Case Report
doi: 10.4183/aeb.2012.119

A CASE OF ACROMEGALY IN THE PRESENCE OF COINCIDENTAL LIVER CIRRHOSIS

S. Topsakal*, F. Akin, E. Yerlikaya, T. Korkmaz

Pamukkale University, Medical Faculty - Endocrinology and Metabolism, Turkey

Abstract

Context. Acromegaly is a rare and serious syndrome and commonly associated with pituitary neoplasm. Classic cause of acromegaly in adults is the tumors of the somatotrophs that secrete growth hormone. Cirrhosis is the end stage of chronic liver disease and commonly a cause of death. It is characterized by diffuse hepatic fibrosis resulting in altered construction of the lobular parenchyma with widespread connective tissue septae, circumscribed regenerative nodules of hepatocytes and anastomoses between vascular channels linking portal and central vessels.

Objective. To report the simultaneous cases of acromegaly and cirrhosis.

Case report. A 62-year old, male patient came to the hospital complaining of severe abdominal swelling. Laboratory and imaging findings were compatible with the presence of hepatitis B virus related cirrhosis together with acromegaly. In this case, he had high GH level but lower IGF-1 level because of hepatic failure which can impair IGF-1 production by the liver. Definitive diagnosis was made by pituitary MR and a 1 cm in diameter tumor was detected.

Conclusion. This paper showed that cirrhosis can result in a low IGF-I level in patients with acromegaly. There is no previous report available of the in the presence of coincidental combination of acromegaly and cirrhosis in a patient.

Key words: acromegaly, liver, cirrhosis

INTRODUCTION

Acromegaly is a syndrome characterized by the release of excessive amounts of growth hormone (GH) and insulin-like growth factor (IGF-1). The mean age of diagnosis is 40-45 and has the same frequency in both sexes. Approximately 95% of cases result from the pituitary adenomas (1).

Liver cirrhosis is defined chronic injury of the liver results in inflammation, necrosis and, eventually, fibrosis. Viral infections such as hepatitis B and C are the most common causes of liver cirrhosis all over the world. Patients with cirrhosis are at risk of developing many potential complications. The most common complication seen in patients with liver cirrhosis is ascites, and the most lethal one is bleeding varices (2).

*Correspondence to: Senay Topsakal, Pamukkale University, Faculty of Medicine, Department of Endocrinology and Metabolism, Denizli, 20070, Turkey, e-mail: stopsakal@pau.edu.tr

There is no previous report of acromegaly together with liver cirrhosis, in the literature. The aim of this study was to report a very rare combination of acromegaly with cirrhosis in a patient.

CASE REPORT

A 62-year old male patient with complaint of abdominal distension was admitted to our clinic (Fig.1). The patient stated that he had complaining of abdominal swelling for 6 months. In the last two weeks the swelling in the legs and feet, shortness of breath and insomnia have been added to the clinical findings. Initial examination revealed diffuse abdominal ascites and pretibial edema. Physical examination revealed findings suggestive of acromegaly, including coarse facial features and enlargement of

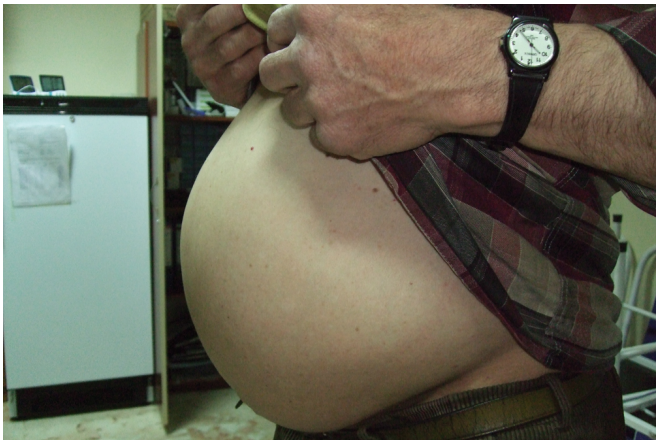

Figure 1. Severe ascites of the acromegalic patient with liver cirrhosis.

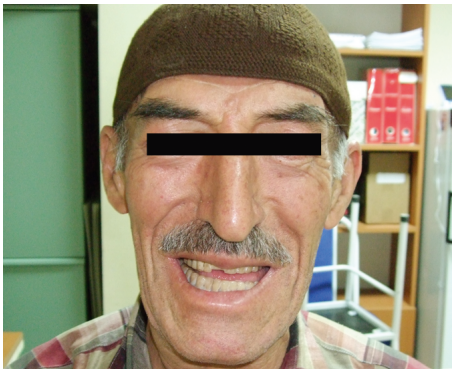

Figure 2. Acromegalic face: pronounced mandibular prognathism, widened and thickened nose, prominent supraorbital ridges, thick and coarsened lips and marked facial lines.

