THE VALUE OF ANGIOGRAPHIC METHODS IN DIAGNOSTIC ASSESSMENT OF LIVER DAMAGE IN PORTAL HYPERTENSION*

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The severity of such liver damage as may induce portal hypertension, and the changes which the organism may undergo when conditions arise where portal hypertension is brought about by pre- or posthepatic obstructions, fully justify the efforts aimed at assessing the seriousness of hepatic damage from vascular alterations which may occur within the gland under such conditions.

The accuracy and reliability of the assessment will be further enhanced if integrated with functional evaluations, blood pressure readings, histological findings and a study aimed at identifying those clinical symptoms which may indicate a portal plethora. Angiograms obtained by arteriography, phlebography or portal angiography may thus be of value, not only in diagnosing the character and degree of portal hypertension, but also in a prognostic evaluation of the condition, at least in so far as they provide a clue to the existence and magnitude of vascular changes.

In the light of the foregoing considerations, a survey was made of the hepatic angiograms of 400 subjects suffering or suspected to be suffering from portal hypertension, as observed during the past 10 years at the Milan University's General Surgery Clinic. Their splenoportograms, hepatic vein phlebograms or hepatic arteriograms were constantly correlated with their clinical and functional data, and occasionally with histopathological and surgical findings, and even with postmortem plastic liver models, in an attempt to determine (*i*) how reliable an indication of the damage suffered by the liver was provided by every individual method and (*ii*) how every individual angiographic feature related to a different type of portal hypertension.

HEPATIC DAMAGE ASSESSMENT THROUGH ARTERIOGRAPHY

Hepatic arteriography is unlikely to prove particularly valuable in evidencing such vascular changes as may indicate liver damage associated with portal hypertension from an intrahepatic obstruction, because the relative skimpiness of the arterial network as compared with the far richer portal and hepatic vascularizations makes the arteriograms so obtainable relatively insignificant.[1,2] The latter may nevertheless be of considerable value when they show a reduction of the vascular bed which is associated with atrophy of the parenchyma—particularly if a comparison may be established with previous arteriograms showing the development of the arterial bed at the time of a hypertrophic condition of the gland.

These findings were confirmed by the morphological changes exhibited by the hepatic artery in plastic models based on postmortem specimens from subjects who had suffered from conditions entailing various forms of atrophy and hypertrophy of the parenchyma. No hepatic arteriogram, moreover, ever showed such arteriovenous anastomoses as might have evidenced a 'splicing' of the arterial and portal vascular systems, thereby confirming the views of those who deny the existence of such anastomoses.

The latter finding was further investigated in our research project by determining the oxyhaemoglobin saturation levels in the supradiaphragmatic inferior cava and hepatic veins of over 30 cases of portal hypertension induced by intrahepatic obstructions. In 8 of these subjects the values were found to be slightly higher than those of mixed venous blood, while in all other instances they turned out to be either normal or definitely lower.

Quite opposite to the results reported above were the findings associated with a particular kind of portal hypertension—that induced by biliary cirrhosis. Our research, confined as it was to comparative investigations of the arterial and portal systems of postmortem livers (with the help of histological examinations and plastic models) invariably showed an increased arterial bed and the presence of intrasystemic (inter-arterial) and intersystemic (artero-portal) anastomoses (Fig. 1). Relative as the value of these

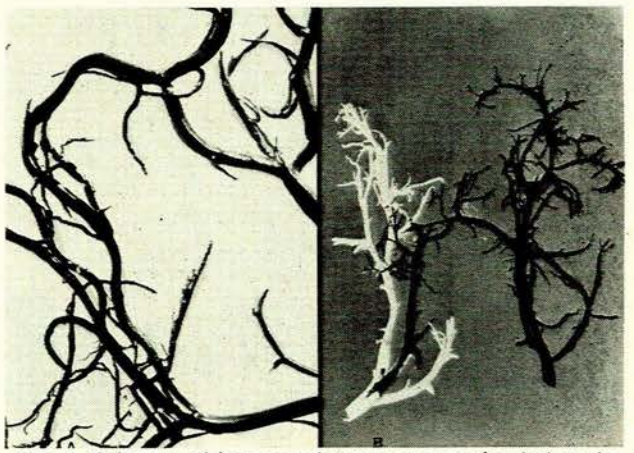

Fig. 1. Evidence of inter-arterial anastomoses in cholestatic biliary cirrhosis (arterial system black, portal system white —10× magnification—plastic models from postmortem liver specimens). *Left*: Full-channel inter-arterial anastomoses. *Right*: Presinusoidal artero-portal anastomoses.

findings may be, they do nevertheless suggest a couple of questions, viz.:

(*a*) Is arterial hypertrophy real, i.e. does it entail an actual numerical increase, or is it merely a compensatory adjustment to hypertrophy of the parenchyma?

