Articular complications in obese patients after jejunocolic bypass

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Jejunocolic shunt was introduced in 1956 as an effective method in treating massive obesity.[1, 2] Although weight loss after this operation was dramatic, the operation was abandoned because of multiple, occasionally fatal complications.[1, 3-9] Joint disease was a significant problem in our experience with 27 obese patients.

Polyarthralgias and polyarthritis developed in eight of these after a jejunocolic shunt procedure.

Patients and results

Between 1959 and 1968, jejunocolic shunt was performed in 27 obese patients at the Cleveland Clinic Hospital. The jejunum was transected 76 cm from the ligament of Treitz. The proximal end was anastomosed end-to-side to the mid-transverse colon (in one case to the ascending colon). In 15 patients, the distal jejunum was brought to the abdominal wall. A silastic tube was secured to it, making a jejunostomy available for fluid and electrolyte replacement.

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Table 1. Complications of jejunocolic shunt in 27 obese patients

Complications	No. affected/ no. tested
Electrolyte imbalance	13/25
Hypocalcemia	20/25
Hypomagnesemia	15/25
Hypoprothrombinemia	16/27
Tetany	6/25
Anemia	10/27
Cholelithiasis	4/27
Nephrolithiasis (oxalate stones)	2/27
Joint symptoms	8/27
Liver abnormalities* (histological)	15/15

* Including nutritional cirrhosis in four, fatty metamorphosis in eight, various degrees of infiltration of the portal areas in two, and encapsulated caseous granuloma in one patient.

There were 19 women and eight men; ages ranged between 22 and 47 years (average 33). Preoperative weights were between 102.8 kg and 204.3 kg (average 151.9 kg). Five patients were lost to follow-up between 1 and 59 months postoperatively (average 16.6). Six patients died 10 days to 62 months after surgery. Thirteen patients had take-down of their jejunocolic shunts 9 to 135 months postoperatively (average 44.1). Three patients still have the shunts 6 years and 2 months, 6 years and 7 months, and 9 years and 10 months after the operation.

Weight loss averaged 57.2 kg per person, the maximum weight loss was 126.7 kg. It was particularly marked in the 12th to 18th postoperative months, and in those patients with the highest preoperative weights. With few exceptions the weights stabilized thereafter.

Postoperative complications included dehydration, electrolyte imbalance, hypocalcemia, hypomagnesemia, hypoprothrombinemia, postural hypotension, tetany, joint symptoms, anemia, cholelithiasis, and nephrolithiasis (oxalate stones) (*Table 1*). Liver abnormalities included fatty metamorphosis, nutritional cirrhosis, and fatal hepatic failure. Similar complications have been reported by other authors.[1, 3-9]

Joint manifestations (*Tables 2 and 3*)

Eight patients, seven women and one man, complained of joint symptoms as early as 2 months and as late as 33 months postoperatively. Ages ranged from 24 to 39 years (average 30). Preoperative weights were from 107.6 kg to 195.2 kg (average 136.4 kg). Weight losses ranged from 15.9 kg to 95.3 kg (average 63 kg).

Joints with pain or synovitis were: ankles in eight patients, wrists in seven, knees in seven, hips in six, shoulders in five, fingers in five, elbows in four, and neck in one. Several joints were involved either simultaneously or successively in all but one patient. Involvement was sometimes symmetrical. Objective findings of arthritis (heat, redness, swelling, effusion, and limitation of motion) were present in only four patients. In two patients migratory polyarthralgias associated with chills and fever appeared. One patient had tenosynovitis of the wrists. Three patients also had diffuse myalgias. The severity of rheumatic symptoms appeared to be out of proportion to the objective findings in all eight. The duration of symptoms varied. In seven patients the syndrome lasted from 3 to 23 months. Arthralgia developed in another patient 2 months postoperatively, and

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Table 2. Clinical findings in patients with joint symptoms

Case no.	Age	Sex	Preop. wt., kg	Wt. loss, kg	Postop. joint symptoms started, mo	Duration of joint symptoms, mo	Joints affected in sequence	Comments
1	26	F	107.6	49.0	4	5½	Ankles; rt. knee; rt. hip; rt. wrist	——
2	31	F	127.1	65.8	10	3	Diffuse polyarthralgia	Myalgias
3	28	F	126.2	15.9	2	?	Lt. hip; lt. ankle; lt. knee; shoulders; rt. wrist	Died
4	38	M	128.9	58.1	10	12	Diffuse polyarthralgia; rt. wrist; migratory polyarthralgias with chills and fever; rt. knee and PIP joints of fingers	Arthritis; ACTH and prednisone treatment
5	24	F	123.5	56.8	24	23	Ankles; wrists; shoulders; diffuse polyarthralgias	Arthritis; tenosynovitis lt. wrist; hyperpigmentation
6	27	F	195.2	95.3	33	?	Ankles	No follow-up
7	27	F	137.6	71.3	24	7	Fingers; wrists; ankles; neck; diffuse polyarthralgias	Myalgias; arthritis
8	39	F	145.3	88.5	6	20	Diffuse polyarthralgias PIP joints of the hands	Arthritis; myalgias; tuberculosis

