Banti’s Syndrome without Portal Hypertension

Toshio Sato, Yasuo Suda, Goro Kakizaki and Hidemi Yamauchi

Department of Surgery (Prof. T. Maki),
Tohoku University School of Medicine, Sendai

The authors picked up eight particular cases from a series of so-called Banti’s syndrome which they had experienced at their surgical department. Although these cases had most of clinical manifestations of this syndrome, they were contrasted with typical cases because of their normal portal pressure. Histologically, spleens of these cases showed pulp hyperplasia of various grades but, unlike in typical cases of this syndrome, no evidence of sinus hyperplasia. Besides, changes in lymph follicles of spleens of these cases included increase in their population, appearance of a germinal center and hemorrhage in perifollicular areas. This paper describes the details of these findings and presents the authors’ view that these eight cases represent an incipient phase of Banti’s syndrome. Discussions are also made in this paper on the relation between Banti’s syndrome in its early phase and so-called primary hypersplenism.

Despite a large number of contributions devoted to Banti’s syndrome since Banti first described it as a disease entity, its nature and pathogenesis are still in considerable dispute. We also have studied the syndrome in a series of patients admitted to our surgical department, with special interest in its pathophysiology, hepatic and splenic histology and their changes after splenectomy. Included in this series were eight particular cases that arrested our attention because they had discernible enlargement of the spleen as well as clinical manifestations of splenic hyperactivity, but no evidence of portal hypertension. In expectation that these cases might represent an incipient phase of Banti’s syndrome and thus might give a clue to elucidation of the syndrome, the cases have been thoroughly re-investigated. This paper describes the results and presents our tentative view on the pathogenesis of this syndrome.

MATERIALS AND METHODS

The objectives of this study were the above-mentioned eight cases among the total of 80 patients of Banti’s syndrome operated on at our surgical department. The hepatic histology was examined by hematoxylin-eosin stain and elastica-Goldner stain, and the splenic histology by hematoxylin-eosin stain with silver

Received for publication, November 16, 1965.
impregnation, and elastica-Goldner stain. Portal pressure was measured directly through a catheter inserted at laparotomy into the portal trunk via the superior mesenteric vein.

On the other hand, the behavior of pulp cords and lymph follicles of the spleen in Banti's syndrome was systematically studied in all the 80 cases including the eight cases in question, to locate the position which the latter cases occupy on the line of development of this syndrome.

RESULTS

I. Clinical and Laboratory Findings

Clinical and laboratory findings in the eight cases are summarized in Tables 1 and 2. The age and sex distributions showed no noteworthy difference from those in other patients with this syndrome. In their past histories, those indicative of infectious or allergic conditions were identifiable in four cases but not in the other four. Complaints of the patients were in most instances probable manifesta-

<table>
<thead>
<tr>
<th>Case</th>
<th>Age &amp; sex</th>
<th>Chief complaints</th>
<th>Past history*</th>
<th>Splenic enlargement†</th>
<th>Hepatic enlargement†</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>33 M</td>
<td>Epigastric discomfort</td>
<td>None</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>52 F</td>
<td>General weakness and tinnitus</td>
<td>Sepsis (44), splenomegaly (15), cholelithiasis (10), nephritis (1)</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>30 M</td>
<td>Epigastric discomfort</td>
<td>None</td>
<td>1.5</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>42 F</td>
<td>Abdominal discomfort and general weakness</td>
<td>Acute nephritis (10) with chronic sequela</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>32 F</td>
<td>Abdominal distention and hemorrhagic diathesis</td>
<td>Diphtheria (18), pneumonia and rheumatoid arthritis (5), hepatitis (3), pulmonary the. (1)</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>44 F</td>
<td>General weakness and loss of weight</td>
<td>None</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>23 M</td>
<td>General weakness and splenomegaly</td>
<td>None</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>8</td>
<td>15 M</td>
<td>Pallor</td>
<td>Jaundice (2)</td>
<td>2.5</td>
<td>2</td>
</tr>
</tbody>
</table>

* Numeral in parentheses indicates time of the episode, in years before admission.
† Maximum vertical measure of palpable area below the costal margin, in fingerbreadth.
Banti's Syndrome without Portal Hypertension

