Histometrical Studies of the Spleen in Banti’s Syndrome
with Reference to Clinicopathologic Correlations*

By

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Histometrical determinations of volume ratio of splenic sinus, the mean breadth of splenic cords and the mean sectional area of sinuses were described. The above quantities were estimated of spleens in Banti’s syndrome in comparison to other conditions of the organ. Banti spleens could be characterized by small values of the mean sinus sectional area. The total sinus length was increased. The condition corresponded to sinus hyperplasia. Distinct correlations were confirmed between the mean sectional area of splenic sinus and hepatic lesions, portal pressure, splenic weight, esophageal varices, leukocyte and platelet counts of peripheral blood. The development of Banti spleens was discussed on the basis of the histometrical results.

Various problems concerning Banti’s syndrome still remain unsolved in spite of a large number of reports in the past. In the author’s Department, attempts have been made to clarify the concept of Banti’s syndrome. It was revealed that the splenic factors were of great importance in the pathogenesis of this syndrome.1-8 The present study deals with the quantitative changes of several splenic structural elements. Histometrical treatments in combination with clinicopathological correlations are expected to contribute to elucidate the pathogenesis of Banti’s syndrome.

MATERIALS AND METHODS

Thirty cases which underwent splenectomy under the diagnosis of Banti’s syndrome are the objects of the present investigation. As the control group, 16 cases were selected for comparative studies, consisting of eight cases of gastric ulcer or cancer without portal hypertension or any hepatic disorder, four cases of pancreatic cancer or pancreatic cyst with involvement of portal system and four cases of liver cirrhosis.

For the purpose of histological examinations, three sections, each measuring

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* This paper is the 9th report of studies on Banti’s syndrome from the Department.

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more than $2 \times 1$ cm, were randomly taken from different portions of spleens. Each section was fixed in formalin, embedded in paraffin and was stained after Gomori’s silver impregnation method. Histologic examination of the liver was performed following the method described by Takahashi$^9$ of the author’s Department. Portal pressure was recorded in the following way: a catheter was inserted in a branch of mesenteric vein until its tip reached the trunk of portal vein, connected to a L-shaped glass tube with a scale and the pressure was read and expressed in mm $H_2O$.

**HISTOMETRICAL METHODS AND RESULTS**

A. Histometrical treatments of splenic structural elements

1. *Volume ratio of sinus*

Histological sections of spleens stained for reticulin fibers were examined with a microscope equipped with an eye piece of the type as in Fig. 1 under high magnification. The sinus-splenic cord boundaries was clearly demonstrated by reticulin fiber architecture of splenic cords. The length of linear intercepts delivered by intersection of sinus and splenic cord with the horizontal line of the eye piece, $s$ and $p$, respectively, was successively measured and registered, while the histological slides were continuously shifted in the direction of the horizontal line of the eye piece. Several hundreds of intercepts were thus deter-

Fig. 1. Linear intercepts are delivered by intersection of the horizontal line of the scale with sinus-splenic cord boundaries.
mined on a number of randomly selected microscopical fields. Blood vessels, lymph follicles and trabeculae were excluded from the measurement. As splenic tissue just beneath splenic capsules was of different structural principles, samples were taken only from splenic tissues at least 1 mm apart from splenic capsules. The sum of the length of intercepts given by splenic cords was designated as $\Sigma p$ and that of sinuses as $\Sigma s$. The volume of sinus $Vs$ in a unit volume of splenic tissue is then obtained from:

$$V_s = \frac{\Sigma s}{\Sigma p + \Sigma s}.$$  

In the present study, $Vs$ was expressed in per cent and employed as the estimate for the volume ratio of splenic sinus.

The results of the determination were:

i) The volume ratio of the sinus in the eight cases of gastric lesions ranged from $40.8\%$ to $47.0\%$ with the average of $42.7\%$.

![Fig. 2. Volume ratio of the sinus ($Vs$) in various disease groups.](image-url)
ii) The volume ratio in the four cases of pancreatic diseases was between 40.9% and 52.4% with the average of 45.2%.

iii) As to the four cases of liver cirrhosis, the volume ratio was between 39.4% and 60.5% with the average of 50.6%.

iv) As to the thirty cases of Banti's syndrome, the volume ratio ranged from 29.1% to 54.7% with the average of 42.9%.