(*b*) Are arterial hypertrophy and arteriovenous anastomoses peculiar to biliary cirrhosis alone, or are they a more general expression of liver damage induced by presinusoidal portal hypertension resulting from the pathological processes in the porto-biliary space whenever this condition is present?

The answers to these questions would be of the greatest practical value as they would explain the discrepancy now existing between findings of different workers and would bring additional evidence to substantiate the occurrence of a particular kind of hepatic damage—the damage caused by presinusoidal portal hypertension. Although we know

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of no arteriograms obtained under other conditions of portal hypertension induced by pre- or posthepatic obstructions, we feel justified in venturing the suggestion that the arterial vascular patterns that can be detected by this method are likely to appear entirely normal even when the above conditions are present.

HEPATIC DAMAGE ASSESSMENT THROUGH PORTAL PHLEBOGRAPHY

Unlike hepatic arteriography, portal phlebography proved to be so valuable in assessing liver damage associated with the various types of portal hypertension that the method aroused some genuine enthusiasm when it was first adopted in clinical practice. Indeed, though obtained indirectly, new light on the alterations of hepatic vascular morphology under various portal hypertensive conditions was being cast by vascular exploration of the hepatic portal system. Experience, however, showed the inadequacy of this method as a means of hepatic damage assessment, due to the slowed-down portal flow determining a further dilution of the contrast medium, to the point where the latter can no longer provide a conclusive picture of intrahepatic portal distribution. For the same reasons, the parenchymatographic images become fuzzy, and deviations are often induced by organic obstructions or haemodynamic interferences.

Our experience shows that while the angiograms are normal-looking during the initial stages of the condition, in the more advanced, extended forms the vascular upset affects the whole intrahepatic portion of the vena porta, to the point where modal distribution may disappear altogether in cases of portal hypertension caused by post-necrotic cicatricial sclerosis. The most significant is the capillary stage, where stagnation of contrast medium in the various districts results in either honeycomb patterns or uneven mottling. Significant as these pictures were, they never provided us with grounds for a less vague diagnosis than that of liver damage in any of the various forms of portal hypertension induced by intrahepatic obstructions which we investigated, although strikingly obvious alterations of the portal district were invariably evidenced by the plastic models. No assessment of liver damage by this method was ever possible when the condition arose from pre- or post-hepatic obstructions.

LIVER DAMAGE ASSESSMENT THROUGH HEPATIC VEIN PHLEBOGRAPHY

Information which can be obtained by exploring the hepatic venous system would afford instead an accurate determination of the most significant aspects of liver damage in the various forms of portal hypertension.

Whenever the condition arises from a prehepatic obstruction, phlebograms will show an enormously developed collateral circulation which becomes even more obvious in the initial stages, accompanied by hypertrophy of the liver, and likewise associated with portal hypertension induced by a presinusoidal intrahepatic obstruction, as in cases of postnecrotic cirrhosis, biliary cirrhosis or schistosomiasis. One wonders whether portal hypertension due to a prehepatic obstruction may be diagnosed on the strength of the presence of collateral circulation patterns alone. In our experience these patterns are invariably apparent whenever a prehepatic obstruction exists, irrespective of its location.

What is the significance which these collateral circulation processes may have from the standpoint of hepatic damage? Collateral circulation is invariably present in hepatic hypertrophy, whether confined to one lobe alone or extending to the whole liver, and irrespective of its aetiology. Secondly, collateral circulation is often (at least in all forms of presinusoidal obstructions) of the intersystemic kind (i.e. between hepatic veins only). It was evidenced, on a more general level, in portal hypertension caused by an intrahepatic obstruction and complicated by digestive haemorrhage. It could be consistently detected in postmortem casts of the livers of all patients who had been suffering from portal hypertension of the latter type. All such findings would indicate that collateral circulation is meant to make up for the functional impairment caused by the obstruction, and would only occur when the liver was hypertrophied and a relatively moderate hepatic damage was present.