<table>
<thead>
<tr>
<th>Case</th>
<th>Red cells</th>
<th>White cells</th>
<th>Platelets</th>
<th>Bone marrow inhibition</th>
<th>Liver functions</th>
<th>Esophageal varices</th>
<th>Portal pressure (mmH₂O)</th>
<th>Weight of spleen (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3,920,000</td>
<td>5,700</td>
<td>245,000</td>
<td>+</td>
<td>Normal</td>
<td>-</td>
<td>170</td>
<td>346</td>
</tr>
<tr>
<td>2</td>
<td>1,500,000</td>
<td>2,000</td>
<td>53,000</td>
<td>+</td>
<td>Normal</td>
<td>-</td>
<td>140</td>
<td>380</td>
</tr>
<tr>
<td>3</td>
<td>3,950,000</td>
<td>3,000</td>
<td>168,000</td>
<td>+</td>
<td>Normal</td>
<td>-</td>
<td>110</td>
<td>430</td>
</tr>
<tr>
<td>4</td>
<td>3,830,000</td>
<td>5,400</td>
<td>193,000</td>
<td>?</td>
<td>Normal</td>
<td>-</td>
<td>130</td>
<td>500</td>
</tr>
<tr>
<td>5</td>
<td>3,770,000</td>
<td>8,000</td>
<td>162,000</td>
<td>?</td>
<td>Impaired</td>
<td>?</td>
<td>175</td>
<td>502</td>
</tr>
<tr>
<td>6</td>
<td>3,290,000</td>
<td>4,300</td>
<td>?</td>
<td>?</td>
<td>Normal</td>
<td>-</td>
<td>200</td>
<td>680</td>
</tr>
<tr>
<td>7</td>
<td>4,350,000</td>
<td>6,300</td>
<td>326,000</td>
<td>+</td>
<td>Normal</td>
<td>-</td>
<td>220</td>
<td>705</td>
</tr>
<tr>
<td>8</td>
<td>3,300,000</td>
<td>4,100</td>
<td>102,000</td>
<td>+</td>
<td>Normal</td>
<td>-</td>
<td>130</td>
<td>1,100</td>
</tr>
</tbody>
</table>

Anemia of various grade was present, the lowest red cell count recorded in this series being 1,500,000 per cubic millimeter. Leucopenia was also common and all but one (Case 3) showed deviation to the left. Cases 3 and 4 had increased relative eosinophil counts, 8.0 per cent and 13.5 per cent, respectively. The platelet count was less than 200,000 per cubic millimeter in five of the seven examined cases. In all the six cases examined, the bone marrow revealed impaired maturity of blood cells. Liver functions (icteric index, B.S.P., colloidal reactions) were normal except for Case 6. In all the cases, roentgenographic evidence for esophageal varices was absent, and translial portography revealed no particular findings such as obliteration and collateral circulation.

The portal pressure as measured on laparotomy fell within the normal range. The weight of extirpated spleens ranged between 346 g and 1,100 g, the average being 580 g. Thus the grade of splenomegaly in this series of patients proved to be moderate for this syndrome.

In summary, the eight cases had most clinical and laboratory features of Banti's syndrome, such as enlargement of the spleen, decrease in number of one or more kinds of peripheral blood cells and impairment of hematopoiesis, but were different from typical cases of this syndrome in that they did not show an elevation of portal pressure.

II. Histology of the Spleen and Liver

1) Spleen

**Sinuses**

Venous sinuses in these eight cases were normal in their size and red cell content. The reticuloendothelial cells that lined the sinuses showed no qualitative nor quantitative changes.
Pulp cords

Pulp cords were the site of various histologic changes in these cases. As described by Koboth and recently confirmed by Kakizaki of our Department, pulp cords of the normal spleen have an architecture of regularly arranged reticulin fibers which are visualized by silver impregnation; a twist of 'Grenzfasern' forms the axis of each cord and segments of 'Mantelmaschenfasern' connect it to 'Ringfasern' that surround venous sinuses. The changes in the reticulin fiber arrangement appearing in these eight cases were classified into four patterns: Disorderly pattern (Fig. 1) was given by moderate, rather simple irregularity in the architecture of Grenzfasern and Mantelmaschenfasern, and such was observed in Cases 1, 2 and 7. Similar conditions were also recognized in spleens of control cases (patients with stomach diseases without any evidence for Banti's syndrome), and this pattern of splenic change is considered to be non-specific. Fine-dissociative pattern (Fig. 2) was characterized by loss of the normal structure of Grenzfasern and dissociation of the latter into fine fibers. Inasmuch as typical examples of such a change were occasionally encountered in acute infectious conditions, this pattern may represent relatively early change of the pulp cord. Thick-sclerotic pattern (Fig. 3) in which Grenzfasern and Mantelmaschenfasern were abnormally thickened and formed coarse meshwork is interpreted to be chronic sequence of the disorderly and the fine-dissociative patterns. Such a pattern was observed in Cases 3 and 8 which were associated with low portal pressure. Proliferative pattern (Fig. 4), observed in Case 6, was marked by strong tendency of reticulin fibers toward proliferation; the pulp cord was dilated and therein were irregularly distributed thick, proliferated fibers. Although the reticulin fiber architecture of pulp cords was thus quite various, a common feature could be pointed out in these eight cases that division of the pulp cord into sinus-like structures, which is characteristic to typical Banti spleens and known as sinus hyperplasia, was never observed.