Table 3. Laboratory data on patients with joint symptoms

Case no.	Serum calcium	Magnesium serum	Uric acid	Serum glycoproteins	Rheumatoid factor	LE prep	Erythrocyte sedimentation rate
1	9.3	0.6	4.8	148	Neg	—	1.2
2	8.7	1.3	5.4	184	—	—	—
3	8.3	1.3	7.2	142	Neg	Neg	—
4	9.2	—	—	196	Neg	Neg	0.8
5	9.1	1.9	6.2	—	Neg	Neg	0.1
6	8.9	1.6	5.5	124	—	—	—
7	9.3	0.7	—	—	—	—	—
8	8.3	1.8	5.5	—	Neg	Neg	33
Normal values							
	8.5–11 mg/100 ml	1.5–2.2 mg/100 ml	3–6.5 mg/100 ml	125–145 mg/100 ml	Neg	Neg	<0.65 mm/min <20 mm/1 hr

she died shortly after. The cause of her death was not determined.

Roentgenographic examinations of the affected joints were normal. Serologic tests including the L.E. preparation, antinuclear antibodies, and rheumatoid factor were negative in patients so tested. The erythrocyte sedimentation rate was mildly elevated in three of four patients tested. Serum glycoproteins were mildly elevated in two of five, and the serum fibrinogen value was elevated in two patients so tested. Serum uric acid was elevated transiently in one patient. All patients had serum calcium values over 8 mg/100 ml. Four patients had serum magnesium values less than 1.5 mg/100 ml. There was no difference between those who had tube feeding jejunostomy and those who did not. Synovial fluid was obtainable from knee effusions in

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Table 4. Synovial fluid analysis from jejunocolostomy patients

	Case 4	Case 8
Color/vol.	Pale yellow/8 cc.	Water white/22 cc.
Clarity	Transparent	Transparent
Viscosity	High	High
Mucin clot	Tight clot	Tight clot
WBC (%PMN)	1400 (10%)	750 (25%)
Crystals	None	None
Inclusion cells	None	None
Latex test	N.T.	Negative
Complement	N.T.	N.T.

N.T. = Not tested.

two patients. Analysis of this fluid revealed a bland, noninflammatory (group I) effusion in both patients (*Table 4*). Neither inclusion cells nor giant macrophages were detected. One fluid was tested for rheumatoid factor (latex fixation) and found to contain none. Complement components were not sought.

The course of joint disease was benign in all patients and without any residual deformity. Generally the arthralgias were modified with analgesic treatment, but in two patients ACTH or corticosteroids were necessary to relieve the arthritic manifestations. Severe musculoskeletal symptoms necessitated the restoration of the bowel continuity in one patient. Symptoms disappeared in this patient as well as in two other patients in whom normal bowel continuity had to be restored for other reasons.

Discussion

Among the complications of jejunocolic shunt in obese patients,[1, 3-9] joint symptoms have been the primary subject of only one report. Shagrin et al[3] described a polyarthritic syndrome in seven of 22 patients with jejunocolic shunt which affected the wrists in seven, the fingers in five, the knees in three, and the ankles in three. In their study arthritis usually lasted less than 12 months, but in two patients it persisted more than 3 years. The weight loss in these patients ranged from 31.8 kg to 78.1 kg. The rheumatoid factor was negative. The course was benign except for one patient in whom persistence of the joint symptoms necessitated the take-down of the shunt with prompt and lasting relief. The clinical and biochemical findings in their patients are relatively similar to our findings. However, objective manifestations of arthritis were present in only four of our patients and myalgias constituted significant symptoms in three.