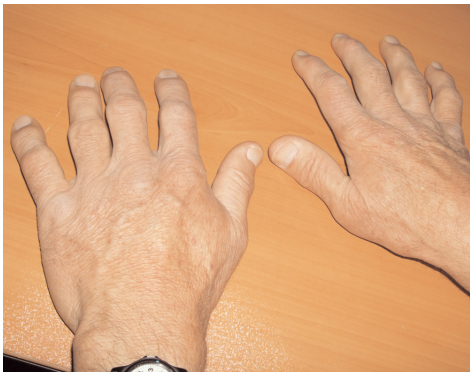

Figure 3. Acromegalic hand: characteristic enlarged hand of the patient, fingers are widened, thickened and stubby, and the soft tissue is thickened

Table 1. Laboratory findings, hormone profiles and hepatitis markers of the acromegalic patient with cirrhosis and reference values

	At admission	Follow up (6 months later)	Reference
Glu (mg/dL)	87	289	80 - 115
Cr (mg/dL)	0,6	1,05	0.7 - 1.2
Na (mmol/L)	141	136	136 - 145
K (mmol/L)	4	4,1	3.5 - 5.1
Tbil (mg/dL)	2,39	2,28	0.2 - 1.2
Dbil (mg/dL)	1,06	1,25	0 - 0.5
Prot (g/dL)	6,3	5,8	6 - 8
Alb (g/dL)	2,8	2,3	3.5 - 5
AST (IU/L)	104	114	5 - 35
ALT (IU/L)	83	86	10 - 50
Wbc (K/µL)	4.37	4.35	3.6 - 9.4
Hgb (g/dL)	11.9	10,2	13 - 17.5
Plt (K/µL)	47.9	49,8	142 - 424
PT	%62	%34	70 - 130
HBsAg (S/CO)	> 250.00	> 250.00	< 0.9
Anti HBs (mIU//mL)	2.06	2.06	< 7
GH (ng/mL)	15.4	51,4	0 - 1
IGF-1 (ng/mL)	50.3	71,7	71-290

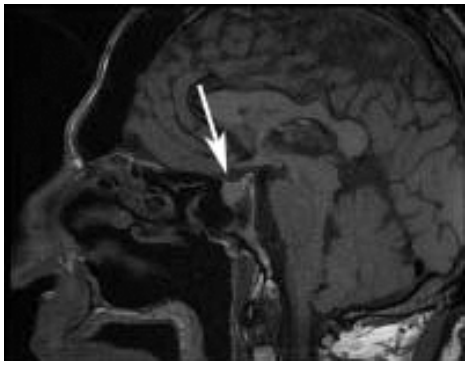

Figure 4. Sagittal T1- weighted MRI image of the pituitary. A tumor (arrow) is visible in and on the sella turcica. The mass is hyperintense compared to the brain parenchyma. The normal posterior pituitary "bright" signal is not seen.

his face, hands and feet (Fig. 2-3). There was no previous history of other feature. Laboratory findings at admission and 6 months follow- up are summarized in the Table 1.

No hepatic or gastrointestinal

tumors were detected at the liver ultrasound. The result of laboratory investigations was hepatitis-B induced liver cirrhosis. Because of high GH level, pituitary MR was taken on and adenoma 1 cm in diameter was detected. The surgery was not considered to patient, because of bleeding complications resulting from cirrhosis. Medical treatment was recommended but patients did not accept any treatment, for that reason follow-up was advised.

DISCUSSION

Acromegaly is a rare, insidious, and potentially life-threatening condition for the patient (3). The most important assays used for the diagnosis, management, and monitoring of acromegaly are GH and IGF-1 measurements (4). GH and IGF-1 levels are generally closely correlated in patients with acromegaly (5); however, discordance between GH and IGF-1 levels has been noted in some patients with acromegaly after treatment (6). IGF-1 is produced primarily by the liver as an endocrine hormone as well as in target tissues in a paracrine/autocrine fashion. Production is stimulated by GH and can be retarded by undernutrition, growth hormone insensitivity, lack of growth hormone receptors, or failures (7). Most discordance involves the measurement of normal GH levels and elevated IGF-1 levels, but some cases exhibit elevated GH levels and normal IGF-1 levels (6, 8). In this case, because of hepatic failure (which can impair IGF-1 production by the liver) IGF-1 level was below the normal level at admission and normal after 6 weeks follow-up.

