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Lymphatic Dynamics of Liver by Hepatic Lymphography Using Lipiodol Ultra Fluid*

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Summary

Hepatic lymphography by intra-parenchymal injection of four to ten millilitres (ml) of lipiodol ultrafluid, our modification of functional hepatography, performed on sixty one patients helped study lymphatic dynamics of liver. In conditions associated with significant hepatic venous outflow obstructions, such as hepatic cirrhosis, inflammatory diseases of liver and primary or secondary malignant lesions of the liver, this study delineated liver lymphatics and portal and para aortic lymph nodes. In one case mediastinal nodes were also delineated by flow of lipiodol from the bare area of the liver via trans-diaphragmatic and pleural lymphatics. The lymphangio-architecture of the opacified nodes depicted the nature of pathology inflicting them and the liver. Lipiodol in the lymphatic system, staying longer than the freely diffusible aqueous contrast, provided more detail and better information.

Introduction

Radiological demonstration of liver lymphatics by functional hepatography, i.e. by intra-parenchymal injection of aqueous contrast media in certain conditions associated with hepatic venous outflow obstruction has been reported by *Moreno* et al. (1963) and *David Clain and McNulty* (1968). The freely diffusible aqueous contrast provided only transient delineation of hepatic lymphatics and the lymph nodes of drainage were not satisfactorily opacified. So, the advocates of functional hepatography concluded that incidental demonstration of liver lymphatics was indicative of some intra-hepatic pathology producing obstruction to hepatic venous outflow, that no specific lymphatic patterns of useful diagnostic value could be obtained and that the investigation would be useful for the study of haemodynamics of liver. X-ray opacification of hepatic lymph nodes following intravenous injection of Tantalum dust has been reported by *Dumont and Martelli* (1969). Hepatic lymphography consists of injection of lipiodol ultrafluid into the liver parenchyma. Lipiodol ultrafluid, being viscous, is cleared along with liver lymph and, since it is non-irritant, does not cause any pain or local damage. Whenever there was obstruction to the venous outflow of the liver by inflammatory, degenerative and primary or secondary neoplastic pathology, liver lymph along with the contrast was drained by beaded lymphatics coursing towards the porta hepatis and upper para-aortic regions. The less diffusible lipiodol ultrafluid, staying longer in the lymphatic system, provided more detail and information about the hepatic lymphatic dynamics. Also the lymphangio-architecture of the opacified lymph nodes in the porta and para-aortic regions indicated the nature of pathologic process inflicting them and the liver. Since the intra-parenchymal injection of lipiodol ultrafluid into the liver had enabled the study of lymphatic dynamics of liver with delineation of liver lymphatics and lymph nodes of drainage comparable to the results obtained by thyroid lymphography (4) and perilingual lymphography (5) we have preferred to designate the technique as hepatic lymphography. The purpose of this communication is to present our results of hepatic lymphography and to emphasize its safety, simplicity and diagnostic utility.

Subjects and Method

Hepatic lymphography has been performed on sixty one patients comprising normals (ten), cirrhosis liver (twenty), amoebic abscess of

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liver (twenty), amoebic abscess of liver (ten), lepromatous leprosy (twelve), primary carcinoma of the liver (one), secondary carcinoma of the liver (five), Hodgkin's disease with lymphomatous involvement of nodes in the porta hepatis (one), adrenal medulloblastoma with secondaries in the liver (one) and hydatid cysts of the liver (one). The age groups varied from one and a half years to sixty - three years. Ten out of the sixty one patients were below twelve years.

Technique of Hepatic Lymphography

Haemorrhagic diathesis and thrombocytopenia were excluded in all the patients by determining bleeding time, clotting time, platelet counts and prothrombin time routinely prior to the study. Liver function tests (total serum proteins, albumen globulin ratio and thymol turbidity) were done only in thirty seven patients (ten normal, twenty cases of cirrhosis and seven patients with malignant involvement of liver). Urine analyses and SGOT estimation were done in all the patients. Serum bilirubin estimation was done only in seven patients with malignancy and jaundice. These investigations were done prior to and the day after hepatic lymphography and there was no change in the laboratory values except in two cases of cirrhosis liver in whom there was a rise of about 20 units of SGOT level. The SGOT values prior to lipiodol injection were 36 and 21 units; on the day after the study the values were 58 and 51 units respectively. In our subsequent studies, we are not routinely undertaking these chemical tests unless indicated. Whenever there was large amount of ascites, paracentesis was done just prior to the procedure.