Phlebograms showing liver damage resulting from

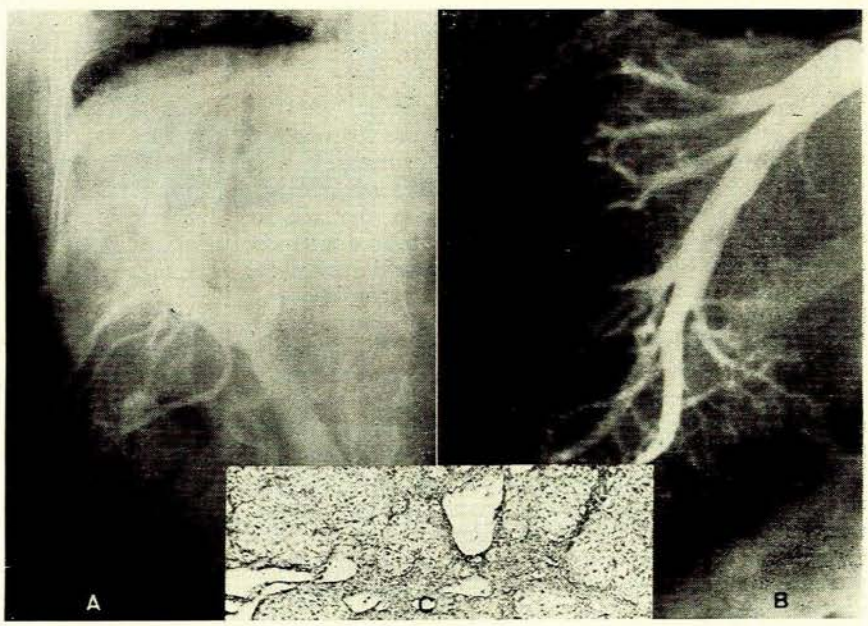

Fig. 2. Portal phlebogram in a case of portal hypertension induced by an intrahepatic obstruction. A: Uneven distribution of 2nd- and 3rd-order branches. Note relatively insignificant parenchymatographic phase. B: Hepatic phlebogram from same subject, featuring cut-off 2nd-order branches and an irregular, uneven, mottled parenchymatographic phase. C: Histological findings from operative biopsy. Note obvious signs of hepatic regeneration and sclerosis.

portal hypertension caused by intrahepatic obstructions, instead, have the greatest story-telling value and are *per se* sufficient to provide early evidence of a vascular alteration when its location is postsinusoidal, for hepatic phlebography is focused on just this district, which is primarily affected in all such degenerative liver processes as tend to evolve towards fibrosis and sclerosis (and are, by the way, the most frequently encountered).

Not only, therefore, will phlebography evidence the severity of liver damage as indicated by alterations affecting the veins and their distribution branches up to the postsinusoidal level, but it may also provide an early clue to the existence of such damage even when no definite clinical or functional symptoms would suggest it, in complete accordance with histopathological findings. Alterations of the above kind would show up in a phlebogram as uneven or suddenly interrupted vasal patterns, or as a thinning-out of the larger veins and/or of 2nd- and 3rd-order branches (Fig. 2). In the parenchymatographic stage, the pattern will appear finely or coarsely irregular due to the existence of intraparenchymatous voids and 'fillings', being the morphological projections of macro- or micronodular cirrhosis which will accurately reflect the corresponding histological features (see Fig. 4). The parenchymatographic stage of the phlebogram is indeed the one that may often show the presence of an intersystemic collateral circulation, which in such cases should be construed in terms of venoportal reflux patterns.

A thorough investigation of such an angiographic expression has led to the following conclusions:

(*a*) Alteration of the suprahepatic area occur earlier than those of the portal system.

(*b*) Alterations associated with the suprahepatic capillary stage are more marked than those of the portal system (Fig. 3).

(*c*) In our study, venoportal refluxes have more frequently found a clinical correlation in patients suffering from digestive haemorrhage than in those with ascites.

(*d*) While venoportal refluxes were fairly regularly found in established forms of portal hypertension caused by biliary cirrhosis, the absence of such refluxes, and particularly a total lack of intrasystemic collateral circulation, found a constant clinical correspondence in ascitic patients suffering from portal hypertension.

