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**Single Case**

# Primary Eosinophilic Panniculitis of the Greater Omentum in a Young Girl: A Case Report

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**Keywords**
Greater omentum · Panniculitis · Eosinophil · Hypersensitivity

**Abstract**
***Introduction:*** Primary (or idiopathic) panniculitis involving the intra-abdominal adipose tissue is rare, and its pathogenesis remains unknown. A case of primary eosinophilic panniculitis that involved the greater omentum of a girl is reported. ***Case Presentation:*** The patient, an 11-year-old girl, complained of dull periumbilical pain and nausea, and radiological examination showed a mass lesion in the abdomino-pelvic cavity. On laparoscopy, a plaque-like, flat mass was seen in the greater omentum, and laparoscopic omental resection was performed. On histopathological examination, the interlobular fibrous septa of omental adipose tissue were widened by inflammatory edema, prominent infiltration of eosinophils, and loose proliferation of myofibroblasts. Dense lymphocytic infiltration was also noted around small veins. Inflammatory changes were mild in the fat lobules, and fat necrosis and infiltration of lipid-laden macrophages were absent. Findings of obliterative phlebitis or arteritis were not seen. ***Conclusion:*** Isolated involvement of the omentum by a panniculitic process is rare, and the pathogenesis of eosinophilic septal panniculitis found in the present case remains unknown, but involvement of a hypersensitivity reaction against some unknown stimuli is presumed, based on the histopathological resemblance of the omental lesions to erythema nodosum or eosinophilic panniculitis of the skin. We should keep in mind the possibility that the omental lesion in this patient is a harbinger of more serious immunological disorders. Careful, long-term follow-up and monitoring of the patient are needed.

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## Introduction

Primary inflammatory disease of the intra-abdominal adipose tissue ("panniculitis") is an uncommon disorder [1–3]. In most cases, it affects the mesentery of the small intestine ("mesenteric panniculitis") or the mesocolon [1–3]. Isolated involvement of the greater omentum by this pathological process ("primary omental panniculitis") is very rare [4, 5]. Most previous reports of primary omental panniculitis discussed its clinical or radiological aspects, and detailed pathological studies are scarce. The etiology and pathogenesis of this disease remain obscure.

A case of an isolated, acute inflammatory lesion that affected the greater omentum of an 11-year-old girl is reported. The omental lesion in this case showed interesting histopathological characteristics: (1) inflammation predominantly affecting the fibrous septa of adipose tissue; (2) inflammatory cell infiltration consisting mainly of eosinophils; and (3) dense lymphocytic infiltration observed around small veins in the septa. Based on the histopathological resemblance to erythema nodosum (EN) and the prominent eosinophil infiltration, it was presumed that a hypersensitivity reaction against some unknown stimuli played a major role in the pathogenesis of the omental panniculitis in the present case.

## Case Report

The patient was an 11-year-old girl who consulted a physician 6 days after she began experiencing vomiting and 5 days of dull periumbilical pain. Although the patient had a history of allergic rhinitis, she did not have a history of other allergic disorders (including food allergy, drug allergy, bronchial asthma), parasitic infection, collagen vascular diseases, infection with coronavirus (COVID-19), or neoplastic disease. She had not had a sore throat suggestive of streptococcal infection and had been in good health until the present episode. No history of abdominal trauma or prior abdominal surgery was elicited. Physical examination showed a well-nourished, afebrile girl with slight tenderness of the lower abdomen. No skin rashes were noted, and the remainder of the physical examination was within normal limits. Computed tomography and magnetic resonance imaging (Fig. 1) showed an accumulation of a small amount of ascites and a heterogeneously enhancing, irregular mass lesion in the left lateral abdomino-pelvic cavity. Laboratory examination showed leukocytosis ($105 \times 10^2/\mu L$) with neutrophilia (78.9%) and elevation of C-reactive protein (4.381 mg/dL), serum CA 125 (46.5 U/mL), and soluble interleukin 2-receptor (645 U/mL). No eosinophilia of the peripheral blood was seen (eosinophils: 1.0%). Straw-colored ascites of 20 mL was obtained by abdominal paracentesis, and CA125 in the ascites was found to be elevated (518.6 U/mL).

Laparoscopic omental resection was performed based on the clinical diagnosis of omental neoplasm. At operation, no ascites was noted. The omentum was thickened and lightly attached to the left abdominal wall. The serosal surfaces of the mesentery, stomach, and intestines were smooth, and neither hyperemia nor fibrinous exudative change was noted. No other inflammatory lesions were found in the abdominal cavity. The patient's postoperative recovery was uneventful. She has been healthy after the operation and followed as an outpatient.