The patient lies supine on the x-ray table with the film set in position to cover the liver area and chest. A preliminary (scout) skiagram of the liver area and chest taken routinely, serves as a useful guide for the needle path and location of the liver and to exclude emphysematous lungs, hepatodiaphragmatic interposition of colon and any pre-existing restrictive respiratory pathology. Under minimal sedation and local xylocaine analgesia, a 21 gauge long needle is passed into the liver to a depth of four to five centimetres through the ninth intercoastal space in the mid axillary line. For local analgesia, a separate syringe with 24 gauge needle and two ml of 2% xylocaine is used. After infiltrating in the intercostal space, when the needle touches the liver, the capsule is just entered and one half ml of xylocaine is injected beneath the capsule and into the substance of liver and the needle withdrawn. Five minutes later liver puncture with the 21 gauge needle is done at the same site. After ascertaining that no blood or bile is drawn, ten ml of lipiodol ultrafluid are manually injected in about one and a half to two minutes. For children upto ten years, we use a dose of one ml per year of age with a minimum of four ml. During injection, quiet respiration is allowed, but, during liver puncture, exposure of the films and withdrawal of the needle, breath is held without any attempt at deep inspiration. Bucky skiagrams covering the liver area and chest are taken when five, eight and ten ml have been injected. In the case of children, when lesser quantity of contrast is injected, films are exposed when one third, two thirds, and the whole of the contrast has been injected. After withdrawal of needle, digital pressure at the site of puncture will help to prevent any oozing of blood or lipiodol. Sequential roentgenograms are obtained at 5, 10, 30 and 60 minutes as well as 2, 4, 6 and 24 hours after injection. Daily studies help to evaluate the clearance of contrast from the liver. Following the investigation patient is kept in bed rest for six to eight hours.

Probable Complications and Management

Lipiodol reaching the pulmonary circulation after clearance from the liver via hepatic veins did not cause any adverse symptoms or effects in most of our patients. Three patients developed cough, minimal dyspnoea and complained a feeling of tightness in the chest, immediately following injection. These symptoms were relieved promptly by intramuscular injection of antihistamine and decadron 4 mg. Lipiodol leaked back along the needle track and spilt into the peritoneal cavity of two

patients with hepatic malignancy and the study had to be repeated after a week by entering the liver at a different site. Leakage of lipiodol into the peritoneal cavity did not cause any ill effects. On the evening of the investigation, a few patients developed mild pyrexia up to 100° F. This was relieved by oral paracetemol. None of the patients developed pain in the liver area, jaundice or impaired appetite. We did not encounter any serious complication in any of the sixty one patients studied.

Results and Discussion

The injected lipiodol gets deposited in the lymph spaces of *Disse and Mall* (Fig. 2, 3 and 5) and its clearance along with liver lymph depicts the lymphatic dynamics of the liver parenchyma. In the normal (Fig. 2), since most of the liver lymph flows via hepatic veins, the parenchymal lipiodol deposit is cleared mostly via hepatic venous channels upwards and medially towards inferior vena cava to reach the right heart circulation and pulmonary vascular tree delineating pulmonary arterioles and capillaries. Roentgenograms obtained 30 to 60 minutes after injection show miliary and submiliary mottling in the lung fields due to pulmonary lipiodol micro-embolization. This usually clears in 48 to 96 hours without any remarkable symptoms or effects. The amount of lipiodol refluxing into and delineating the intra-hepatic portal tree is variable in different cases. This contrast also flows into hepatic sinusoids and gets cleared but slowly by the hepatic veins and also by flowing into a fine interlacing network of lymphatics within the liver. Skiagrams obtained at 24 hours and later show the hepatographic phase consisting of a mottled ground glass appeareance due to the contrast in the hepatic sinusoidal capillary bed as well as fine interlacing lymphatic network. The lipiodol molecules are phagocytosed by the cells lining the sinusoids as well as the lymphatics and cause prolonged opacification. So, in the normal liver, delineation of intra hepatic portal and hepatic venous channels and hepatographic phase could be obtained; but the large lymphatics coursing towards portal hepatis are usually not visualized.