These results did not indicate any significant difference of the sinus volume ratio in the examined spleens (Fig. 2).

2. Mean breadth of splenic cords

The mean breadth of splenic cord $D$ is defined as the thickness, when the total volume of splenic cords in a certain sufficiently large splenic volume is supposed to be uniformly extended between two equal planes, each of which has a surface area equal to a half of the total surface area $S$ of sinus-splenic cord boundaries in the same volume. For the determination of $D$, accordingly, an estimation of the total surface area of sinus walls $S$ in a certain splenic volume is required. It is now assumed that the direction of sinus walls is sufficiently randomized and the density of splenic cords is uniform in the space. We take under these conditions a cube with edges of $l$ in length in splenic tissue. When the direction of sinus walls is randomized in the cube, they can be separated into an infinitely large number of small pieces of equal size and displaced within the cube to form an arbitrary number of spheres of equal radii, without changing their total surface area. When the number of spheres is $n$, and their radii are $r$, we obtain:

$$4 \pi r^2 n = S$$

(1)

by definition.

We suppose now that the sphere group in the cube is sampled by a straight line vertical to one of the faces of the cube. The possible position of the sampling line is only restricted by the extension of the face of the cube and in other respect completely undetermined. The procedure is naturally reproduced by drawing a straight line across a square with edges of $l$ in length on a histological slide taken out of the cube as representing an arbitrary plane parallel to one of the faces of the cube.

The expected value $E$ for any one of these spheres to intersect with the sampling line is then given by dividing the surface area $\nu$ of the equatorial plane of a sphere by the surface area $l^2$ of a face of the cube as:

$$E = \frac{\nu}{l^2} = \frac{\pi r^2}{l^2}$$

and that for the total sphere group as:
\[ nE = \frac{n \nu}{l^3} = \frac{n \pi r^2}{l^2}. \] (2)

If the count of intersecting points is designated as \( C \), \( C \) must be equal to \( 2nE \), because every sphere delivers two crossing points with an intersecting straight line. Consequently, we obtain from (1) and (2):

\[ \frac{C}{2} = \frac{S}{4l^2} \]

and

\[ S = 2C l^2. \]

The total surface area \( S_0 \) of sinus-splenic cord boundaries in a unit splenic volume is then given by:

\[ S_0 = \frac{S}{l^2} = \frac{2C}{l}. \]

On the other hand, the total volume of splenic cords \( V_p \) in a unit splenic volume is determined by:

\[ V_p = \frac{\Sigma p}{\Sigma p + \Sigma s}. \]

Accordingly,

\[ \bar{D} = \frac{2V_p}{S_0} = \frac{V_p l}{C}. \]

Fig. 3. A cube with edges of \( l \) in length is taken in the red pulp and sinus and splenic cords in the cube are regarded to be randomized in their directions. The sinus walls are supposed to be divided into an infinite number of small squares (f). \( S \): sinus, \( P \): splenic cord, \( SL \): sampling line, \( C \): intersections of the sampling line with sinus walls.
The results are:

i) In the eight cases of gastric lesions, the mean width of the splenic cords ranged between 16.1µ and 20.8µ with the average of 18.8µ. The normal value was approximately in the range from 15 to 22µ.

ii) As to the four cases of pancreatic diseases, the value ranged from 10.3µ to 19.1µ with the average of 16.0µ.

iii) As to the four cases of liver cirrhosis, the minimum value was 10.1µ and the maximum 17.4µ with the average of 13.5µ.

iv) In the 30 cases of Banti’s syndrome, the value ranged between 7.6µ and 37.9µ with the average of 14.4µ. Out of the cases, 70% had values lower than 15µ and in only two cases (6.7%) the estimates exceeded 22µ.

It is interesting that in contrast to the group of gastric lesions, in which all the cases had values for $D$ between 15 and 22µ, 37.5% of the cases of stagnant spleens and 70% of the cases with Banti’s syndrome had the values lower than 15µ.

