

Case Report

Effective Treatment of Advanced Hepatocellular Carcinoma with Extensive Peritoneal Dissemination Using Lenvatinib

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Patients with hepatocellular carcinoma (HCC) and extensive peritoneal dissemination generally have a poor prognosis and are often resistant to systemic therapy. We report the case of a 47-year-old woman with HCC and massive peritoneal dissemination who presented with malignant ascites requiring repeated cell-free and concentrated ascites reinfusion therapy and peritoneovenous shunt placement, as well as malignant pleural effusion requiring pleurodesis. Combined immunotherapy with durvalumab/tremelimumab was initiated; however, disease progression was observed after three treatment courses, prompting a switch to lenvatinib therapy. Two months after initiation of lenvatinib, CT imaging demonstrated complete disappearance of arterial enhancement in the primary hepatic lesion, along with reduction in the size of peritoneal dissemination nodules. Thirteen months after switching to lenvatinib (16 months after the initial diagnosis), the alpha-fetoprotein level continued to decrease, and the disease remained stable under treatment. Despite the extremely high tumor burden, lenvatinib achieved disease stabilization and symptomatic improvement.

Key words: diagnostic laparoscopy, hepatocellular carcinoma, peritoneal dissemination, lenvatinib

The introduction of immunotherapy and molecularly targeted agents has improved the prognosis of patients with unresectable hepatocellular carcinoma (HCC) [1-3]. However, patients with HCC and extensive peritoneal dissemination have a particularly poor prognosis, and treatment resistance is highly likely even with systemic therapy [4]. Herein, we report a case of HCC with extensive peritoneal dissemination treated by aggressive palliative intervention and lenvatinib, which may have contributed to longer survival.

Case Presentation

A 47-year-old woman (height 156 cm, weight

69 kg, and body mass index 28.4 kg/m²) presented with a chief complaint of abdominal distension. Upon visiting her previous physician, computed tomography (CT) revealed an ovarian tumor, peritoneal carcinomatosis, and ascites. Suspecting ovarian cancer, her physician referred her to the gynecology department of our institution. The patient had no history of pregnancy, childbirth, notable medical conditions, or alcohol consumption. Contrast-enhanced CT showed numerous disseminated nodules in the peritoneum and omentum, accompanied by ascitic fluid, as well as a 110-mm tumor in liver segment S2/3 with heterogeneous enhancement (Fig. 1A-B). The right ovary exhibited irregular enlargement (Fig. 1C). Liver function tests were normal, and both the serum hepatitis B surface

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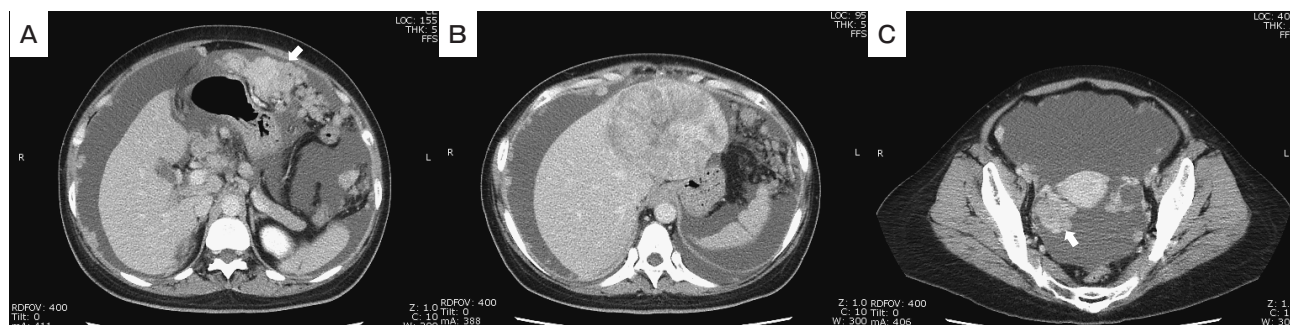


Fig. 1 Computed tomography findings. **A**, Numerous peritoneal and omental disseminated nodules (arrows) are noted, accompanied by a significant amount of ascites; **B**, A mass measuring 110 × 94mm is detected in segment S2/3 of the liver; **C**, An irregular-surfaced mass lesion (arrow) is observed in the right ovary.