Whenever hepatic venous network is choked by hepatic cirrhosis or malignant infiltration, lipiodol flows into a network of beaded lymphatics within the liver coursing towards porta hepatics and para-aortic regions delineating cisterna chyli and thoracic duct (Fig. 3). Twenty four hour and later skiagrams show the portal and para aortic nodes (Fig. 4 and 5). The lymphangio architecture of the opacified nodes help to assess the nature of pathology inflicting them.

An eleven year old boy was given complete course of treatment for histologically verified tuberculous lymphadenopathy in the cervical, axillary and inguinal regions on both sides. He reported one year after treatment with recurrence of these lymph node masses, hepatosplenomegaly and jaundice. Liver biopsy done twice was negative. The patient was put on anti-tuberculous therapy for the second time on the basis of histology report of caseating tuberculosis on the axillary lymph node. Hepatic lymphography demonstrated a leash of liver lymphatics coursing towards portal and para-aortic lymphatics, cisterna chyli and thoracic duct (Fig. 5). There was remarkable absence of delineation of hepatic outflow and portal reflux. Portal and para-aortic lymph nodes depicted lymphoma pattern on delayed lymphograms (Fig. 5 and 6). The nodes were uniformly enlarged with preservations of margins and multiple large intranodal filling defects giving rise to a foamy appearance. Based on lymphographic features, a diagnosis of lymphoma (Hodgkin's disease) was made and this was subsequently confirmed by biopsy report on infraclavicular nodes that were increasing in size while under anti-tuberculous therapy. In this case of co-existent tuberculosis and Hodgkin's disease, the diagnosis of lymphoma was first suggested by the portal and para-aortic nodes delineated by hepatic lymphography performed for the investigation of hepatosplenomegaly and jaundice. After satisfactory response to cyclophosphamide therapy with regression of hepatomegaly, peripheral nodes and opacified nodes in the ab-

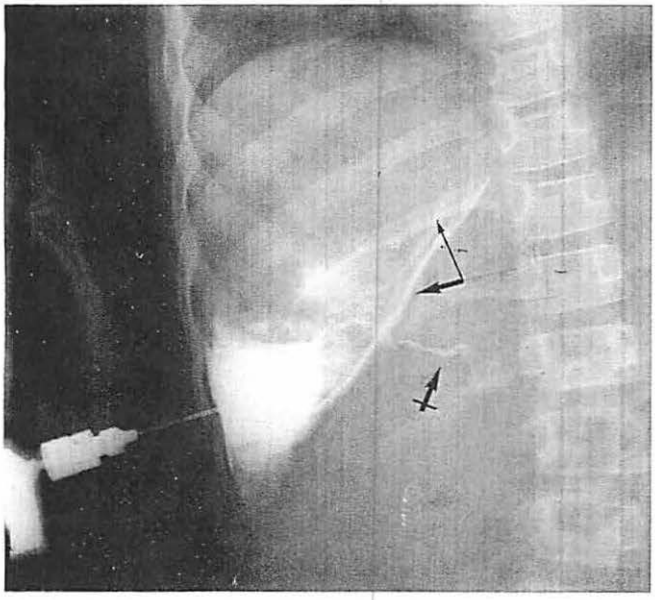

Fig. 1. Functional hepatography by intrahepatic parenchymal injection of aqueous contrast showing hepatic venous flow (→) and delineation of single liver lymphatic (↠) coursing towards porta. (Subsequent lipiodol study in this case showing liver lymphatics better is shown in Fig. 5 & 6.)