*Pathological Findings*

On gross examination, the resected greater omentum showed a blunt-ended triangular shape and measured $18.5 \times 9.5$ cm$^2$. The surface was smooth, and attachment of fibrin or inflammatory exudate was not seen. The omental adipose tissue was normal in amount. In the center of the omentum, a poorly circumscribed, plaque-like, thickened area of elliptical shape,

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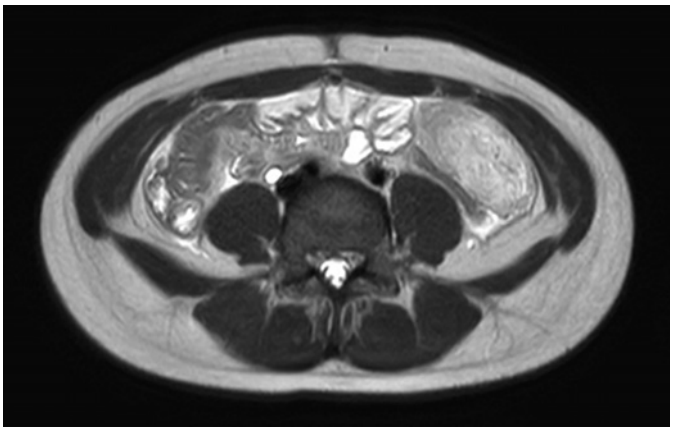

**Fig. 1.** Abdominal MRI, T1-weighted image with contrast enhancement. A heterogeneously enhancing, poorly circumscribed mass lesion is seen in the left lateral abdomino-pelvic cavity. MRI, magnetic resonance imaging.

measuring about 8 × 6 × 1.4 $cm^3$, was observed (Fig. 2). The thickened area was slightly hard and whitish on the cut surface.

On histopathological examination, the lobular structure of the omentum was well retained, and the surface was covered by mesothelial cells that focally showed mild hyperplastic change. In the thickened area, inflammatory changes predominantly involving the interlobular septa of adipose tissue ("septal panniculitis") were observed (Fig. 3a, b). The septa were widened by exudation of edema fluid, inflammatory cell infiltration, an increase of thin collagen fibers, and loose proliferation of fibroblasts or myofibroblasts (Fig. 3c). Although the septa showed sheet-like expansion in some places, the inflammatory changes were largely restricted to the septa, and extension of the changes into the periphery of fat lobules remained mild. The absence of fat necrosis, lipid-laden macrophages, and histiocytic giant cells was a notable finding. Dystrophic calcification was also not observed.

Inflammatory cell infiltration consisted mainly of eosinophils, and lymphocytes and histiocytes were intermingled. The density of eosinophils reached more than 100 cells per high-power field in the highest area (Fig. 3d). Plasma cells were scant, and neutrophils were not observed. Formation of lymph follicles or epithelioid cell granulomas was not seen. Small blood vessels were increased in number, and the vascular lumina were dilated. Dense lymphocytic infiltration often surrounded small veins, and a few veins showed intramural lymphocytic infiltration (Fig. 3e). However, the lumina of the involved veins remained patent, and features of obliterative phlebitis or necrotizing arteritis were not observed. Lymphatic vessels in the omentum were unremarkable, except mild dilatation of their lumina.

Immunohistochemical studies were conducted using an automated immunostainer, Leica Bond-Max (Leica Biosystems, Wetzlar, Germany). Monoclonal or polyclonal primary antibodies against the following substances were used: α-smooth muscle actin (α-SMA) (clone 1A4, Dako, Glostrup, Denmark, 1:100), desmin (clone D33, Dako, prediluted), anaplastic lymphoma kinase (ALK) (clone ALK01, Ventana Medical Systems, Tucson, AZ, USA, prediluted), β-catenin (clone 14/beta-catenin, Becton Dickinson Transduction Laboratories, San Jose, CA, USA, 1:400), podoplanin (clone D2-40, Dako, prediluted), CD3 (polyclonal, Dako, prediluted), CD4 (clone 4B12, Dako, 1:80), CD8 (clone C8/144B, Dako, 1:100), CD20 (clone L26, Dako, prediluted), CD34 (clone QBEnd, Leica Biosystems, 1:400), CD68 (clone KP1, Dako, prediluted), and CD138 (clone MI15, Dako, prediluted). Pretreatment by protease digestion or heat-induced antigen retrieval was performed when indicated.