The quantitative change of reticulum cells of the pulp cord ran approximately parallel with that of reticulin fibers. These cells, as well as endothelial cells of sinuses, appeared to have little phagocytic activity, although the study was not based on supervital stain. Hemosiderosis was usually confined to sinus endothelia, but in Case 5 it was also observed in pulp cords and perivascular areas.

Lymph follicles

In Cases 1, 5, 6, 7 and 8, lymph follicles of the spleen were often larger than normal and more than half of them had germinal centers. Hyperemia and/or hemorrhage of considerable grade were common in perifollicular areas of these cases. In Cases 2, 3 and 4 in which enlargement of the spleen was slight and portal pressure was comparatively lower, on the other hand, lymph follicles were normal in size and they were rarely associated with germinal centers or with discernible changes in the perifollicular areas.
Trabeculae

Case 6 revealed shattered lesions of splenic trabeculae, probably the same change that Dürr\textsuperscript{14} referred to as ‘Aufsplitterung’. Such a finding was not observed in the other cases.

2) Liver

Histology of the liver which was examined by biopsy at laparotomy was normal in two cases (Cases 3 and 6). But other three cases (Cases 1, 5 and 7) had cellular infiltration of the Glisson capsules, and fibrosis was observed in the remaining three (Cases 2, 4 and 8). None of the cases showed typical cirrhosis. The obliteration ratio of intrahepatic portal branches was below 30 per cent in all but one of the cases; the exceptional one (Case 6) had a value of 34 per cent. Thus, the ratio in these cases was only slightly higher than in control cases in which it was usually less than 25 per cent as reported previously.\textsuperscript{7}

III. Characteristics of Pulp Cords and Follicles of the Spleen in Banti’s Syndrome

On the basis of the type and grade of changes occurring in the architecture of reticulin fibers, Kakizaki\textsuperscript{11} of our Department previously classified the splenic histology in Banti’s syndrome into four basic types, Types 1—IV. When this classification was applied to the total of 80 cases of Banti’s syndrome we had experienced the present eight cases and exclusively those belonged to Type I. Thus, the eight cases in question may be termed as the cases with Type 1 splenic histology. The frequencies of the other types were: Type II, 26; Type III, 34; and Type IV, 12. With reference to this classification, this chapter systematizes the changes of pulp cords and lymph follicles in Banti’s syndrome on the basis of the above-mentioned 80 cases.

Pulp cords

v. Herrath\textsuperscript{15} estimated the normal spleen to be composed of 78 per cent red pulp, 18 per cent white pulp and 5 per cent capsule and trabeculae. In Banti’s syndrome, on the other hand, Seki\textsuperscript{12} of our Department found that venous sinuses, proper or newly formed, constitute 40–50 per cent of the splenic tissue regardless of its histologic type, the remaining 50–60 per cent being pulp cords and other elements. On such bases and taking into consideration the average weight of the spleen for each histologic type, Fig. 5 was prepared schematically to indicate the composition of the Banti spleen in its various histologic types.

Changes of pulp cords hold the most important part in development of the Banti spleen and we can differentiate in time two principal patterns; one is rather simple proliferation known as pulp hyperplasia, and the other is its transformation into sinus-like structures, so-called sinus hyperplasia. The proportion of pulp vs. sinus hyperplasias is various, and this proportion served as the criterion in Kakizaki’s classification mentioned above — only pulp hyperplasia is observed.
in Type I, both pulp and sinus hyperplasia exists but the former is predominant in Type II, sinus hyperplasia predominates in Type III, and almost exclusive sinus hyperplasia is seen in Type IV. Now, it may be suggested from Fig. 5, especially from continuity of the curves, that the above-mentioned Types I, II, III and IV directly indicate the stage of development of the Banti spleen, thus implying that the Type I histology corresponds to the incipient phase and the Type IV histology to the ultimate phase of this syndrome. Fibrosis in trabeculae, perivascular areas and perifollicular areas was not significant in Type I, but it made a considerable component of the Banti spleen in the other types.
As shown in Figs. 6 and 7, the populations of plasma cells and lymphocytes in the pulp cord were the largest in Type I and decreased as the histologic type changed to II, III and IV.