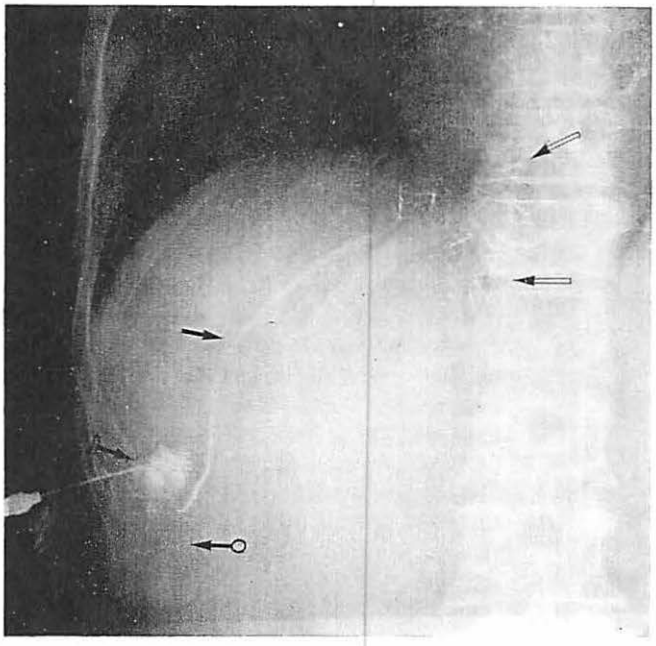

Fig. 2. Demonstrates the technique of hepatic lymphography in the normal with reflux of lipiodol from lymph spaces of Disse into hepatic (→) and portal (○→) veins. Note the fast clearance of lipiodol via hepatic vein (→) and its appearance in the pulmonary arterioles (⇒) and a little stasis in the portal veins.

domen, a repeat hepatic lymphography revealed re-establishment of hepatic venous outflow, delineation of normal intra-hepatic portal tree and intensification of the regressed nodes in the abdomen. The lymphatic delineation then was minimal. Liver biopsies did not provide evidence of Hodgkin's involvement of liver, though lymphographic features were strongly in favour of this diagnosis. This case substantiated our claim that hepatic lymphography could be more revealing in certain situations when liver biopsy studies might be inconclusive.

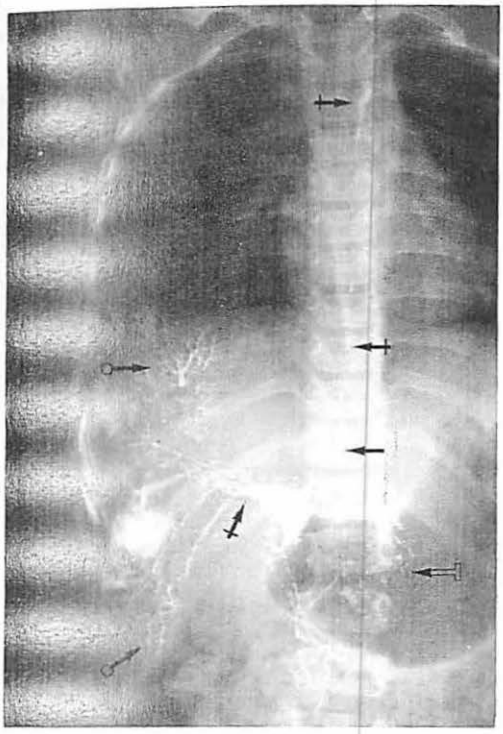

Fig. 3. Eight year old boy, a case of histologically verified hepatic cirrhosis showing liver lymphatics (→) leading to porta hepatis, para-aortic vessels (→). Cisterna chyli (⇒) and thoracic duct (→). Note the remarkable absence of hepatic venous clearance of lipiodol. Repeat study in post-treatment cases of hepatic cirrhosis with satisfactory recovery, demonstrated clearance of lipiodol more by hepatic veins with poor or absence of delineation of liver lymphatics.