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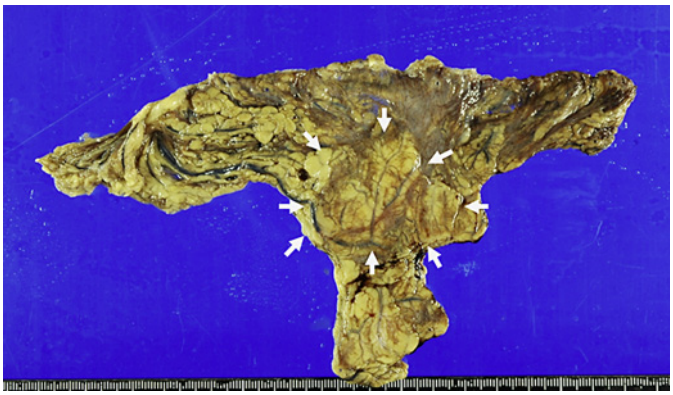

**Fig. 2.** Gross appearance of the resected greater omentum. In the central portion, a plaque-like, flat mass lesion is found (arrows).

Immunohistochemical examination showed that most of the proliferating spindle cells within the septa were α-SMA-positive and desmin-negative myofibroblasts (Fig. 3f). Spindle cells were negative for ALK and β-catenin. Lymphocytes were distributed predominantly in the perivenous region, and CD20-positive cells and CD3-positive cells were almost equal in number. The majority of CD3-positive lymphocytes were also CD4-positive. CD138-positive plasma cells were far fewer than lymphocytes and distributed mainly within the fat lobules. In contrast, CD68-positive macrophages showed a diffuse distribution in both septal and intralobular areas, being more common in the septal areas. Macrophages with swollen, lipid-laden cytoplasm were not observed.

## Discussion

Mesenteric panniculitis is a benign fibro-inflammatory disease of the mesenteric adipose tissue [1–3], and omental panniculitis that involves adipose tissue of only the greater (or occasionally lesser) omentum [4, 5] is considered a localized form of mesenteric panniculitis [1]. Mesenteric (including omental) panniculitis often occurs in association with a variety of intra-abdominal processes such as surgery, trauma, inflammatory, or vascular conditions such as mesenteric thrombosis [1–3]. Primary (or idiopathic) panniculitis not associated with these disorders is rare.

Primary mesenteric panniculitis occurs mostly in adults and is radiologically recognized as a localized flat mass, multinodular lesion, or diffuse thickening of the mesenteric adipose tissue [1–3]. It also occasionally affects children [4], and the disease usually pursues a self-limited clinical course resolving within a few weeks or months [1–5]. However, since clinical or radiological differentiation from neoplasms such as malignant lymphoma or various mesenchymal neoplasms, or from malformative lesions like a lymphangioma, is difficult, surgical exploration is deemed necessary for diagnostic purposes. Definitive diagnosis of panniculitis usually depends on histopathological examination of biopsied or excised specimens. Idiopathic, global, or segmental infarction is another condition of the omentum that is more frequently encountered than omental panniculitis, and its histopathology is characterized by fat necrosis and reparative changes [6]. In the present case, there were neither clinical nor pathological findings suggestive of omental infarction.

From a histopathological perspective, mesenteric panniculitis forms a wide spectrum of fibro-inflammatory disease that ranges from mesenteric lipodystrophy (a degenerative