**Lymph follicles**

The number of lymph follicles per unit area of the histologic section was the
greatest in Type I and decreased in the descending order of Types II, III and IV, as shown in Fig. 8. The average size of the follicles showed little difference between the histologic types, the mean diameter being 512μ in Type I, 480μ in Type II, 510μ in Type III and 445μ in Type IV.

As shown in Fig. 9, the incidence of the germinal center and that of perifollicular hemorrhage were both the highest in Type I and the lowest in Type IV.

Fig. 9. Incidence of perifollicular hemorrhage and germinal center in the Banti spleen.

- ■: Frequency of cases in which more than a third of lymph follicles of the spleen had perifollicular hemorrhage.
- ○: Frequency of cases in which more than a third of lymph follicles of the spleen had a germinal center.

Fig. 10. Incidence of fibrosis of perifollicular areas and periarterial areas in the Banti spleen.

■ (+)  ○ (+)  (-)
On the other hand, as seen in Fig. 10, the incidence of fibrosis in perifollicular areas and in perivascular areas showed a reversed relation, being the highest in Type IV and lowest in Type I.

**Splenic histology and portal pressure**

Relation between the histologic type of the spleen and the portal pressure is shown in Table 3. The pressure was mostly in the normal range in the Type I cases, as mentioned before, but was usually higher than normal in the other cases and tended to increase with progress of the histologic types. An implication of this fact may be that when the spleen with Type I histology is influenced by an increase in portal pressure, sinus-like transformation of proliferated pulp cords is initiated and the splenic histology is converted to Type II, Type III and eventually to Type IV. In Figs. 11 and 12 examples of the Type II histology are shown. Sporadic foci of sinus hyperplasia in proliferated and dilated pulp cords suggest transition from Type I to Type II, apparently supporting the above hypothesis.

**DISCUSSION**

Our series of Banti’s syndrome included a few particular cases, eight out of a total of 80, in which portal pressure was within the normal range unlike in typical cases, although they had other clinical manifestations of the syndrome such as splenomegaly, decrease in one or more formed elements of peripheral blood and impairment of bone marrow hematopoiesis. Histologically, the reticulin fiber architecture of the spleen was quite different in these cases from that of typical cases of Banti’s syndrome; Grenzfasern and Mantelmaschenfasern of the pulp cord showed various changes ranging from mere disarrangement to remarkable proliferation, but their division into sinus-like structures, so-called sinus hyperplasia, was never observed. Such a finding was classified by Kakizaki of our laboratory as Type I and contrasted with Types II, III and IV observed in typical Banti’s syndrome. In these cases, lymph follicles were generally larger than normal and often had germinal centers, and perifollicular areas were frequently associated with hyperemia and/or hemorrhage but rarely

---

**Table 3. Relation between splenic histology and portal pressure**

<table>
<thead>
<tr>
<th>Splenic histology</th>
<th>No. of cases</th>
<th>Portal pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Range</td>
<td>Average</td>
</tr>
<tr>
<td>Type I</td>
<td>8</td>
<td>110–220</td>
</tr>
<tr>
<td>II</td>
<td>26</td>
<td>180–470</td>
</tr>
<tr>
<td>III</td>
<td>34</td>
<td>180–480</td>
</tr>
<tr>
<td>IV</td>
<td>12</td>
<td>250–550</td>
</tr>
</tbody>
</table>
with fibrosis. Furthermore, the population of free cells, such as plasma cells and lymphocytes, in the pulp cord was significantly larger in the Type I cases than in other Types. All these facts suggest that the Type I cases have more active, rather acute pathologic process of the spleen than in other Types. On such histologic and biometric bases, the evidence was presented in this paper to show that the Type I histology represents an incipient phase of Banti’s syndrome and typical Banti spleens that are associated with the Types II, III and IV histologies develop on the basis of such a condition.

