Treatment of Ascites During Regular Dialysis Treatment with Intraperitoneal Colloidal Chromic (³²P) Phosphate

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Summary

This is a study of a patient on RDT who, after 3 months of PD and 23 months of HD, developed idiopathic ascites. Explorations performed in an effort to arrive at an aetiology diagnosis, as well as therapeutic measures used in treatment of the ascites, were negative. The ascites still persisted after one year. The general condition of the patient had deteriorated to severe cachexia. It was in this situation that 30 mCi of radioactive colloidal chromic phosphate (³²P) was administered intraperitoneally. In the weeks following this treatment the ascites disappeared and the serum albumin and the general condition of the patient returned to normal.

Introduction

The appearance of idiopathic ascites in which a defined aetiology could not be demonstrated has been described in patients receiving RDT (Mahoney et al, 1972; Zerefos et al, 1972). Therapeutic measures generally fail and the prognosis is very poor due to progressive cachexia. This is a case study of idiopathic ascites treated successfully with intraperitoneal injection of radioactive phosphorus in the form of colloidal chromic phosphate.

MATERIALS AND METHODS

The case presented is that of a 30-year old male diagnosed in February 1972 as having hypocomplementaemic membrano-proliferative glomerulonephritis, with
crescents in over 30% of the glomeruli studied in the renal biopsy. In June 1973 the patient was in terminal uraemia and dialysis treatment was initiated. Fourteen sessions of peritoneal dialysis of 48 hours per session were performed during the first 14 weeks. During one of the sessions, a sample of the peritoneal liquid gave a positive culture for Staphylococcus. This was treated with cloxacillin, after which the clinical symptoms disappeared and all further cultures of the peritoneal fluid were negative. Regular haemodialysis was initiated in October 1973, two sessions a week. A bilateral nephrectomy was performed in March 1974, after which the patient slowly began to gain body weight, so that in 4 months he had gained 3.5 kg but, at the same time, the serum albumin dropped from 3.2 g/100 ml to 1.5 g/100 ml. The patient complained of abdominal distension and physical examination revealed a moderate degree of ascites. Further examination at this time showed the following.

There was no evidence of cardiac insufficiency or fluid overload as shown by dyspnoea, elevation of the central venous pressure, oedema, tachycardia or hepatomegaly. The chest X-ray was negative for venous congestion, pleural effusion and pericardial effusion. There were no findings to indicate a clinical pericarditis, neither radiologically nor in the electrocardiographic studies.

Liver function tests were normal, including bilirubin, SGOT, SGPT, LDH, alkaline phosphatase and liver biopsy. The serum was negative for HBsAg.

There was no evidence of tumour or gastrointestinal or peritoneal malignancy. Radiological studies of the stomach and upper and lower gastrointestinal tracts were normal. Cytological studies of the ascitic fluid on three occasions showed no evidence of malignancy.

Microbiologic studies of three samples of ascitic fluid were negative in normal culture and in special media for fungi. There was no evidence of acid-fast bacilli.

Biochemical analysis of the ascitic fluid was similar to the serum. The total protein and albumin concentrations were high, 4.8 and 2.3 g/100 ml respectively, with an ascites/serum ratio of 0.80 and 0.77. The amylase level was normal in both the ascitic liquid and serum.

These negative findings established the diagnosis of idiopathic ascites (Figure 1). Treatment commenced with water restriction and ultrafiltration during dialysis. However ultrafiltration produced hypotension and had to be discontinued. The patient continued to gain weight and the ascites increased. In November 1974 the frequency of HD was increased to 3 sessions a week, but in spite of this the patient continued to gain weight, reaching a total increase of 6 kg. Because of progressive deterioration the patient entered hospital in February 1975 and a strict control was placed on his diet, including restriction of water and salt and an increase of protein intake to 80 g/daily. During the following 3 months there was a slight increase in the serum albumin from 2.6 to 3.0 g/100 ml but neither the ascites nor the general condition of the patient improved. His weight continued to increase, reaching a total of 7 kg over his normal weight. In a further effort to control the ascites, salt-free albumin was administered in
each HD session. A total of 980 g of albumin was administered during a 3 month period. Water restriction, the high protein diet and an attempt at ultrafiltration (unsuccessful because of hypotension) were continued during this period. With this treatment the serum albumin level increased from 3.1 to 4.1 g/100ml, but there was no weight reduction, and the patient appeared to have more ascites at the same weight, and was more cachectic. Treatment was suspended and at the end of one month the clinical picture was the same, but the serum albumin level had fallen to 3.8 g/100ml.