The major comorbidities associated with acromegaly are cardiovascular disease, diabetes, hypertension, sleep apnea, arthritis, and metabolic bone disorders (osteoporosis) (9). A serious problem in patients with acromegaly is increased risk of colorectal and thyroid cancer (10). In present case, at the clinical examination acromegalic hands and face were observed together with ascites. However, low IGF-1 level, high GH level was suspected for acromegaly. After MR examination pituitary adenoma was diagnosed. There is no previous report about acromegaly and cirrhosis association.

Major complications of cirrhosis include ascites, spontaneous bacterial peritonitis, hepatic encephalopathy, portal hypertension, variceal bleeding, and hepatorenal syndrome (11). Hepatitis B incidence is increasing rapidly in developing countries. At the 50% of untreated patients is progress to chronic liver disease. The most common complaint of these cases is ascites (12). Our patient's major complaints were abdominal distension and ascites. These signs were classical suspected findings of cirrhosis. Laboratory investigations supported the diagnosis.

Patients with hepatitis are at increased risk of hepatocellular carcinoma (10). Acromegaly may accelerate the risk of cancer. Treatment was advised for both acromegaly and cirrhosis but the patient did not accept any treatment and close monitoring was recommended (13).

Acromegaly is not a common disease in human and cirrhosis

generally associated chronic liver diseases. In this case, patient's complaints were related to cirrhosis. At the clinical examination, acromegaly was diagnosed. This is the first case which shows acromegaly and cirrhosis together in a person.

Conflict of interest.

The authors declare that there is no conflict of interest.

References

1.Nachtigall L, Delgado A, Swearingen B, Lee H, Zerikly R, Kibanski A. Extensive clinical pattern in diagnosis and therapy of acromegaly over two decades. J Clin Endocrinol Metabol 2008; 93: 2035-2041.

2.Diaz-Brito V, Cardoso H, Sarmento A. Chronic viral hepatitis: protocol proposal for the management of cirrhosis. Acta Med Port, 2008; 21(2):179-192.

3.Giustina A, Chanson P, Bronstein MD, Klibanski A, Lamberts S, Casanueva FF, Trainer P, Ghigo E, Ho K, Melmed S. A consensus on criteria for cure of acromegaly. J Clin Endocrinol Metab, 2010; 95(7):3141-3148.

4.Pokrajac A, Wark G, Ellis AR, Wear J, Wieringa GE, Trainer PJ. Variation in GH and IGF-1 assays limits the applicability of international consensus criteria to local practice. Clin Endocrinol (Oxf) 2007; 67:65–70.

5.Ho KY, Weissberger AJ. Characterization of 24-hour growth hormone secretion in acromegaly: implications for diagnosis and therapy. Clin Endocrinol (Oxf) 1994; 41:75–83.

6.Alexopoulou O, Bex M, Abs R, T'Sjoen G, Velkeniers B, Maiter D. Divergence between growth hormone and insulin-like growth factor-1 concentrations in the follow-up of acromegaly. J Clin Endocrinol Metab 2008; 93:1324–1330.

7.Miura Y, Kato H, Noguchi T. Effect of dietary proteins on insulin-like growth factor-1 (IGF-1) messenger ribonucleic acid content in rat liver. British J Nut 2007; 67: 257-265.

9.Freda PU. Monitoring of acromegaly: what should be performed when GH and IGF-1 levels are discrepant? Clin Endocrinol (Oxf) 2009; 71:166–170.

10.Bex M, Abs R, T'Sjoen G, Mockel J, Velkeniers B, Muermans K, Maiter D. AcroBel the Belgian registry on acromegaly: a survey of the 'real-life' outcome in 418 acromegalic subjects. Eur J Endocrinol 2007; 157:399–409.

11.Tsai WL, Chung RT. Viral hepatocarcinogenesis. Oncogene, 2010; 29(16):2309-2324.

12.Heidelbaugh JJ, Sherbondy M. Cirrhosis and chronic liver failure: Part II. Complications and treatment. Am Fam Physician 2006; 74(5):767-776.

13.Kew MC. Hepatitis viruses and human hepatocellular carcinoma. Yale J Biol Med 1997; 70(1): 119–125.

14.Renehan AG, Brennan MB. Acromegaly, growth hormone and cancer risk. Best Practice Res Clin Endocrinol Metab 2008; 22 (4): 639–657.