  C P T D C F R K H P  EA  HCV1b-E2⁵⁸¹


ALK F L A S L L E L  PEF  Tg⁵⁶⁷               TRE  R L G V N V T W K S  R   Tg¹³⁶¹
    * * * * * +  * +                                * * * *     + +
QTG F L A S L F Y T  HKF  HCV2b-E2⁴³⁷         RGP  R L G V R A T R K T  S   HCV2b-C⁴³


TL  F V P A C T S E G H  FL  Tg⁶⁸³            HG  T R Q Q G E P P S  CAE  Tg³⁴⁹
    + * * * * *   +   +                            * * *   * * * *
GRL V P A C T Y M A L  GM  HCV6a-p7⁷⁸⁶         VC  A R A Q A P P P S  WDE  HCV3a-
                                                                           NS3¹⁵⁹⁵


PGK M C S A D Y A G  LLQ  Tg¹³⁰⁶              SRK  R A F G V L K T  LVP  Pendrin⁶⁰
    + * * * * *   *   +                            *   * * * * * +
AAT M C S A L Y V G  DMC  HCV3a-E1¹²⁷³         RRC  R A S G V L T T  SCG  HCV1c-
                                                                          NS5b²⁷⁰⁰
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Fig. 1 Amino acid sequence homologies between the thyroid antigens and the HCV polyprotein (see text for abbreviations). *, Identical residues; +, conservative substitution. In each sequence, the first left-side residue inside the box corresponds to the number of the amino acid sequence. Amino acids in standard single letter code

HCV2c (GI:555104), HCV3a (GI:514395), HCV3b (GI:676877), HCV4a (GI:402474), HCV5a (GI:2462303) and HCV6a (GI:2326455).

Regarding the thyroid gland, we examined the following tissue-specific antigens: the thyroglobulin (Tg) (GI:12644093), the thyroid peroxidase (TPO) (GI:129830), the thyrotropin receptor (TSHr) (GI:136448), the sodium/iodide symporter (NaIS) (GI:12643359) and Pendrin (GI:6174895).

Sequence alignments were carried out using the BLASTp, short nearly exact matches and BLASTp2 protein–protein comparison program (available at www.ncbi.nlm.nih.gov/BLAST).

Amino acid sequence homologies between the HCV polyprotein and the thyroid antigens are given in detail in Fig. 1, showing the presence of identical/conservative residues in the peptides. The following proteins of the HCV polyprotein have been examined: C (capsule, core protein), E1 (envelope glycoprotein 1), E2 (envelope glycoprotein 2, NS1), p7, NS2 (non-structural protein 2), NS3 (non-structural protein 3, protease/helicase), NS4a (non-structural protein 4a), NS4b (non-structural protein 4b), NS5a (non-structural protein 5a) and NS5b (non-structural protein 5b, RNA polymerase).

The homologies between the thyroid and the viral peptides ranged from 62.5 % (five identical residues out of eight amino acids in the sequence) to 87.5% (seven identical residues out of eight amino acids in the sequence). The frequency of the homology increased up to 100%, when the conservative substitutions were included in the analysis (ten out of ten identical/conservative amino acids in the sequence, as indicated in Fig. 1 for NaIS⁴⁴⁴ and HCV1a-NS4a¹⁶⁶⁵).

We found the presence of short peptides (eight to eleven amino acids) with a high degree of homology (62.5–100%) between the HCV polyprotein and five thyroid antigens (Tg, TPO, TSHr, NaIS and Pendrin).

The homology was not restricted to a single HCV genotype or to a single thyroid antigen.

The highest degree of homology was between the NaIS and the HCV1a-NS4a protein. The Tg antigen had the highest number of homologies with the different HCV genotypes.

Previous studies examining 20 amino acid-length peptides showed 41.7–58.3% sequence homologies between TPO and HCV-NS5a and HCV-NS2, increased to 75.0% when including conservative/identical residues [3].

We found mimicry between the TSH-r and the N-terminal hypervariable region 1 (HVR1) of E2 in HCV1a, which is well known to be involved in chronic HCV infection [8].