3. Mean sectional area of sinus

The mean sectional area of splenic sinuses is defined as follows. Splenic sinuses are considered to be bent tubes oriented in randomized directions in splenic tissue. The transverse sections of the tubes are assumed to be of such shapes which make it possible to define a single center in each cross section in some way. The loci of the center constitute continuous curves with randomized direction in the space. The total length of the curves in a cube out of splenic
Fig. 5. The breadth of splenic cord ($\bar{D}$) in various disease groups.

tissue with edges of $l$ in length is designated as $L$. If the total sinus volume in a unit splenic volume is $V_s$ the mean sectional area of sinus $S_s$ is defined as:

$$S_s = \frac{V_s P}{L}.$$

Accordingly, a preliminary estimation of the total length of linear structures with randomized directions in a certain volume is necessary to determine $S_s$.

We take a cube with edges $l$ in length. The linear structures in the cube are now supposed to be divided into an infinitely large number $n$ of small fractions of equal length $\eta$. Each fraction can under this condition be regarded to be an infinitesimally small fraction of a straight line. When the direction of the linear structures is randomized in the space, we can displace the fractions without changing their directions to a certain point $O$ in the cube, so that the middle point
of each fraction is situated on \( O \) and all the fractions assembled in this way fill up a sphere of the radius \( \eta/2 \) with uniform density in reference to every direction. We suppose now that the fractions are sampled by a plane parallel to one of the faces of the cube. The possible range of the sampling place is restricted by the length \( l \) of an edge of the cube. The effective length of a fraction in the direction vertical to the sampling plane is given by \( \eta \sin \theta \), if \( \theta \) is the angle of inclination of the fraction to the sampling plane. Consequently, the expected value \( E \) for a single fraction to intersect with the sampling plane is given by \( (\eta \sin \theta)/l \). The number of fractions \( dn_\theta \) of which angles of inclination are between \( \theta \) and \( \theta + d\theta \) is given by dividing the surface area of the belt on the surface of one of the hemispheres made by fractions which satisfy the above condition by the total surface area of the hemisphere. Consequently, we obtain:

\[
d n_\theta = n \cdot 2 \pi (\eta/2) \cos \theta \cdot d\theta/2 \pi (\eta/2)^2 \]

\[
= n \cdot \cos \theta \cdot d\theta.
\]

The expected value \( dC'\_\theta \) for all the fractions of the above range to intersect with the sampling plane is the product of \( E \) and \( dn_\theta \), and we obtain:

\[
d C'\_\theta = E \cdot d n_\theta \]

\[
= n \cdot \eta \sin \theta \cos \theta \cdot d\theta/l \]

\[
= L \cdot \sin \theta \cos \theta \cdot d\theta/l.
\]

If \( dC'\_\theta \) is integrated from \( \theta = 0 \) to \( \theta = \pi/2 \) the result \( C' \) represents the expected value for all the fractions of every possible direction to intersect with the sampling plane. This means at the same time, that \( C' \) is equal to the count of crossing points of the original linear structures on a sampling plane with the surface area \( l^2 \). The integration is:

\[
C' = \frac{L}{l} \int_0^{\pi/2} \sin \theta \cos \theta \cdot d\theta
\]

\[
= \frac{L}{l} \cdot \frac{1}{2} [\sin^2 \theta]_0^{\pi/2}
\]

\[
= \frac{L}{2l}
\]

or

\[
L = 2 C' \cdot l.
\]

The total length of the linear structures in a unit volume is then

\[
L_0 = \frac{2 C' \cdot l}{\beta^2} = \frac{2 C'}{\beta^2}.
\]
From the above results, we finally obtain:

\[ S_S = \frac{V_S}{L_0} = \frac{V_S l^2}{2 C'} . \]

Fig. 6. Sinus sections on a certain surface area of a histological section are counted by the aid of an eye piece of this type.

Fig. 7. A cube with edges \( l \) in length is taken in red pulp and sampling plane \((SP)\) is placed parallel to a face of the cube. Number of intersecting points between the sampling place \((SP)\) and the center of the sinus is expressed as \( C' \). \( P \): splenic cord. \( L \): total length of the sinus in the cube.
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Fig. 8. Fractions, divided in an infinitely large number \( n \), and displaced toward a point \( O \), fill a sphere of \( \eta/2 \) in radius with uniform density.