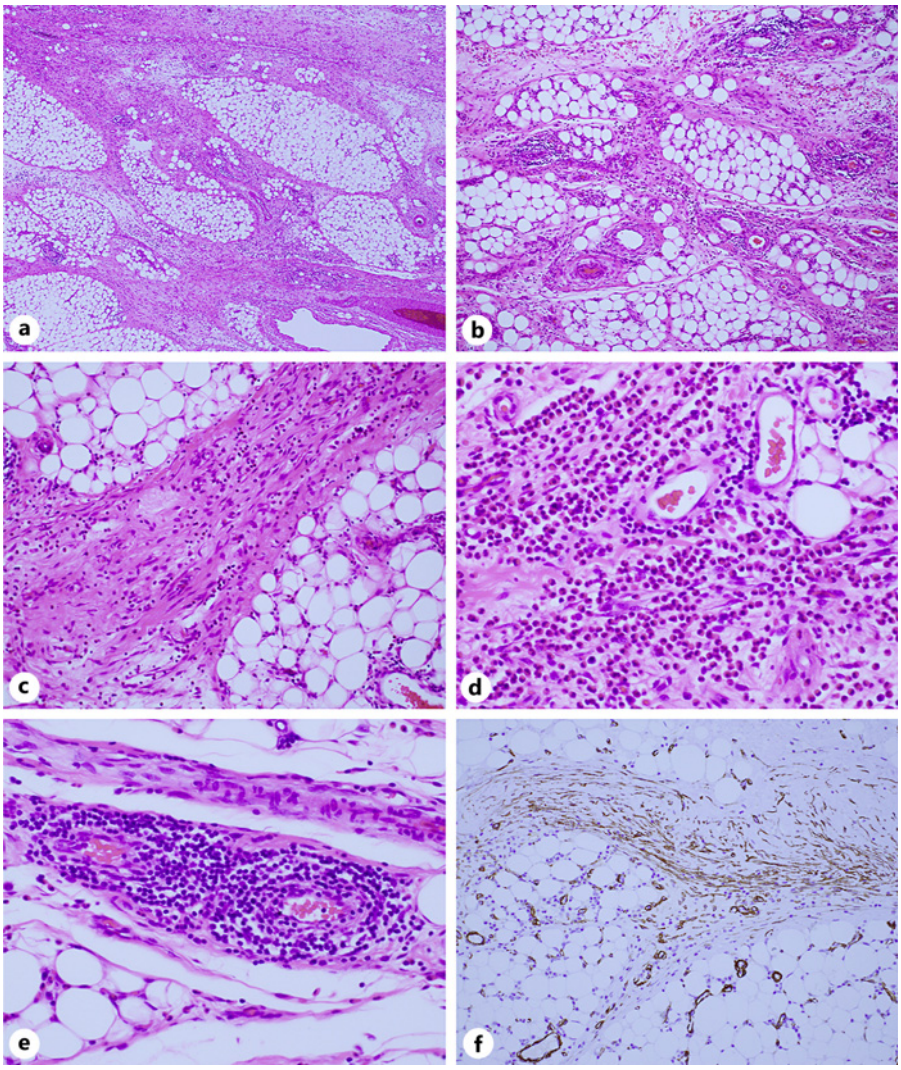


**Fig. 3.** Microscopic findings of the omentum. **a** The septa of omental adipose tissue are widened by edema, inflammatory cell infiltration, and proliferation of fibrous tissue. The septa occasionally show sheet-like expansion, but fat lobules are well preserved (HE stain, ×40). **b** The widened septa show inflammatory cell infiltration consisting mainly of eosinophils. Dense lymphocytic infiltration is seen around small veins in the septa. Extension of the inflammatory changes into the periphery of fat lobules is mild (HE stain, ×100). **c** Spindle cells have proliferated forming loose fascicles in the septa (HE stain, ×200). **d** In some areas, the inflammatory infiltrate in the septa is almost exclusively occupied by eosinophils (HE stain, ×400). **e** Lymphocytes densely surround small veins in the septa and partly show intramural infiltration, but the venous lumina remain patent (HE stain, ×400). **f** Most spindle cells proliferating in the septa are immunoreactive for α-SMA (immunostained for α-SMA, ×200).

change of adipose tissue), via mesenteric panniculitis (an inflammatory reaction), to sclerosing mesenteritis (a fibrosing, reparative process) [1]. The pathological differential diagnosis includes neoplastic lesions, such as inflammatory myofibroblastic tumor, intra-abdominal fibromatosis (desmoid tumor), and malignant lymphomas [1–3]. As a rare tumorous lesion specifically affecting the omentum and mesentery, "omental-mesenteric myxoid hamartoma" [7] should also be considered. This disease affects infants and is considered an infantile variant of myofibroblastic tumor [7]. Histopathologically, it shows sparse proliferation of primitive mesenchymal cells on the background of a prominently myxoid

matrix containing various numbers of inflammatory cells. However, the differentiation from these neoplastic disorders was not difficult in the present case because the lobular architecture of omental adipose tissue was well retained, and neither mass-forming proliferation of myofibroblasts nor myxoid change of the stroma was observed.

