Treatment of tuberous xanthomas using Carbon dioxide laser

Xanthomas are deposits of lipids in the skin or sometimes in the subcutaneous tissue. They are a dyslipidemia-related condition, and appear clinically as yellowish, spots, plaques or nodules, depending on the site of lesions: xanthomas palpebrarum, planar xanthomas, xanthomas disseminatum, tendinous xanthomas. Tuberous xanthomas (TX) are another clinical subtype of lipid metabolism trouble, rarely observed, presenting as yellow-orange nodules, involving all body but mainly located over knees and elbows. Many lasers, including carbon dioxide laser, have been proposed to treat xanthelasma (1-8), but few data are available on treatment modalities of TX especially when lesions are widespread. The aim of this article is to report the case of a young woman with a type IIa primary hypercholesterolaemia presenting for multiple scattered TX which were treated with carbon dioxide laser.

Observation

A 21-year-old woman, born to nonconsanguinous parents, followed at the endocrinology department for familial hypercholesterolemia was referred to our dermatological department with excessive TX of the knees, the elbows and the buttocks. The parents first noticed xanthomas in early childhood. She developed intertriginous xanthomas at the age of five years. At the age of 10 years, she developed multiple TX of the elbows and the knees. Xanthelasma palpebrarum appeared since she was 18-years-old. Clinical examination revealed the presence of multiple flat-topped, firm, welldemarcated, yellowish-orange, nodules varying in diameter from 1 to 5 cm, localized at the elbows, knees, buttocks (Fig. 1) and interdigital space of the fingers. In the periorbital area, bright yellow plaques were present. Mucosa, hair and nails were normal. The patient didn't suffer from chest pain and shortness of breath. Cardiac examination and electrocardiogram didn't reveal any pathological findings. Respiratory and central nervous system examinations were found to be normal. Skin biopsy of a nodule of the knee revealed within the dermis a proliferation of foamy histiocytes arranged in sheets, admixed with lymphocytes and scattered Touton giant cells. The diagnosis of TX was made. The serum lipid profile was abnormal: total cholesterol was increased at 19.32 mmol/l (4,7 mmol/l), and level of triglycerides was normal at 0.8 mmol / l. Thus, the lipid profile was consistent with the diagnosis of hypercholesterolemia type IIa according to Fredrickson. The liver analyses were normal. Parents were also followed for a mild hypercholesterolemia.

According to the personal and familial medical history, the clinical examination and the lipid profile, the diagnosis of homozogous familial hypercholesterolemia type IIa was made. Although the patient was receiving *simvastatin* and *cholestyramin* daily for many years associated to cholesterol-

lowering regimen, she continued to have high levels of cholesterol with no reduction in the size of the multiple TX. Moreover, skin lesions have an important psychological impact on our patient. Because the patient was afraid about laser therapy, she asked to treat buttocks lesions first. CO2 laser (CO2 Laser, Sharplan, 10600 nm), was carried out under local anaesthesia using 2% lidocaine solution, in continuous cutting mode (Power=15 W), to reduce thickness of the exuberant tissue in larger xanthomas. Fine sculpting was achieved by using the CO2 laser in either in resurfacing mode or in continuous mode at 10 W using a defocused 2-3-mm beam. All xanthomas over the buttocks were treated in one session (Fig. 2). Post-treatment, vaseline gauze and topical antibiotic (fucidic acid) were applied daily. Re-epithelialization normally completed within one month (Fig 3) with scarring sequelae. Postoperative courses were simple and the aesthetic result was good 3 months later. No recurrence was noted after 1 year of follow-up.

Figure 1 : Tuberous xanthomas of the buttocks



Figure 2 : Tuberous xanthomas immediately after CO2 laser treatment



Figure 3 : Good results 1 month after CO2 laser treatment



Conclusion

Unlike xanthelasma treatment, few data are available on therapeutic modalities for TX in familial hypercholesterolemia as this illness is extremely rare. In some cases treatment of the underlying medical disorder may cause regression of xanthomas. But, as observed in this case, the patient continued to develop new cutaneous xanthomas although she was receiving *simvastatin* and *cholestyralamin*.

We used CO2 laser in our patient because it allows treatment of widespread TX in one session under local anesthesia. To our knowledge this is the first reported case of TX treated by CO2 laser with a good cosmetic result. CO2 laser appears as safe and effective for the treatment of TX and should be considered a good alternative to surgery for widespread lesions. Further studies including more patients should be performed to confirm such results.