### Table I. Results of Measurement in the Group of Gastric and Pancreatic Lesions and Liver Cirrhosis

<table>
<thead>
<tr>
<th>Gastric Lesions</th>
<th>Pancreatic lesions with obstruction of portal vein</th>
<th>Liver cirrhosis</th>
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<tbody>
<tr>
<td><strong>Case</strong></td>
<td><strong>Age</strong></td>
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Tables I and II show the total length of the sinuses $L_0$ in a unit volume of splenic tissues. The results of the determination of $S_S$ demonstrated:

i) The mean sectional area of the sinus in the eight cases of gastric lesions ranged between $515.9\mu^2$ and $783.8\mu^2$ with the average of $610.7\mu^2$.

ii) In the 4 cases of pancreatic lesions with involvements of portal veins, the value ranged between $409.2\mu^2$ and $712.0\mu^2$, with the average of $532.4\mu^2$.

iii) In the 4 cases of liver cirrhosis, the mean sinus sectional area was between $496.5\mu^2$ and $919.2\mu^2$ with the average of $713.0\mu^2$.

iv) In the 30 cases with Banti’s syndrome, the mean sinus sectional area ranged from $133.8\mu^2$ to $764.6\mu^2$ with the average of $354.2\mu^2$. Out of the total cases, 23.3% had values between 500 and $800\mu^2$ and in 76.7% they were lower than 500 $\mu^2$.

It was thus revealed, that Banti spleens were characterized by small sinus sectional area as a whole.
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Fig. 9. Mean sectional area of sinus ($S_s$) in various disease groups.

CORRELATIONS OF HISTOMETRICAL RESULTS TO OTHER ANATOMICAL FINDINGS AND CLINICAL SYMPTOMS

1. Correlation to the histologic patterns of the spleen

Kakizaki\(^7\) classified the spleen in Banti's syndrome into 4 types according to reticulin fiber architecture of splenic cords. The classification could be correlated to portal pressure and other histological findings of the spleen. In the present study, the author examined Kakizaki's classification in reference to the histometrical results. There was no distinct correlation between the volume ratio of the sinus and the histologic pattern of the spleen. With the advancement of the histologic patterns from I to IV, however, there was a tendency of decrease in the mean breadth of splenic cords and the mean sectional area of the sinus (Figs. 10 and 11).
2. Correlation of portal pressure

There was only indistinct correlation between the volume ratio of the sinus and portal pressure. In regard to the mean breadth of splenic cords, it tended to decrease with the enhancement of portal pressure, although the coefficient of correlation -0.32 failed to attain statistical significance at 5% level. On the other hand, the mean sectional area of the sinus showed, as demonstrated in Fig. 12, a definite tendency of decrease with rising portal pressure, indicating a significant negative correlation at 1% level with a coefficient of correlation of -0.59.
Fig. 12. Correlation between portal pressure and the mean sectional area of the sinus ($S_s$), $r = -0.59$ ($p < 0.01$).

Fig. 13. Correlation between splenic weight and the mean sectional area of the sinus ($S_s$), $r = -0.39$ ($p < 0.05$).
3. Correlation to splenic weight

Splenic weight varies according to ages. In cases of splenomegaly, the weight ratio to the normal spleen was calculated in reference to the normal splenic weight reported by Amano\textsuperscript{11} and the relation to the reticulin fiber architecture of splenic cords was studied.

As a result, there was no correlation among splenic weight, volume ratio of the sinus and the mean breadth of splenic cords. However, there was a negative correlation between splenic weight and the mean sectional area of the sinus significant at 5\% level with a coefficient of correlation \textasciitilde -0.39 (Fig. 13).