In the presence of idiopathic exudative ascites, it was decided to initiate the treatment that is the subject of this paper: the intraperitoneal injection of $^{32}$Phosphorus in the form of Colloidal Chromic Phosphate with a particle size between 600 and 1,000 Å. The total dose was 30 mCi divided in 2 doses of 15 mCi each.

The patient showed no evidence of local or general reactions from the treatment. Fifteen to 20 days after the first injection of the isotope, the weight and the ascites began to decrease. During the first month he lost 2.3 kg and during the following 4 months 2.0 kg more, reaching a total of 4.3 kg; at the same time the water restriction and the attempts at ultrafiltration were suspended.

As the ascites disappeared, general condition improved. The nutritional status also improved and the serum albumin, that had fallen as low as 3.0 g/100ml, increased to 3.4 and at the present time is 4.1 g/100ml. It is important to note here that given the poor nutritional state of the patient, the 7 kg weight gain during the year of intractable ascites was the result of the trapped ascitic fluid.
less the amount of the lean body weight lost during that time. The 4 kg lost represents the opposite, the loss of ascites less the weight gain resulting from the period of improved nutrition.

COMMENTS

In the last few years there have been several case reports describing the development of ascites in patients receiving RDT. In several of these cases (Arismendi et al, 1976) it was possible to identify a concrete etiology, thus allowing specific treatment. However, in the majority of cases it was impossible to identify a cause for the ascites and treatment was generally ineffective, the patients developing cachexia and having a generally poor prognosis. The patient described here falls within the latter group since there was no evidence of fluid overload, cardiac insufficiency, hepatic insufficiency, peritoneal infection, malignancy or pancreatic ascites. Traditional treatment failed completely, and the patient reached an advanced stage of cachexia.

The cause of the ascites is not clear. Possibly it can be included within the ‘exudative ascites group’ because of the non-specific peritoneal inflammation and the increased exudation from the lymphatic vessels. This suggestion is supported by the high concentration of total protein and albumin in the ascitic liquid with an ascites/serum ratio of almost unity (Craig et al, 1974), the numerous lymphocytes to be found on examination of the ascitic fluid, and the pathological findings of non-specific peritoneal inflammation (Rodriguez et al, 1974).

The case reported here meets the first 2 conditions. Idiopathic ascites seen in patients receiving RDT has been called ‘intractable ascites’ because of the failure of the various therapeutic measures employed. In this case, fluid restriction, high protein diet, intravenous albumin and ultrafiltration, all proved to be ineffective. The administration of almost 1 kg of albumin raised the serum albumin level from 3.0 to 4.1 g/100ml but the ascites was unaffected. It was impossible to use ultrafiltration during HD because of the resultant hypotension. A bilateral nephrectomy (Feingold et al, 1974) had been performed before the appearance of the ascites in preparation for a cadaver transplant. A kidney transplant is an effective therapeutic measure in ‘intractable ascites’ (Sing et al, 1974; Wang et al, 1974) but a suitable kidney was not available and the patient’s situation was deteriorating rapidly. Therefore treatment with the phosphorus isotope was instituted.

In intractable malignant ascites intraperitoneal administration of $^{195}$Au or $^{32}$P phosphorus ameliorates ascites in 60% of cases. This action does not seem to be fundamentally due to inhibition of tumour growth, but rather to the production of fibrosis of the peritoneum and the lymphatics. The effect is mediated by the emission of beta rays of limited penetration so that their action is confined to the peritoneum (O'Bryan et al, 1968).

Peritoneal and lymphatic fibrosis decrease the exudation of protein, thereby
decreasing the oncotic pressure of the peritoneal fluid, facilitating the reabsorption of the ascites, and increasing the serum protein level, reducing shift of interstitial fluid into the peritoneal space.

This mode of action of $^{32}$P would explain the sequence observed in this patient. The disappearance of the ascites did not begin until 2 weeks after the first injection of the isotope and was not complete for 5 months. Simultaneously, but at a lower pace, the serum albumin began to return to normal.

CONCLUSIONS

One cannot generalise from the therapeutic results in one case. However, due to the difficult and often futile therapy involved in the management of ‘intractable idiopathic ascites’ in patients receiving RDT, we feel that the successful treatment of this case with $^{32}$Phosphorus suggests that this treatment may have value in similar patients.

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