The cause of this syndrome is not known. Rheumatoid arthritis and gout seem to be unlikely in both series. Hyperuricemia was absent in all but one of our patients despite marked weight loss and occasional metabolic acidosis. Available roentgenographic studies of the joints of these patients exclude the possibility of chondrocalcinosis. No connective tissue disease was present. The occasional elevation of serum fibrinogen and glycoprotein values is nonspecific and indicative of

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an inflammatory process which is neither highly active nor present in all cases. Hypomagnesemia which was present in four of our patients has not been reported in association with any joint symptoms.[10]

Shagrin et al[3] considered this syndrome in the group of the so-called entero-arthropathies, i.e., diseases of the joints which are associated with various bowel disorders. Arthritis is known to occur in 2% to 22% of patients with chronic ulcerative colitis and in 3% to 10% of patients with Crohn's disease.[11-14] The arthritis is often monoarticular with an acute or subacute course, and usually involves the knees, ankles, and proximal interphalangeal joints of the fingers. Attacks and remissions of arthritis generally follow exacerbations and remissions of colitic symptoms. The course of arthritis in patients with jejunocolic shunt is dissimilar. In addition there was no active bowel inflammation in our patients with jejunocolic shunt. In 15 patients who had restoration of bowel continuity, histologic examination of the bypassed jejunal segment revealed either normal structure or villous atrophy. The functioning segment of the jejunum was either normal or hyperplastic. Furthermore, additional differences exist between the arthritis of inflammatory bowel disease and that seen in intestinal bypass. For instance, involvement of sacroiliac joints[15] and ankylosing spondylitis[16] were not present in our patients.

Arthritis was reported in 65% of patients with Whipple's disease,[17] a disorder characterized by diarrhea, malabsorption, and weight loss, symptoms similar to those present in our patients. In one of our patients skin hyperpigmentation, which is seen frequently in Whipple's disease,[17] appeared. However, arthritis usually precedes other symptoms in Whipple's disease and in addition jejunal biopsy in this as well as in other patients was negative. No evidence of the bacterial enteritides (Shigella or Salmonella), occasionally complicated by arthritis, was found in our patients. Although Reiter's syndrome may follow diarrheal states,[18] neither the clinical features of urethritis, keratodermia, conjunctivitis, nor typical synovial fluid findings were present in our series. Similarly, a self-limiting migratory nondestructive synovial reaction involving knees, ankles, elbows, and wrists may accompany diarrhea and other gastrointestinal manifestations in Behçet's syndrome,[19-20] but no clinical features of this were noted in our patients.

The fact that arthritis has not complicated jejunoileal shunt merits comment. It is conceivable that the preservation of the ileocecal valve prevents the contamination of small bowel by the bacterial flora of the colon. Whether a toxic product of metabolism of these bacteria absorbed in the small bowel injures synovia is not known. Improvement of the joint symptoms in one patient described by Shagrin et al[3] coincided with antibiotic treatment. However, this does not explain the absence of arthritis in other patients with jejunocolic shunt. Furthermore, joint symptoms are not a feature of the intestinal stasis syndrome where there is bacterial overgrowth in the small bowel.

Arthritis of obscure origin occasionally occurs in certain malignancies.[21-22] In these circumstances where a catabolic state prevails, the products of breakdown of tissues may play a role in

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arthritis either because of the intrinsic toxic effects or because of inability of these debilitated patients to metabolize and detoxify them. An autoimmune etiology of arthritis in these patients has been also suggested.[22] The iatrogenic malabsorption in patients with jejunocolic shunt creates a hypercatabolic state which may be a factor in the pathogenesis of arthritis. Indeed, our patients with joint symptoms lost more weight on average than those without joint symptoms. Conceivably, latent infectious organisms (virus, mycoplasms, chlamydia) could have a more salutary milieu when the host suffers accelerated catabolism.

Joint symptoms have been described in patients with acute or chronic liver disease.[23-26] The cause of arthritis in these disorders is not clear, although in viral hepatitis immune complex deposits cause the synovitis.[26] Two of our patients had histologic evidence of nutritional cirrhosis, two had lymphocytic infiltration in the portal areas and one had severe fatty metamorphosis of the liver. In another patient examination of the liver at autopsy revealed an encapsulated caseous inflammation of the liver without evidence of active tuberculosis. In the remaining two patients, no liver biopsy was performed. However, both had abnormal bromsulphalein retention of 10% and 14% respectively. However, in nine other patients with abnormal liver histology no joint symptoms were present. Therefore, we cannot surmise a uniform relationship between hepatic abnormality and joint difficulty in this group.

Summary

Articular symptoms and diffuse myalgia developed in eight of 27 patients who had undergone jejunocolic shunt for the treatment of massive obesity. Polyarthritis developed in four patients. Migratory polyarthralgias with chills and fever developed in two. Symptoms persisted for 3 to 23 months. The course was benign, without residual deformity, and synovial fluid obtained from two patients was noninflammatory. Analgesics controlled joint symptoms in all except two patients who required treatment with adrenocorticotropin (ACTH) or prednisone. In one patient the arthritic pain was relieved only after take-down of the shunt. In two other patients restitution of the normal bowel continuity for other reasons resulted in the disappearance of symptoms.

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