ELLP syndrome is used in these [1]. The patient described above developed a HELLP syndrome without hemolysis, complicated by hepatic rupture. Liver capsule rupture, following subcapsular hematoma is a rare complication of pregnancy with related maternal (0–24%) and fetal-neonatal mortality (7.7–60%). Microscopic study of these cases showed areas of acute necrosis throughout the hepatic parenchyma and the presence of fibrin in the vessels of the liver, thus supporting the possible pathogenetic significance of disseminated intravascular coagulation leading to thrombohemorrhagic complications [4].

In the present case report, dynamic enhanced CT scan and MRI imaging techniques were both useful in detecting hypoperfused areas of the liver and in the follow-up of the patient until total recovery. Chiang et al. [5] suggested that the management of such patients could be modified if liver hematomas were diagnosed early, before rupture. The same authors described a patient with toxemia of pregnancy and the HELLP syndrome, in whom a follow-up abdominal CT scan 14 days after liver hemorrhage revealed a reduction in the areas of hepatic necrosis. Unfortunately, the patient died on day 16 and the time course of the hepatic lesions could not be evaluated. In our case report, an abdominal CT scan showed marked hepatic regeneration, and MRI images were consistent with scar formation. It is of interest to note that, 3 months later, both CT scan and MRI evaluation revealed a seemingly normal liver.

References

1. Sibai BM, Taslimi MM, El-Nazer A et al (1986) Maternal-perinatal outcome associated with the syndrome of hemolysis, elevated liver enzymes, and low platelets in severe preeclampsia-eclampsia. Am J Obstet Gynecol 155:501–509
2. Blanco JL, Ginestal RJ, Vicente JR (1991) Síndrome de HELLP. Med Intensiva 15:345–351
3. Sibai BM (1990) The HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets): Much ado about nothing? Am J Obstet Gynecol 162:311–316
4. Nelson EW, Archibald L, Albo D (1977) Spontaneous hepatic rupture in pregnancy. Am J Surg 134:817–820
5. Chiang K, Athey P, Lamki N (1991) Massive hepatic necrosis in the HELLP syndrome: CT correlation. J Comput Assist Tomogr 15:845–847

P. Saura (✉) · Ll. Blanch · E. Capdevila
F. Baigorri · J. Martín · H. Corona
A. Artigas
Intensive Care Department,
Hospital de Sabadell,
Parc Taulí s/n, E-08208 Sabadell, Spain

K. Terasako
N. Seo
T. Murayama
T. Kai
S. Hirata
T. Fujiwara

Are autoimmune mechanisms involved in critical illness polyneuropathy?

Received: 21 December 1993
Accepted: 27 July 1994

Sirs: We recently encountered a condition that was diagnosed as critical illness polyneuropathy (CIP) on the basis of the clinical features [1, 2]. In this case, we detected a high protein content with pleocytes in the CSF and high titers of IgG antibody against GM_1 ganglioside in the patient's sera.

An 81-year-old man developed multiple organ failure following multiple hepatic abscesses caused by *Klebsiella pneumoniae* infection. The patient had complained of muscle tenderness and presented a considerably elevated serum CPK level of 12310 IU/l (CPK-MB 113 IU/l). After drainage of the abscesses, the patient's condition gradually improved: muscle tenderness subsided and serum CPK levels were normal (50 IU/l) on day 17. However, the patient still remained on a ventilator and neck stiffness was observed. Lumbar puncture revealed an increased protein content (467 mg/ml) with pleocytes (14 cells/3 fields, monocyte 10, segmented cell 4) in the CSF. A week later, he developed flaccid, areflexic tetraparesis with mild peripheral sensory disturbances. The cranial nerves were intact, and CT head and cervical scan, normal. Electromyographic studies were almost normal, and nerve conduction studies of the median nerve revealed normal motor and sensory conduction velocity and M-wave amplitude, but prolonged F-wave latency. Muscle and nerve biopsy in the sural nerve region demonstrated no specific changes. There was no serologic evidence of *Campylobacter jejuni* infection. Steroid pulse therapy followed by six plasmapheresis treatments did not alleviate the symptoms. About 3 months after admission, thin-layer chromatography with immunostaining revealed that serum IgG from the patient had reacted with GM_1. Enzyme-linked immunoadsorbent assay showed that anti-GM_1 antibodies (IgG) were obviously elevated in the patient's serum (absorbance at 492 nm was 1.11), compared with healthy volunteers (mean ± standard deviation of absorbance = 0.036 ± 0.009). Electromyographic studies revealed long-duration and high-voltage waves during voluntary contraction of the biceps. The patient was not completely weaned off the ventilator until about 4 months later.

CIP is similar to the Guillain-Barré syndrome (GBS), but appeared to be related to severe infection and nutritional disorders [1–3]. The condition in our patient was diagnosed as CIP on the basis of its clinical features.

It has recently been suggested that the pathogenic mechanisms of GBS are related to cell-mediated immunity, antibody-targeted macrophage-mediated demyelination or complement-dependent demyelination [4]. However, attribution of the autoimmune response remains to be defined in CIP.

The high protein content with pleocytes in the CSF was not characteristic of CIP and high serum concentrations of anti-GM, antibodies have never been reported in CIP. The pathophysiology of these findings was unclear, but might correspond to symptoms of acute axonal polyneuropathy in a subgroup of GBS [5]. This case suggests that autoimmune mechanisms may also be involved in the pathogenesis of CIP.