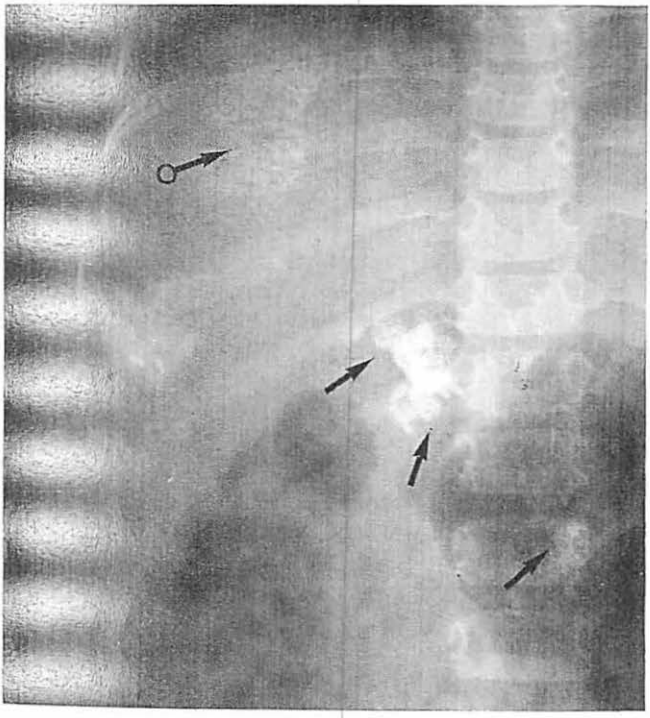

Fig. 4. 24 hour skiagram of the same case showing portal (↑) and para-aortic (↗) nodes.

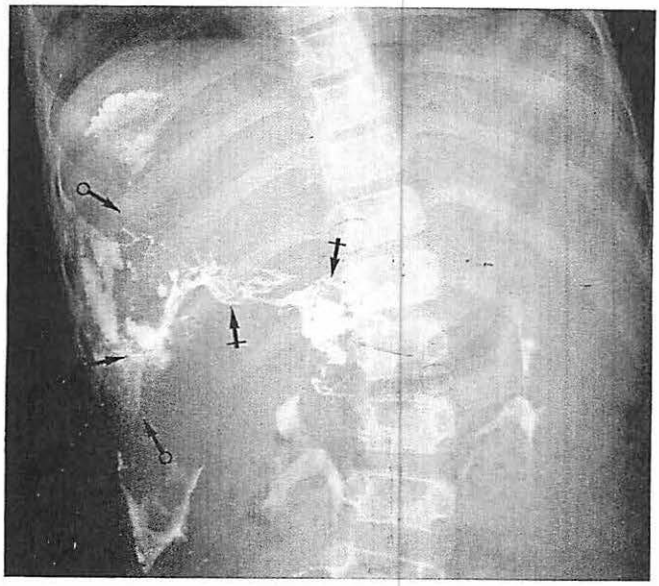

Fig. 5

Fig. 5, 6, 7. Eleven year old boy, a case of biopsy proved tuberculous cervical and axillary lymphadenopathy partially responding to anti-tuberculous therapy, reported with heapto-splenomegaly and jaundice.

Hepatic lymphography using lipiodol ultra fluid (Fig. 5) shows more liver lymphatics (→) than that obtained by functional hepatography (c.f. Fig. No. 1). 24 hours skiagram (Fig. No. 6) shows lymph nodes in the portal hepatis (↑) and para-aortic regions (↑) with lymphoma pattern. Hodgkin's disease was subsequently confirmed by histology of the infraclavicular lymph nodes that were growing in size while under antituberculous therapy. Liver biopsy revealed evidence of hepatic cirrhosis though the clinical and lymphographic impression was Hodgkin's infiltration of liver.

Repeat study (Fig. 7) after satisfactory response to cyclophosphamide therapy, showed intensification of regressed lymph nodes of drainage (↕) (↑) and flow of lipiodol mostly via the venous pathways (→ ○→).

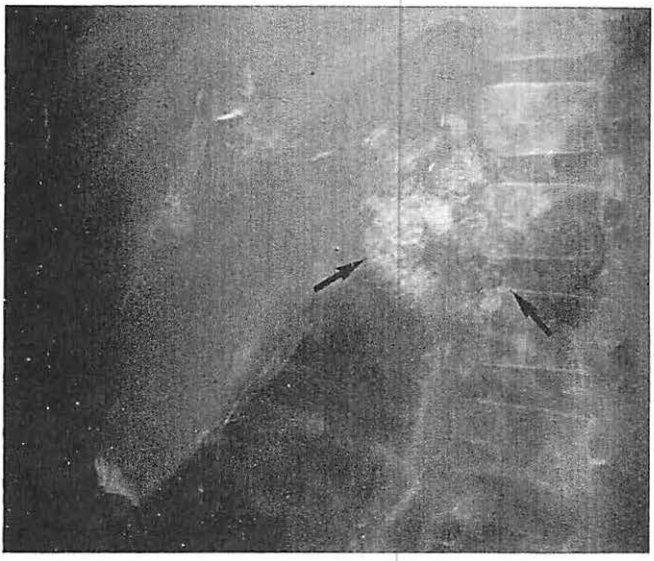

Fig. 6

Even though previous workers have recommended that liver biopsy and intraparenchymal injection studies could be done at the same sitting, we prefer to perform liver biopsy subsequent to the completion of intra-parenchymal injection studies so as to obtain histological confirmation or otherwise of our interpretation on hepatic lymphographic studies.