4. Correlation to the histologic patterns of the liver

Takahashi\textsuperscript{9} in the author’s department classified the histological features of the liver in Banti’s syndrome into three types. In Type 1, there is no noticeable

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\textbf{Fig. 14.} Relation between the mean breadth of splenic cord ($\bar{D}$) and histologic patterns of the liver in Banti’s syndrome.
Hepatic change. Type II has chronic inflammation or fibrosis of Glisson’s sheaths. Type III comprised the cases with liver cirrhosis.

The author subclassified the Type II into Type IIa and Type IIb, the former having cell infiltration in Glisson’s sheaths and the latter exhibiting fibrosis of the structures. The mean breadth of the splenic cords became smaller with the progress of pathological changes of the liver (Fig. 14). The mean sectional area of the sinus showed a tendency of decrease with advancing hepatic injuries (Fig. 15).

5. Correlation to esophageal varices

Both the volume ratio of the sinus and the mean breadth of splenic cords failed to show any relation to esophageal varices. On the other hand, clinical
evidences of esophageal varices were more frequently confirmed in the group with lower sectional area of splenic sinus. In cases with normal mean sectional sinus area, little evidence of esophageal varices was noticed (Fig. 16).

6. Correlation to blood cell counts

The counts of white blood cell, red blood cell and platelet, which are essentially influenced by splenectomy in Banti's syndrome, were examined in connection with histometrical results.

a) White blood cell (WBC).

There was no significant correlation between the mean breadth of splenic cords and WBC. The correlation between the volume ratio of the sinus and WBC was significant at 1% level with a coefficient of correlation +0.48, and the decrease in volume ratio was approximately in proportion to the decrease of WBC.

Fig. 16. Relation between the mean sectional area of sinus ($S_s$) and esophageal varices.
Fig. 17. Correlation between volume ratio of sinus ($V_s$) and WBC, $r=+0.48$ ($p<0.01$).

Fig. 18. Correlation between the mean sectional area of sinus ($S_s$) and WBC, $r=+0.69$ ($p<0.001$).
Fig. 19. Correlation between the mean sectional area of sinus ($S_s$) and platelet count, $r=+0.39$ ($p<0.05$).

(Fig. 17). Between the mean sectional sinus area and WBC distinct correlation, significant at 1% level, was observed with a coefficient of correlation of +0.69.
b) Red blood cell (RBC).
No distinct correlation was confirmed between RBC and histometrical results.
c) Platelet count.
Significant correlation at 5% level was noted between the mean sectional sinus area and the platelet count with a coefficient of correlation of +3.9 (Fig. 19).

These results suggest that in Banti's syndrome there are certain correlations between histological changes of the spleen and blood cells.

DISCUSSION

Since Banti$^{12}$ in 1894 defined the so-called Banti's disease as an independent disease entity and sought its histological characteristics in fibroadenia of splenic follicles, the pathogenesis of the disease has been a problem of many controversies. Recent investigations indicated that portal hypertension was one of the most important factors in the development of Banti spleens.$^{13-15}$ By means of histometrical studies, Abe$^{16}$ demonstrated that obliteration of small intrahepatic portal veins could be correlated to portal pressure readings at surgical operations. The result indicated that in Banti's syndrome elevation of portal pressure was
induced in the majority of cases by intrahepatic fibrosis of portal area. Histological patterns of the liver are variable. They are sometimes those of well-defined liver cirrhosis, but more frequently they are represented by relatively slight portal fibrosis with distinct involvement of portal veins.

The influence of portal hypertension on the spleen can be histologically recognized, because splenic tissues exhibit characteristic aspects which are generally called sinus hyperplasia. Splenic pulp is composed of a large number of small sinus-like structures and the configuration of splenic cords is remarkably obscured. Sinus hyperplasia is regarded to be a more important and reliable histological characteristic of Banti spleens than follicular fibrosis, because the former is a more direct expression of portal hypertension, while the latter is of more or less non-specific character. In this respect, sinus hyperplasia in Banti spleens deserves more detailed examinations. In the present study, attempts were made to define sinus hyperplasia histometrically and to obtain unbiased clinicopathological correlations.

Although there is little doubt about the rôle of portal hypertension in inducing histological patterns of Banti spleens, no appreciable sinus hyperplasia is caused by elevated portal pressure acting on normal spleens. In cases with obstruction of portal veins by thrombosis or tumors, no distinct splenomegaly with sinus hyperplasia is observed. This indicates there must be some preceding pathological process in splenic tissue in order that portal hypertension induces sinus hyperplasia.