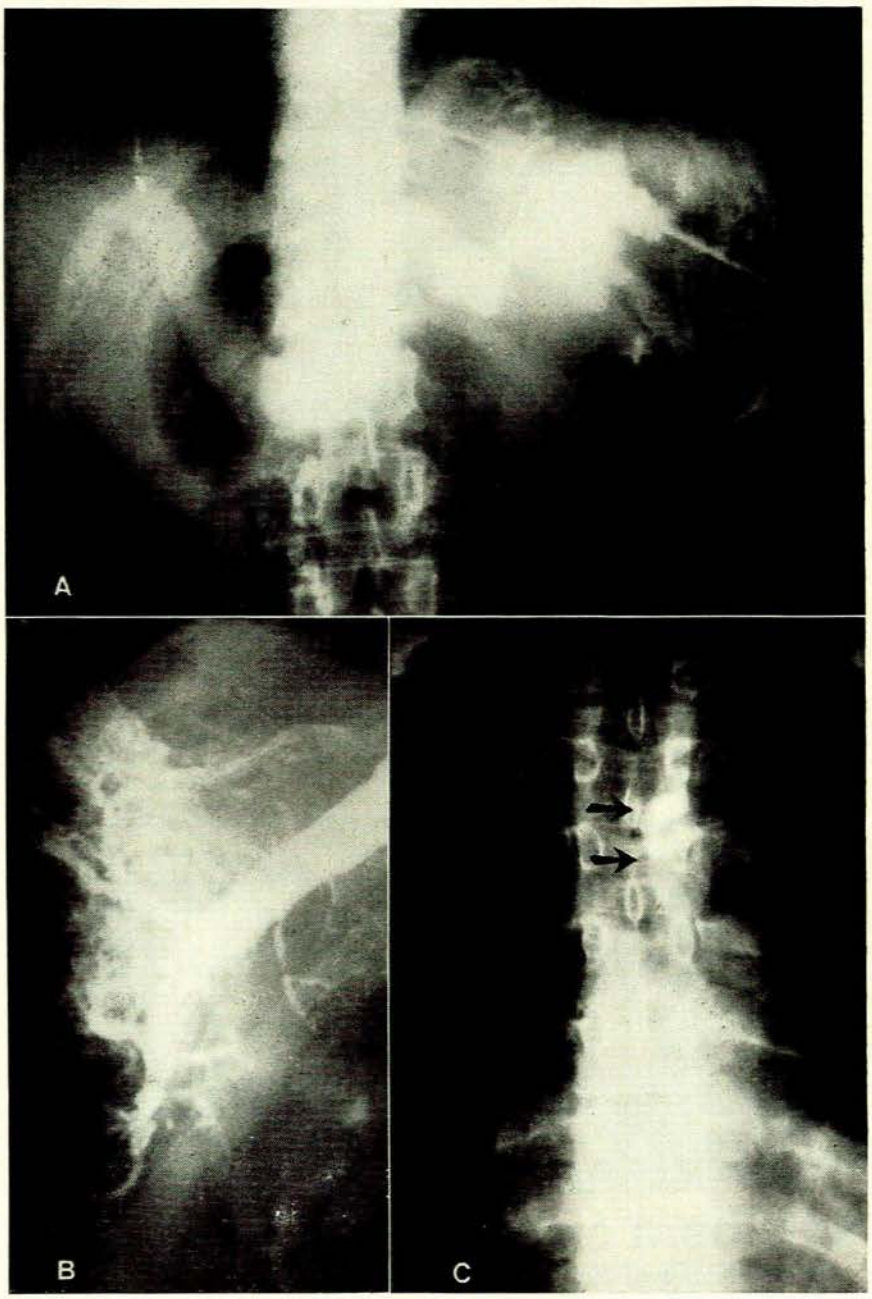

Fig. 3. Portal hypertension from a postsinusoidal intrahepatic obstruction (ascitogenous cirrhosis of the liver). A: Portal phlebogram providing inadequate visualization of intrahepatic vasal distribution. B: Hepatic vein phlebogram: a typical picture of postsinusoidal cirrhosis. C: Lymphangiogram evidencing a dilated thoracic duct at venous confluent level after 60 minutes.