Table 1 Laboratory examination

Hematology		Biochemistry			
WBC	5,600/ μ l	TP	7.7 g/dl	BUN	7.3 mg/dl
Hb	11.0 g/dl	Alb	4.0 g/dl	Cre	0.5 mg/dl
Ht	37.7%	AST	38 U/l	Na	139 mEq/l
Plt	49.1×10^3 / μ l	ALT	9 U/l	K	3.4 mEq/l
Serology		LDH	195 U/l	Cl	100 mEq/l
CEA	0.7 ng/ml	ALP	101 U/l	T-Cho	200 mg/dl
CA19-9	13.1 U/ml	γ -GTP	26 U/l	CRP	2.91 mg/dl
CA125	714 U/ml	T-Bil	0.5 g/dl	Coagulation	
CA72-4	0.9 U/ml	D-Bil	0.2 g/dl	PT(%)	79.9%
AFP	240,563 ng/ml	ChE	220 U/ml	APTT	27.4 sec
SCCA	0.2 ng/ml	AMY	19 U/l	D-dimer	5.4 μ g/ml

WBC, white blood cell; Hb, hemoglobin; Ht, hematocrit; Plt, platelet; CEA, carcinoembryonic antigen; CA, carbohydrate antigen; AFP, alpha-fetoprotein; SCCA, squamous cell carcinoma antigen; TP, total protein; Alb, albumin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; ALP, alkaline phosphatase; γ -GTP, γ -glutamyl transpeptidase; Bil, bilirubin; ChE, cholinesterase; AMY, amylase; BUN, blood urea nitrogen; Cre, creatinine; T-Cho, total cholesterol; CRP, C-reactive protein; PT, prothrombin time; APTT, activated partial thromboplastin time.

antigen and hepatitis C antibody tests were negative. Serum tumor markers indicated elevated levels of alpha-fetoprotein (AFP) (240,563 ng/ml) and carbohydrate antigen (CA) 125 (714 U/ml), whereas carcinoembryonic antigen (CEA), CA19-9, CA72-4, and squamous cell carcinoma antigen levels remained within normal limits (Table 1). Based on these findings, peritoneal dissemination and hepatic metastases secondary to ovarian cancer were considered likely. For definitive diagnosis, diagnostic laparoscopy with bilateral salpingo-oophorectomy and resection of disseminated omental nodules from the omentum was performed. Histopathological examination revealed that the tumor was predominantly localized to the ovarian surface rather than within the ovarian stroma (Fig.2A).

The tumor cells demonstrated a cord-like structural pattern resembling HCC morphology (Fig.2B). Immunohistochemistry revealed positive staining for hepatocyte, glypican-3, and AFP (Fig.2C,D), and negative staining for estrogen and progesterone receptors. Based on these results, a diagnosis of primary HCC with peritoneal carcinomatosis was confirmed. At the time of diagnosis, the patient had an ECOG performance status (PS) of 0. The patient had Child-Pugh class B (7 points), attributable to ascites (3 points), with otherwise preserved liver function. On the 36th day after her initial visit, the disease was classified as Barcelona Clinic Liver Cancer (BCLC) stage C unresectable HCC, and treatment with durvalumab plus tremelimumab (Dur/Tre) was initiated. However, her

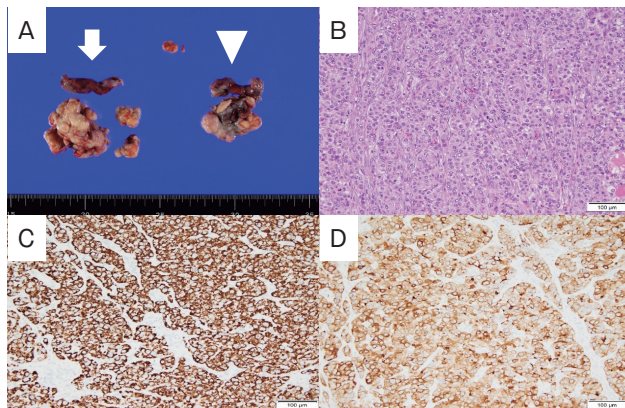


Fig. 2 Macroscopic and microscopic imaging. **A**, Macroscopic findings. The arrows indicate the right ovary and fallopian tube, and the arrowheads indicate the left ovary and fallopian tube. The tumor cells are predominantly distributed on the ovarian surface rather than within the ovarian stroma; **B**, Hematoxylin and eosin staining showing that the tumor cells exhibited a trabecular arrangement, a pattern frequently observed in hepatocellular carcinoma; **C**, Immunohistochemistry positive for hepatocyte antigen; **D**, Immunohistochemistry positive for glypican-3.