References

1. Bolton CF, Gilbert JJ, Hahn AF, Sibbald WJ (1984) Polyneuropathy in critically ill patients. J Neurol Neurosurg Psychiatry 47:1223–1231
2. Bolton CF, Laverty DA, Brown JD, Witt NJ, Hahn AF, Sibbald WJ (1986) Critical ill polyneuropathy: electrophysiological studies and differentiation from Guillain-Barré syndrome. J Neurol Neurosurg Psychiatry 49:563–573
3. Lopez JB, Garcia A (1990) Acute polyneuropathy in critically ill patients. Intensive Care Med 16:159–162

4. Ropper AH (1992) The Guillain-Barré syndrome. N Engl J Med 326:1130–1135
5. Yuki N, Yoshino H, Sato S, Miyatake T (1990) Acute polyneuropathy associated with anti-GM_1 antibodies following *Campylobacter* enteritis. Neurology 40:1900–1902

K. Terasako · N. Seo (✉) · T. Murayama
T. Kai · S. Hirata · T. Fujiwara
Department of Anesthesia
and Intensive Care,
Omiya Medical Center,
Jichi Medical School,
1-847 Amanuma-cho, Omiya-City,
Saitama Prefecture 330, Japan

R. Vialet
F. Illouz
M. Pibarot
L. Papazian
F. Gouin

False aneurysm of the brachial artery after an attempt to place an axillary venous access

Received: 17 December 1993
Accepted: 26 April 1994

Sir: The mechanical complications of venous catheterisation are well known [1]. We should like to report an uncommon case: false aneurysm of the brachial artery.

A 58-year-old man was treated in the ICU following right nephrectomy for renal carcinoma with involvement of the vena cava. On day 49, septicaemia was suspected. A decision was taken to remove the central venous catheter in the internal jugular vein which had been in place for 45 days. Since the patient had already undergone tracheostomy, venous access via the left axillary vein was chosen instead. Both coagulation and platelet count were normal. An attempt was made to puncture the axillary vein with a 16-G needle (Hasselcath, Plastimed 6346 F, Saint-Leu La Forêt, France) on the proximal third of the humerus without using a tourniquet. Palpation of the brachial pulse was difficult due to oedema and the first attempt resulted in an arterial puncture. The needle was withdrawn and, despite compression, a haematoma developed rapidly, evolving into a mass 7 cm in diameter. The lump was firm, painless, without pulsation, and could not be compressed. Auscultation revealed a faint murmur. Ultrasonography showed an oval (4 cm length) collection of liquid in the biceps. CT scan revealed that blood from the brachial artery was circulating in that space. The diagnosis of false aneurysm was confirmed by angiography (Fig. 1) which showed that the brachial artery had been pushed slightly backwards. The patient underwent surgery on day 77 under local anaesthesia. Six months later, there had been no recurrence of false aneurysm.

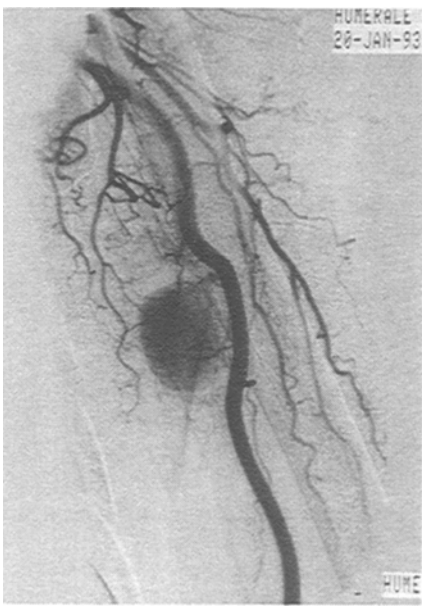

Fig. 1 Angiography: false aneurysm of the brachial artery

Severe mechanical complications of central venous access, such as pneumothorax, nerve injury, haemorrhage, aneurysm or arteriovenous fistulae are fortunately rare. Only 175 mechanical complications have been reported by the Food and Drug Administration over 2 years. However, these complications led to the death of the patients in 52 cases [1]. The incidence of arterial puncture, the most frequent complication, is estimated as about 3% for internal jugular vein access [2]. The incidence of arterial puncture for the transaxillary route varies between 6 and 13% [3]. The incidence of aneurysm or arteriovenous fistulae as complications of central venous catheterisation, is difficult to assess. We could only find 7 cases reported in the Medline Library between 1990 and 1992, involving the subclavian or vertebral artery [4]. These complications resulted from internal jugular vein or subclavian vein approaches. To our knowledge, it is the first time that such a complication has been reported with respect to the axillary vein. The physician must bear these complications in mind when deciding to use a central venous access in a high-risk patient.

References

1. Scott WL (1988) Complications associated with central venous catheters: a survey. Chest 94:1221–1224
2. Schwartz AJ, Jobes DR, Greenhow DE, Stephenson LW, Ellison N (1979) Carotid artery puncture with internal jugular cannulation. Anesthesiology 51:160
3. Martin C, Eon B, Auffray JP, Saux P, Gouin F (1990) Axillary or internal jugular central venous catheterization. Crit Care Med 18:400–402
4. Verrières D, Bernard C, Dacheux J, Reizine D, Echter E (1986) Fistules artério-veineuses cervicales après cathétérisme jugulaire interne. Ann Fr Anesth Reanim 5:162–164

R. Vialet · F. Illouz · M. Pibarot
L. Papazian (✉) · F. Gouin
Département d'Anesthésie-Réanimation,
Hôpital Sainte-Marguerite,
F-13009 Marseille, France