The length of the short peptides is consistent with the presentation of the self/viral antigens with the I class HLA molecules to CD8 positive lymphocytes, as the II class HLA molecules usually bind longer peptides, and the mimic peptides may be involved in the acceleration of autoimmune disorders occurring in chronic HCV infection [9]. In our examination, the RLGVRATRK-HCV2b-C⁴³ sequence presented homology with the RLGVNVTWK-Tg¹³⁶¹ sequence; the same viral peptide has been recently identified as a HLA-A3 supertype-restricted cytotoxic T-lymphocyte epitope in patients with HCV infections [10].

The more frequent and earlier appearance of anti-Tg antibodies in the clinical course of the thyroid autoimmunity in HCV IFN-treated patients may be related to the high number of homologies between the Tg antigen and the HCV polyprotein, whereas the anti-TPO antibodies reflect a more advanced and aggressive autoimmune thyroid destruction [11].

Further studies are necessary in order to evaluate the clinical relevance of the presence of the molecular mimicry between the HCV and the thyroid antigens in the progression of autoimmune disease.

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Transient massive hyperlipidaemia in a type 2 diabetic subject

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A 50-year-old man, in apparently good health, was referred to the Lipid Center of San Luigi Gonzaga Hospital, Orbassano (Turin, Italy), by his primary care physician (PCP) because clinical tests at the time of blood donation showed milky plasma with serious hypertriglyceridaemia (>5000 mg/dl) and hyperglycaemia (381 mg/dl), diagnostic for diabetes. It was not possible to perform further blood chemical analyses because hypertriglyceridaemia would have provided abnormal results. The patient entered the hospital to prevent acute pancreatitis, which is often associated with severe hypertriglyceridaemia.

The clinical history revealed *H. pylori*-associated gastric ulcer in the prior year. At that time, laboratory tests disclosed "mild" hyperglycaemia and hypertriglyceridaemia (the patient's records were misplaced.) An ill-defined hypolipidaemic diet was recommended at discharge, but the suggestion was neglected. He was a former smoker (30 pack year, stopping about 10 years before), and consumed a diet rich in saturated fats and carbohydrates while alcohol ingestion was mild and occasional (2–3 drinks per week). His living mother was affected by type 2 diabetes; his father died by accident at a young age, while a brother suffered a myocardial infarction at age 43, seemingly not related to traditional cardiovascular risk factors. The patient weighed 79 kg and was 1.75 m tall (Body Mass Index, BMI=25.8 kg/m^2), and there had been no significant recent weight modification. No significant abnormalities were detected at physical examination.

Biochemical analyses (Table 1) corroborated the findings of severe hypertriglyceridaemia (6.594 mg/dl), showing also high cholesterol levels (658 mg/dl) and low plasma high-density lipoprotein cholesterol (HDL-C, 15 mg/dl); apolipoprotein A-I was 75 mg/dl and apolipoprotein B was 141 mg/dl, both within normal laboratory range. After refrigeration overnight at +4°C, serum showed a creamy surface layer and a turbid infranatant, while lipoprotein electrophoresis disclosed lipids at origin and a broad pre-β band: both these tests indicated the presence of chylomicrons and very low-density lipoproteins (VLDLs). Plasma creatinine was within normal limits, while urinalysis detected trace amounts of glucose and ketone bodies; liver and thyroid functions were also normal; γ–glutamyl-transferase was slightly increased and serum sodium decreased. Glucose level (215 mg/dl) and glycated haemoglobin (HbA1c) were increased, C-peptide was in the normal-high range, while immune parameters were unaffected, in particular anti-islet cells antibodies (ICA) and glutamic acid decarboxylase antibodies (GADA) were not detected.

Preparative ultracentrifugation was carried out, showing cholesterol enrichment in the density fraction <1.006 g/ml, corresponding to chylomicrons+VLDLs. Apolipoprotein E was homozygous for the most common isoform (ε3/ε3 genotype). We did not disclose a deficiency of apolipoprotein C-II, which represents the physiological lipoprotein lipase (LPL) activator, but we could not directly evaluate LPL activity.

Supra-aortic and lower limb echo-doppler examination revealed increased intima-media thickness but no plaques, while liver echotomography showed diffuse, high-grade steatosis.

Along with clinical evaluation, dietetic therapy and a four-injection insulin regimen was administered (3 short-acting pre-prandial insulin, and one bedtime long-acting dose). Treatment resulted in rapidly improving and rather steady glycaemic control with progressive reduction of insulin requirement. At the same time the dyslipidaemia was treated with low-fat diet, subcutaneous calcium heparin (2000 IU,