abdominal distension worsened owing to significant ascites. Consequently, she underwent three sessions of concentrated ascites reinfusion therapy (CART), followed by placement of a peritoneovenous (PV) shunt for palliation of malignant ascites. Left-sided pleural effusion and atelectasis necessitating thoracic drainage were also observed. Cytological examination confirmed a malignant pleural effusion, after which pleurodesis was performed. Despite PV shunt placement, ascites accumulation and abdominal distension persisted, necessitating CART every two weeks for ascites control. In terms of anticancer therapy, after three cycles of durvalumab maintenance therapy following Dur/Tre combination therapy, serum AFP levels increased dramatically (426,022.5 ng/ml), and the tumor size had enlarged; therefore, the disease was judged to be progressive. Nevertheless, the patient's PS and nutritional condition were maintained through intensive symptom management, and liver function remained at Child-Pugh class B (7 points), allowing a switch to lenvatinib. Following the introduction of lenvatinib at 12 mg/day, tumor marker levels decreased steadily: serum AFP declined from 426,022.5 ng/ml to 277,355.1 ng/ml, and PIVKA-II from 55,938 mAU/ml to 6,546 mAU/ml. CT imaging demonstrated complete disappearance of arterial enhancement in the primary hepatic lesion along

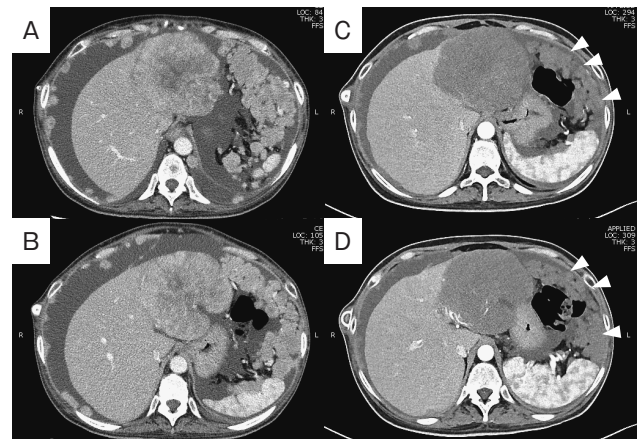


Fig. 3 Computed tomography (CT) scan before and after lenvatinib administration. **A, B**, CT scan obtained prior to lenvatinib treatment; **C, D**, CT scans obtained 8 weeks after initiation of lenvatinib; **C**, The primary hepatic lesion, measuring 106 × 92 mm, remained similar in size compared with baseline; however, arterial enhancement within the lesion disappeared; **C, D**, Peritoneal dissemination nodules (arrowheads) decreased in size and demonstrated reduced contrast enhancement.

with a reduction in the size of peritoneal dissemination nodules (Fig. 3A-D). Ascites was managed without the need for further CART sessions. Thirteen months after switching to lenvatinib (16 months after the initial diagnosis), the AFP level had further decreased to 150,490.6 ng/ml (Fig. 4). No recurrence of ascites-related symptoms was observed, and the disease remained stable under treatment.

Discussion

A nationwide survey conducted in Japan during 2014-2015 underscored the rarity of peritoneal dissemination in patients with HCC: it was observed in only 63 of 17,754 cases (0.4%) [5]. When multiple peritoneal disseminations, liver tumors, and ovarian tumors are observed, ovarian cancer should be considered first, as it is common for ovarian cancer to accompany liver metastases and peritoneal dissemination [6]. In the present case, liver biopsy was not attempted due to ascites; instead, laparoscopic exploration and bilateral adnexal resection were performed for histological diagnosis. Histopathologically, tumor tissue was observed at the ovarian surface margins, indicating peritoneal dissemination lesions rather than a primary ovarian origin. The patient was therefore diagnosed with pri-