References

- Raulin C, Schoenermark MP, Werner S, Greve B. Xanthelasma palpebrarum: treatment with the ultrapulsed CO2 laser. Lasers Surg Med 1999;24:122-7.
- Alster TS, West TB. Ultrapulse CO2 laser ablation of xanthelasma. J Am Acad Dermatol 1996;34:848-9.
- Lieb WE, Klink T, Münnich S. CO2 and erbium YAG laser in eyelid surgery. A comparison. Ophthalmologe 2000;97:835-41.
- Carpo BG, Grevelink SV, Brady S, Gellis S, Grevelink JM. Treatment of cutaneous lesions of xanthoma disseminatum with a CO2 laser. Dermatol Surg 1999;25:751-4.
- Basar E, Oguz H, Ozdemir H, Ozkan S, Uslu H. Treatment of xanthelasma palpebrarum with argon laser photocoagulation. Argon laser and xanthelasma palpebrarum. Int Ophthalmol 2004;25:9-11.
- Karsai S, Czarnecka A, Raulin C. Treatment of xanthelasma palpebrarum using a pulsed dye laser: a prospective clinical trial in 38 cases. Dermatol Surg 2010;36:610-7.
- Park EJ, Youn SH, Cho EB, et al. Xanthelasma Palpebrarum. Treatment with a 1,450-nm-Diode Laser. Dermatol Surg 2011;37:791-6.
- Fusade T. About the treatment of xanthelasma palpebrarum using a 1,064 Qswitched neodymium-doped yttrium aluminum garnet laser. Dermatol Surg 2011;37:403-4.

Samy Fenniche, Talel Badri, Md, Houda Hammami, Rym Benmously, Inç af Mokhtar,

Dermatology Department. Habib Thameur Hospital, Faculty of Medicine, University of Tunis El Manar, Tunisia Liver cirrhosis localized in the left lobe: an unusual presentation of small duct primary sclerosing cholangitis.

Liver cirrhosis is defined as a diffuse process characterized by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules [1]. To our knowledge, there is no report of cirrhosis localized only in a part of the liver. However, cirrhosis is a heterogeneous condition with differing clinical manifestations and prognosis depending on the etiology and the severity of hepatic architectural distortion [2]. Primary sclerosing cholangitis (PSC) is a chronic cholestatic liver disorder characterized by inflammation, obliteration and fibrosis of the intra-hepatic and/or extra-hepatic biliary ducts [3]. The course of PSC is often progressive, leading to biliary cirrhosis. Small duct PSC is a variant with normal cholangiogram but typical biochemical and histological features of PSC [3.4].

We report herein the case of a woman with small duct PSC at the stage of cirrhosis localized in the left lobe.

Case-report

A 42 year-old woman was referred to our department for a 2 year history of right upper quadrant pain. There was no pruritus, no jaundice, no chills, and no fever or weight loss. She complained of peripheral arthritis and inflammatory low back pain. In her past medical history, she reported 2 spontaneous abortions and one pre-eclampsia in the last delivery 8 years earlier. She was on no medication and did not consume alcohol. A part from a mild back stiffness, physical examination was normal and revealed no signs of chronic liver disease. Her body mass index was 21 kg/m2. Laboratory data disclosed anicteric cholestasis with elevated vglutamyl transpeptidase level of 107 IU/l (normal range (NR) <35 IU/l), alkaline phosphatase of 245 UI/l (NR<110 IU/l) and normal bilirubin level. Serum aspartate aminotransferase was 35 IU/l and alanine aminotransferase 77 IU/l (NR< 45 IU/l). The same cholestatic biochemical picture was present one year ago. The erythrocyte sedimentation rate was 94mm (first hour) and protein C reactive 39 mg/l. Microcvtic anemia and elevated ferritinemia was present. hemoglobin was 10 g/dl, while reticulocyte count was low, consistent with an inflammatory origin. Leucocytes count and platelet count were normal. Total cholesterol was 6.85 mmol/l (NR<6 mmol/l), albumin was 32.3 g/l with vglobulins 26g/l (IgG = 21.2g/l). Prothrombin time was 91%, factor V 100%. Serological tests for hepatitis B and C were negative. Antismooth muscle, antimitochondrial, anti nuclear, anti liver kidney mocrosome1, anti liver cytosol1, anti GP210, anti PML and anti SLA antibodies were not detected. Human leucocytes antigen was A31 B50.

Abdominal ultrasound and computed tomography (CT)