Case report

Dietary treatment of chylous ascites in yellow nail syndrome

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SUMMARY Chylous ascites has rarely been reported in yellow nail syndrome. A case of chylous ascites in yellow nail syndrome is described which was treated successfully with dietary restriction of fat and supplements of medium chained triglycerides.

Samman and White first described yellow nail syndrome in 1964 and many cases have been reported since. The clinical features are yellow dystrophic nails, pleural effusions, bronchiectasis and lymphoedema. The underlying pathology is thought to be the result of a lymphatic abnormality. The occurrence of chylous ascites in yellow nail syndrome is rare and the first case was not reported until 1985 by Duhra et al whose patient was successfully treated with a Le Veen shunt and dietary manipulation. I report a case of chylous ascites in yellow nail syndrome responding to dietary manipulation without a shunt.

Case report

A 75 year old woman presented in April 1987 with two weeks' history of dyspnoea on exertion, cough with production of yellow sputum, and progressive abdominal swelling since December 1986. There was no history of altered bowel habit, anorexia, jaundice or alcohol abuse. Yellow nail syndrome was diagnosed at the age of 55 when she had recurrent pleural effusions, bronchiectasis, yellow dystrophic nails and lymphoedema. She has had repeated pleural aspirations and finally chemical pleuradeses. She also suffered from autoimmune hypothyroidism and was being treated with 0.1 mg thyroxine once daily.

On examination, she had yellow dystrophic finger and toe nails, lymphoedema of both lower limbs, bilateral pleural effusions and gross ascites. There was no rectal or pelvic mass.

Investigations showed a normal haemoglobin level of 12.8 g/dl, leucocytosis of 14×10^9 (85% neutrophils), thrombocytosis of 828×10^9/l, and and ESR of 102 mm in one hour. Biochemical profile revealed abnormal liver function tests; albumin reduced at 28 g/l, normal total protein of 64 g/l, AST and alkaline phosphatase were both raised at 91 IU/l and 270 IU/l respectively. Screenings for viral hepatitis, autoimmune antibodies and faecal occult blood were negative. Immunoglobulins and thyroid function test were with normal range. Rheumatoid factor was positive at 1:1024 dilution. Chest x-ray examination confirmed bilateral pleural effusions.

Paracentesis of the abdomen showed milky fluid with 45 g/l of protein, cholesterol and triglyceride levels consistent with that of chylous ascites. The ascitic fluid was negative on culture for acid-alcohol fast bacilli and other bacteria; cytology of the fluid showed no malignant cells and there was a heterogenous population of lymphoid and plasma cells. Ultrasound examination of the abdomen on two occasions and an abdominal computed tomography scan did not reveal any intra-abdominal pathology other than the ascites.

Her existing treatment with frusemide and spironolactone failed to improve the ascites and she was started on a low fat diet supplemented with medium chain triglycerides (MCT oil 50 ml/day) to avoid deficiency of essential fatty acids. The aim of this diet was to provide approximately 2500–2700 KJ of energy in the form of medium chain triglycerides and to reduce the intake of long chain fatty acids.

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On this regime of diuretics and diet, her weight reduced by 14 kg over the next three weeks. At 12 months follow up, she had lost 23 kg in weight and her abdominal girth reduced from 123 cm to 84 cm. She felt well and there was no obvious ascites or lymphoedema of her legs. Small pleural effusions and yellow dystrophic nails persisted, however. The biochemical profile and full blood count had returned to normal.

Discussion

The underlying pathology of yellow nail syndrome is probably that of a lymphatic abnormality. Patients with yellow nail syndrome have been shown to have abnormal lymphangiograms of the lower limbs and abnormal lymphatic histology of the pleura. The formation of chylous ascites is probably caused by oozing of lymph from the lymphangiectatic intestinal wall. The reason for the nail changes remains unclear.

Chylous ascites in adults usually result from intra-abdominal malignancy such as lymphoma or peritoneal TB. Its occurrence in yellow nail syndrome is very rare and the first case was reported by Duhra et al in 1985. Their 57 year old patient had a laparotomy which revealed diffusely thickened proximal jejunum and a peroral jejunal biopsy showed features of intestinal lymphangiectasia. A peritoneovenous shunt (Le Veen) was inserted and a diet low in long chain triglycerides and supplemented with medium chain triglycerides was started. The patient was free from ascites during the following 12 months. The authors attributed the success of their treatment to the shunt.

In this case, treatment with dietary manipulation and diuretics was entirely successful. The physiological basis of this treatment is to reduce the throughput of the intestinal lymphatic system and hence the formation of chylous ascites. Long chain triglycerides are absorbed into the lacteals while medium chain triglycerides are absorbed directly into the portal venous system. Therefore by replacing long chain triglycerides with medium chain triglycerides, the load on the abnormal lymphatic system is reduced.

In children, the treatment of chylous ascites of various aetiologies with dietary manipulation have had variable success. Unger et al reported a two-thirds success rate in children with chylous ascites treated with dietary manipulation alone. Treatment of chylous ascites in adults has not been well documented. Le Veen shunt has been tried but complications include sepsis, haemorrhage, leakage of ascitic fluid and disseminated intravascular coagulation. As shown in this case, treatment of chylous ascites in yellow nail syndrome with dietary manipulation and diuretics can be sufficient. Dietary manipulation should therefore be tried for a period of at least a month before considering surgery.

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References

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