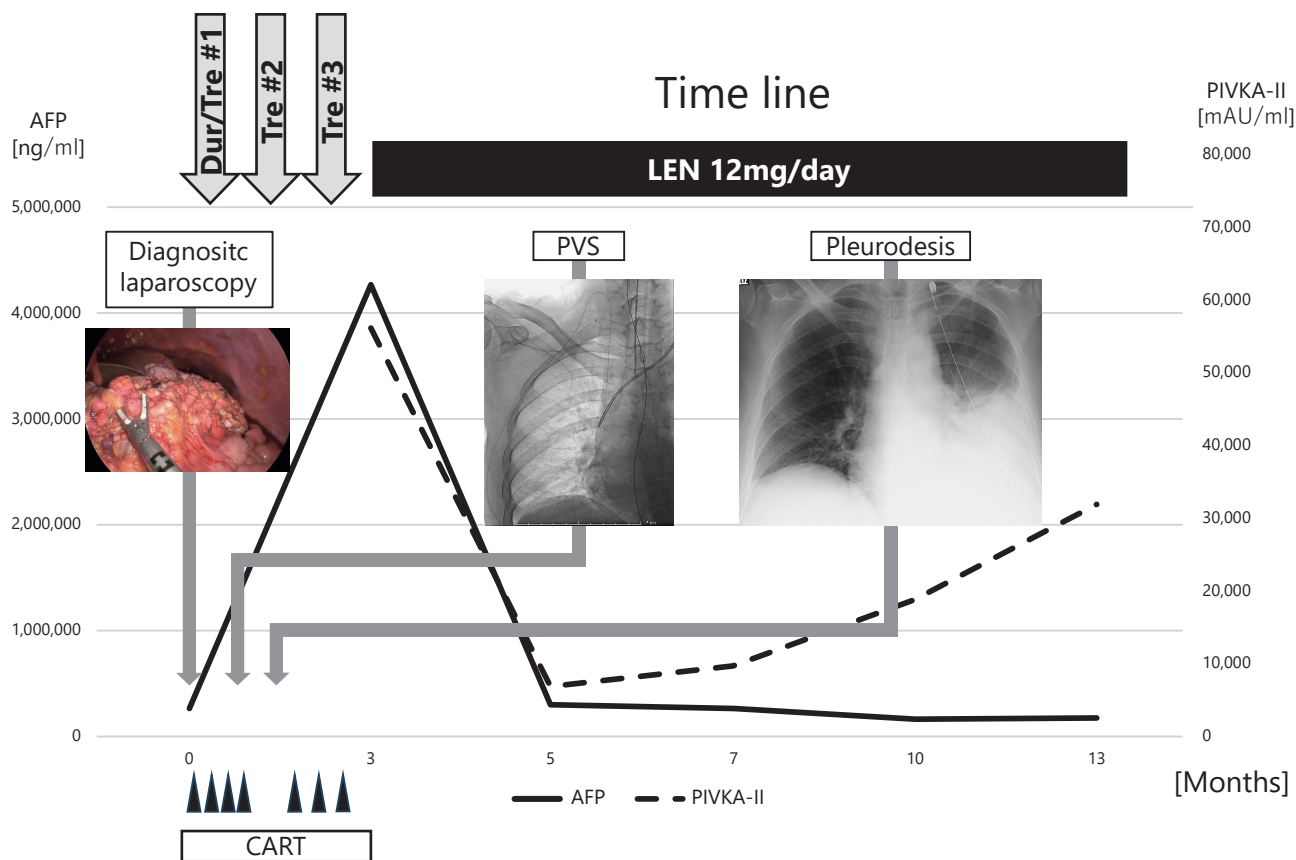


Fig. 4 Treatment timeline. Following diagnosis, the patient initially received durvalumab/tremelimumab (Dur/Tre) and was switched to lenvatinib (LENV) treatment after three cycles. She underwent four sessions of concentrated ascites reinfusion therapy (CART), followed by placement of a peritoneovenous shunt (PVS) for malignant ascites. Pleurodesis was also performed for left-sided pleural effusion. Despite PVS placement, ascites accumulation and abdominal distension persisted, necessitating CART every two weeks until LENV initiation. After the introduction of LENV, tumor marker levels decreased steadily. AFP, alpha-fetoprotein; PIVKA-II, protein induced by vitamin K absence or antagonist II.

mary HCC accompanied by multiple peritoneal disseminations.

Although a few case reports have described long-term disease control with immunotherapy in HCC patients with peritoneal dissemination [7,8], no previous reports have documented successful long-term control of massive and diffuse peritoneal dissemination by systemic therapy alone. HCC with extrahepatic metastases, including peritoneal dissemination, corresponds to an advanced stage in the BCLC staging system, for which systemic therapy is the standard treatment [9]. However, because systemic therapy has generally been regarded as ineffective against peritoneal dissemination, surgical resection and cytoreductive strategies have occasionally been attempted in selected cases with limited peritoneal disease [4,10,11]. Our

case therefore represents a unique clinical scenario in which lenvatinib achieved disease control and symptomatic improvement despite an extraordinarily high peritoneal tumor burden. This suggests that lenvatinib may provide clinical benefit even in patients with extensive peritoneal dissemination, a population that has traditionally been considered refractory to systemic therapy.