The last finding appears to be rather significant in so far as it may provide a further explanation of such pathogenetic processes as may lead to ascitic effusion in portal

hypertension—which effusion would, according to our findings, occur only when no compensatory circulation was provided by intervenous anastomoses. Such a view appears to be further substantiated by a particular kind of hepatic damage from posthepatic portal hypertension, such as cardiac cirrhosis where ascites occurs in spite of the extension and magnitude of suprahepatic venous system dilation caused by stagnation and where the compensatory circulation pattern previously described could never be evidenced in our research project.

HEPATIC DAMAGE ASSESSMENT THROUGH LYMPHANGIOGRAPHY

Clinical application of lymphangiography in 12 patients suffering from portal hypertension with ascites made it possible to ascertain a slowed-down flow-rate as shown by the sluggish progress of the contrast medium resulting in a markedly delayed thoracic duct visualization in all cases considered, and to evidence a dilated thoracic duct in 9 of our cases (Fig. 3).

These findings, which agreed with those of other workers,[3] have come to cast further, if indirect, light on the problem of hepatic damage assessment as relating to the presence of ascites.

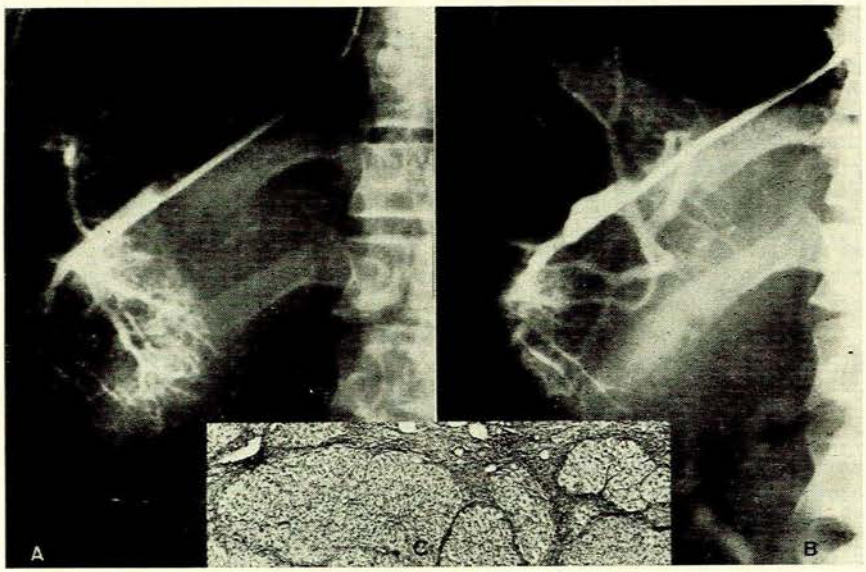

Fig. 4. Hepatic vein phlebogram. Portal hypertension with ascites from a post-sinusoidal intrahepatic obstruction. A: Uneven 2nd- and 3rd-order branch patterns and 'honeycomb' parenchymatographic phase. B: Example of venoportal reflux. Note slighter portal branch alterations. C: Histological findings. Regenerative-type cirrhosis.

CONCLUSIONS

Phlebographic assessment of hepatic damage, irrespective of haemodynamic findings, provides an indication as to which type of portacaval anastomosis may prove most appropriate for surgical treatment of portal hypertension in each individual instance. More particularly, the existence of intervenous anastomoses would suggest a termino-lateral kind of splicing, whereas the latero-lateral solution would be preferable if no such anastomoses were present.

Likewise, the phlebographic image of a dilated thoracic duct would suggest the advisability of resorting to a lymphovenous shunt to allay a portal plethora and to treat ascites.

SUMMARY

An investigation of over 400 subjects suffering from recognized or presumed portal hypertension was carried out with the help of vascular semeiotic tests commonly used in the Milan University Surgical Clinic (splenoportography, phlebography of suprahepatic veins, selective hepatic arteriography and angiolymphography) and with such additional information as could be obtained from haematochemical determinations and the histopathological data provided by hepatic biopsy.

Suprahepatic phlebography appears to be the only method for assessing the amount of damage suffered by the liver. Selective arteriography and splenoportography proved inadequate when it came to identifying structural liver changes in all kinds of portal hypertension.

Phlebography of the suprahepatic venous system will also reveal any kind of liver damage that may subsequently develop into a cirrhosis, as the method supplies valuable information on the finer morphology of venous roots.

The use of all angiographic tests currently performed on patients with portal hypertension also proves extremely valuable when it comes to determining whether or not surgery is indicated, and which portal-systemic shunt should be selected.

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