Transformations in splenic structures are most conveniently visualized by analysis of reticulin fiber architecture of splenic cords. Observation on the reticulin fiber architecture of the spleen was first made by Oppel in 1891 and Matsui reported a similar investigation in 1911. The most detailed and systematic observation of the normal reticulin fiber architecture of splenic cord was probably that of Koboth in 1939. However, these reports were confined to studies of normal spleens and an analysis of pathological processes of splenic cords by means of reticulin fiber architecture was not hitherto attempted. Moreover, these observations were entirely based upon rather qualitative findings of splenic structures, and effective and reliable quantitative treatments became possible only after integral geometry was introduced into histometrical methods.

According to Suwa, splenic cords undergo a series of structural changes in splenitic reaction, which are distinctly recognized in their reticulin fiber structures. Proliferating reticulum cells in splenic cords causes remarkable distension of splenic cords. Reticulin fibers produced by reticulum cells gradually fix splenic cords in structures quite different from those in normal spleens. The total splenitic process can consequently be designated as pulp hyperplasia from anatomical viewpoints. Kakizaki assumed that hyperplastic splenic cords were progressively subdivided into small sinus-like structures when splenitic process was accompanied by remarkable portal hypertension. He sought the development
of histological patterns of Banti spleens in this process and classified Banti spleens into 4 groups according to the advancement of sinus hyperplasia.

The author's own investigation revealed a distinct negative correlation between portal pressure and the mean sinus sectional area. The sinuses in Banti spleens are narrower than those in normal spleens, but their total length is evidently much increased. On the other hand, no definite correlation was confirmed between portal pressure and volume ratio of sinus. The total volume of splenic cords is more or less preserved in spite of progressive increase of sinus-like structures. These results indicate that sinus hyperplasia is not caused by dilatation of preexisting sinuses, but represents proliferation of small sinus-like structures out of hyperplastic splenic cords. The present investigation substantiated the assumption of Sato and others,1-8 that Banti spleen develops through sinus hyperplasia by way of subdivision of splenitic and hyperplastic splenic cords. Venous stagnation acting upon normal spleens does not cause any distinct sinus hyperplasia, and preceding hyperplasia of splenic cords on account of splenitis is regarded to be indispensable for proliferation of small sinus-like structures. The introduction of histometrical methods into analysis of splenic structures is expected to demonstrate structural changes of the spleen in more reliable and exact forms than in previous reports based on histological observations.

It is generally acknowledged that splenectomy alleviates hematological disturbances in Banti syndrome.21-31 This is one of the grounds which support the view that clinical symptoms in Banti syndrome are induced by some splenic factors. Banti12 himself anticipated the presence of some toxin of splenic origin. Welch32 and others attributed hematological symptoms in Banti's syndrome to a certain hormone-like substance released in excess from enlarged spleen. Abnormally enhanced blood cell destruction in the spleen33 or production of some autoimmune antibodies34 are also regarded to belong to possible causative agents of the so-called splenic anemia. Tomoda35 proposed the concept of splenic toxicosis in a similar sense. These investigators all sought splenic activity in the reticuloendothelial system of the organ. The present investigation revealed a negative correlation between the mean sinus sectional area and WBC or platelet count of peripheral blood, although RBC could not be correlated to the structural change of the spleen. The results make participation of reticulum cells in the reduction of cellular elements of blood rather improbable, because in spleens with advanced sinus hyperplasia endothelial cells are distinctly predominant in number over reticulum cells. It is required further to study hematological disturbances in Banti's syndrome in close association with the structural changes of the spleen.

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References

3) Sato, T., Watanabe, K., Miya, M. & Ottomo, M. *ibid.*, 1963, 80, 254.
4) Sato, T., Watanabe, T., Saito, Y. & Seki, K. *ibid.*, 1961, 81, 1.
7) Kakizaki, G. *ibid.*, 1964, 82, 387.
10) Suwa, N., Personal communication.