Durvalumab plus tremelimumab (Dur/Tre) is a combination immunotherapy indicated as a first-line treatment for unresectable HCC [1-3]. Similarly, atezolizumab plus bevacizumab (Atezo/Bev), a combination of immunotherapy and anti-VEGF therapy, is also approved for first-line treatment of unresectable HCC [3]. Lenvatinib, a molecularly targeted agent, is indicated as a first-line treatment for unresectable HCC

[2] and is commonly used as a second-line treatment following immunotherapy [12]. Previous reports have indicated treatment outcomes for these agents, with progression-free survival (PFS) of 3.8 months, 6.9 months and 7.4 months, and objective response rates of 20.1%, 29.8% and 24.1% for Dur/Tre, Atezo/Bev and lenvatinib, respectively [1-3]. Dur/Tre and Atezo/Bev were equally recommended as first-line therapeutic options in the patients applicable for immunotherapy according to the Japanese guidelines [13]. In the current case, the patient was relatively young with preserved PS. Because the patient was considered able to tolerate immune-related adverse events, Dur/Tre was selected. However, since the disease was judged as progressive disease after the evaluation of therapeutic efficacy, the treatment protocol was shifted to lenvatinib. In the current case, the pretreatment CRAFTY score was 2 (defined as CRP > 1 and AFP > 100), classifying the patient into the poor-response group for immunotherapy [14].

Although somewhat reiterative, this case is notable in that lenvatinib demonstrated marked efficacy not only against the primary hepatic lesion but also with measurable regression of peritoneal dissemination. PFS was 13 months after initiation of Lenvatinib, slightly longer than the 7.4 months reported in clinical trials [2]. Given the extensive peritoneal dissemination, this represents a remarkable outcome.

Lenvatinib was identified through exploratory research of agents with various tyrosine kinase inhibitory activities related to angiogenesis [15]. Lenvatinib targets vascular endothelial growth factor receptor 1-3 and fibroblast growth factor receptor 1-4 to suppress angiogenesis and inhibit the proliferation of HCC [16]. HCC is widely known as a hypervascular tumor, which may account for the relatively greater efficacy of anti-angiogenic therapies in HCC compared with other cancer types. The poor vascular perfusion of peritoneal dissemination generally has been implicated as a cause of reduced chemotherapeutic efficacy [11]. In the case of HCC, its hypervascular characteristics may play an important role in the antitumor efficacy observed not only in the primary tumor but also in the peritoneal dissemination.

The peritoneal cavity is considered an immunological niche due to the presence of the peritoneal-plasma barrier, which limits access to both chemotherapy and the immune system, as well as the influence of para-

crine factors such as cytokines, chemokines, and growth factors [17]. Evidence from colorectal and gastric cancer has demonstrated significantly poorer survival in patients with peritoneal metastasis accompanied by ascites compared with those with peritoneal metastasis without ascites or with non-peritoneal metastatic sites [18]. Thus, patients with HCC who present with peritoneal metastasis and ascites may not be suitable candidates for immunotherapy.

Synergistic effects of lenvatinib with immunotherapy, including immune activation, reduction of regulatory T cells, and suppression of TGF- β signaling, have been reported and are considered promising [19]. In clinical practice, second-line lenvatinib following Atezo/Bev has demonstrated both safety and efficacy [12]. Similarly, lenvatinib after Dur/Tre, another immunotherapy regimen, may have contributed to the long-term disease control observed in the current case.

Moreover, prognostic factors associated with favorable outcomes for lenvatinib include good liver function at the time of initiation [20] and adequate nutritional status [21]. In the present case, despite exhibiting resistance to first-line treatment, the patient maintained a good PS, which may have contributed to the observed treatment response. Prior to treatment initiation, proactive measures were taken to manage pleural effusion and ascites. These aggressive palliative interventions, including PV shunt surgery and pleurodesis, may have contributed to maintaining the patient's good performance status.

Conclusion

We report a case of HCC with extensive peritoneal dissemination in which lenvatinib treatment achieved long-term disease control. Despite the extremely high tumor burden, lenvatinib led to disease stabilization and symptomatic improvement. Our findings suggest that lenvatinib may provide clinical benefit even in patient populations that have traditionally been considered refractory to systemic therapy.

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