The omental panniculitis in the present case showed the following characteristic features: (1) inflammation predominantly involved the fibrous septa of adipose tissue ("septal panniculitis") and lacked fat necrosis of the lobules and infiltration of lipid-laden macrophages; (2) inflammatory cell infiltrates mainly consisted of eosinophils; and (3) dense lymphocytic infiltration was observed around small veins and also within the walls of some veins. The absence of fat necrosis and infiltration of lipid-laden macrophages was a notable finding, but it may be ascribed to surgical intervention at the early stage of disease progression in the present patient. No previous case report documenting the pathological findings similar to those in the present case, i.e., septal panniculitis in a girl that was restricted to the omentum and showed predominant eosinophil infiltration, could be found in the literature.

The etiology and pathogenesis of mesenteric panniculitis remain unknown. In addition to a relationship with the various associated disorders mentioned before, the possibility that it represents a manifestation of IgG4-related disorders [8] or a paraneoplastic phenomenon [9] has also been suggested. Furthermore, deep adipose tissue including the mesentery has attracted attention in recent years as an inflammatory organ in the human body [2, 10]. Some investigators suggest that, in mesenteric panniculitis, preadipocytes within adipose tissue have more potential to convert into macrophages, and various proinflammatory adipocytokines secreted from the converted macrophages initiate and maintain chronic, low-grade inflammation of adipose tissue [2].

In the present case, the inflammatory changes predominantly affected the septa of adipose tissue, producing a histopathological resemblance to EN of the skin, although granulomatous lesions or histiocytic giant cells were not observed. EN is a prototypical septal panniculitis affecting subcutaneous adipose tissue [11]. It is considered a hypersensitivity reaction and is often associated with various inciting disorders [11]. Whereas streptococcal infection is the most frequent etiological factor for EN in children, inflammatory bowel disease is one of the most commonly associated diseases in adults with EN [11].

The predominance of eosinophils in the present case also resembled eosinophilic panniculitis of subcutaneous adipose tissue [12]. The pathogenesis of eosinophilic panniculitis is unknown, but, similar to EN, this disorder is also frequently associated with streptococcal infection, various hematological disorders, collagen vascular diseases, or solid neoplasms, thus suggesting a close relationship to a hypersensitivity reaction [12]. There are also several autoimmune or immune-mediated disorders characterized by an intense eosinophilic infiltration, including eosinophilic granulomatosis with polyangiitis. We consider that the histopathological resemblance of the omental lesion to EN or subcutaneous eosinophilic panniculitis suggests that a hypersensitivity reaction localized in the omental adipose tissue played an important role in the pathogenesis in the present case.

Another pathogenetic possibility is that eosinophilic omental panniculitis in the present case might be an extension of eosinophilic gastro-intestinal disorder (EGID) [13]. In a subtype of EGID, eosinophil infiltration predominantly involves the subserosa of the stomach or intestines and is associated with eosinophilic peritonitis [13, 14]. In this subtype of EGID, it is possible that eosinophilic inflammatory changes may extend to the mesentery or omentum, and, actually, a very rare case of EGID of an adolescent male that accompanied eosinophilic omental panniculitis has been reported by Tan-Lim and colleagues [15]. In the present case, since peripheral eosinophilia was absent and the clinical symptoms subsided after omentectomy, endoscopic examination of the gastro-intestinal tract is not scheduled for the time

being. At laparoscopy, at least, no findings suggestive of the subserosal subtype of EGID were macroscopically observed.

In conclusion, the histopathological resemblance to EN or subcutaneous eosinophilic panniculitis of the omental lesion in the present case suggests that a hypersensitivity reaction played an important role in its pathogenesis, although the inciting stimulus or stimuli that had caused the reaction remain unknown. We have to keep in mind the possibility that the omental lesion in this patient may be a harbinger of other allergic or immune-mediated disorders. Careful, long-term follow-up and monitoring of the patient for possible evolution of these disorders are needed.

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## Statement of Ethics

This study protocol was reviewed, and the need for approval was waived by the Ethics Review Board of Kansai Medical University Hospital. Written informed consent to use clinical data, pathological material, and any accompanying images was obtained from the parents of the patient in accordance with the 1964 tenets of the Declaration of Helsinki and its later amendments. The CARE Checklist for this case report is available as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000544861).

## Conflict of Interest Statement

The authors declare that they have no conflicts of interest.

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## Author Contributions

Masayuki Shintaku drafted the manuscript and figures. Tokiko Okunobo provided the clinical information and the radiological figures. Hiroki Nakamura, Takashi Doi, Akira Tanaka, and Koji Tsuta supervised the work and gave critical advice on the contents of the article. All the authors read and approved the final manuscript.

## Data Availability Statement

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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