Such a procedure also excludes the possibility of lipiodol leak from the liver via previous biopsy tract and distortion of liver architecture. Another advantage would be that biopsy could be obtained under fluoroscopic control from a suspicious area indicated as a filling defect in the hepatographic phase of hepatic lymphography.

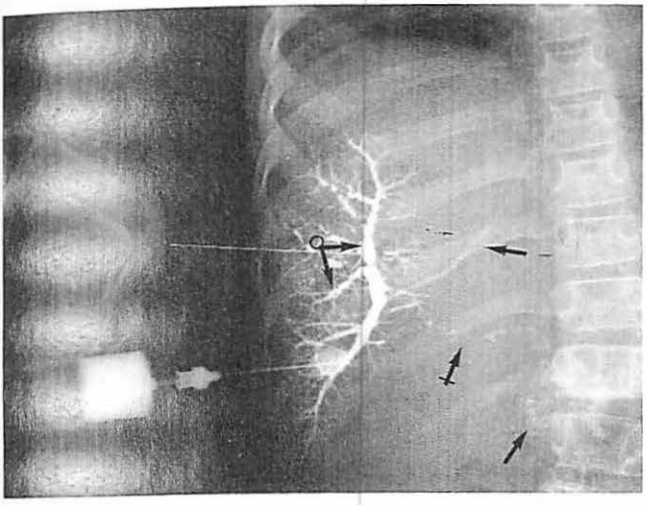

Fig. 7

The third pathway of liver lymph from the bare area of liver via trans-diaphragmatic and pleural lymphatics draining into the mediastinal lymph nodes has been demonstrated in a case of metastatic carcinoma of the liver (Fig. 8). In this case lymphatic drainage towards the porta could not be demonstrated.

Conclusions

Hepatic lymphography is a simple and safe procedure for the study of lymphatic dynamics of liver and the lymph nodes of drainage in the porta hepatis, paraaortic and mediastinal regions. Lipiodol ultrafluid provides better information and detail than the more diffusible aqueous contrast media.

Addendum

Subsequent to the preparation of this paper, we have gained further experience by studying seventy four patients by this method. Our findings in hepatic cirrhosis, hepatic amoebiasis and lepromatous leprosy with granulomatous lesions in the liver as well as hepatic venous and inferior venacaval occlusive pathology will be reported shortly.

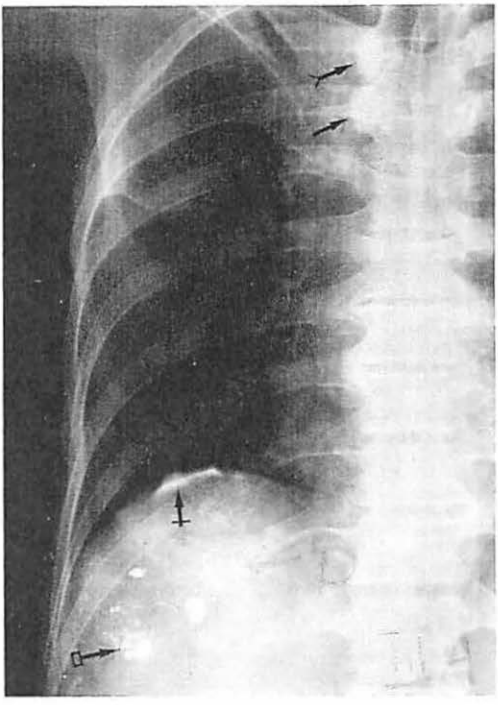

Fig. 8. A case of gastric carcinoma with extensive metastasis showing lymphatic drainage from the bare area of the liver via trans-diaphragmatic and pleural lymphatics into the mediastinal nodes as a result of impediment by metastatic disease to the flow of hepatic lymph via hepatic veins as well as lymphatics normally coursing towards the porta.

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