Two fundamental problems are aroused here concerning the above presumption; these are (1) what is the cause of the Type I histopathology that is considered to be an incipient phase of the Banti spleen, and (2) what factors transform the Type I histology into Types II, III and IV, i.e., into typical Banti spleens. As regards the first problem, some authors assert that an increase of portal pressure may be a cause of such changes of the spleen that we described as the Type I histology. Moschowitz, for instance, found widening of splenic pulp cords and their fibrosis in the case of acute or subacute thrombosis of the portal vein, associated with moderate increase of portal pressure, and concluded that the changes of the spleen were due to increased portal pressure. McMichael described a syndrome, ‘hepatolienal fibrosis’, in which hepatitis and splenomegaly coexisted; the splenic histology in this syndrome was characterized by fibrosis following hemorrhage of perivascular and perifollicular areas, and he ascribed such changes to increase of portal pressure due to pylephlebitic process. In the eight cases of our series, however, although histology of the spleen resembled those described by the above authors, portal pressure was in the normal range. Therefore, at least in these cases, increase of portal pressure could not be the cause of the splenic change.

On the other hand, in histologic examination of the eight cases, we could point out a number of findings that were suggestive of splenic response to noxious stimuli — increase in number and size of lymph follicles, appearance of germinal centers, hyperemia and/or hemorrhage in perifollicular areas, increased population of free cells in pulp cords, etc. It has been suggested by Hueck that the pattern of mesodermal reaction of the spleen to exogenous stimuli varies according to the kind of stimuli; for example, reticulin fibers of the sinus system chiefly react in the case of portal hypertension, but reticulin fibers which proliferate in chronic sepsis are usually those little related to venous sinuses. At any event, it is believed that proliferative process in pulp cords and in follicles follows various stimuli to the spleen, particularly those related to its circulatory disturbances. Ono emphasized arterial hyperemia as a common picture in the Banti spleen, and Yonekawa subsequently observed in follicles of the Banti spleen remarkable budding of arterioles in distal segments of central arteries. Furthermore, as reported earlier, the spleen responds to an infectious stimulus, on
cessation of an acute stage, with remarkable proliferation of reticulin fibers in
dilated pulp cords, i.e., with pulp hyperplasia. On the basis of our own observa-
tions as well as the above-mentioned facts, we conclude that the splenic
histopathology in question falls within the same category as splenitis.

Concerning the second problem, we would emphasize increase of portal pres-
sure as the factor which triggers transformation of the Type I histology into
Types II, III and IV, in other words as the factor which stimulates progress of
Banti's syndrome from its incipient phase to intermediate and ultimate phases.
This may be justified from our observation that portal pressure, which, as mention-
ed above, was within the normal range in Type I cases, became higher in parallel
with progress of the histologic type and was the highest in Type IV. Thus we
believe that when increased portal pressure exerts its effect to the spleen which
has been prepared by some mechanism to have the Type I histology, prolifera-
ted pulp cords are gradually divided into sinus-like structures and the typical Banti
spleen, characterized by sinus hyperplasia, develops. It is in this sense that
we consider the Type I histology as representing an incipient phase of Banti's
syndrome.

As reported previously, we take obliteration of intrahepatic portal branches
as the factor responsible for increase of portal pressure in the above-mentioned
process. Banti described the hepatic condition in this syndrome as cirrhosis,
but this was later corrected by Mitamura who differentiated it from true cir-
rhosis as pseudocirrhosis. Furthermore, recent studies have revealed that there
are occasions on which the hepatic change is so mild that it is more properly
termed as fibrosis rather than as pseudocirrhosis, and also such occasions that
the change consists chiefly of cellular infiltration in the Glisson capsules. As
shown in Table 4, cellular infiltration of the Glisson capsules and/or fibrosis
predominated in our own series of Banti's syndrome. Examination of this series

<table>
<thead>
<tr>
<th>Splenic histology</th>
<th>Type</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatic histology</td>
<td>I</td>
<td>II</td>
</tr>
<tr>
<td>Minimal changes</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Fibrosis and/or</td>
<td>6</td>
<td>17</td>
</tr>
<tr>
<td>cellular infiltration</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pseudocirrhosis</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>8</td>
<td>21</td>
</tr>
</tbody>
</table>
* In 75 cases with known hepatic histology. Numeral in parentheses is the number of cases with extrahepatic obliteration of the portal vessel.
revealed that the grade of increase of portal pressure was significantly correlated with the grade of obliteration of intrahepatic portal branches, apparently supporting the above-mentioned view. Although most of the eight Type I cases showed histologically perceptible hepatic changes (cf. Table 4), they were moderate in grade and the obliteration ratio of intrahepatic portal branches was almost comparable to the control value. This fact coincided with the observation that portal pressure was within the normal range in these cases.

It may be appropriate to discuss here as to whether Banti's syndrome in its incipient phase, such as represented by the eight reported cases, is different from a number of hematological diseases which Doan and his collaborators categorized as primary hypersplenism — congenital hemolytic jaundice, primary splenic neutropenia, idiopathic thrombocytopenic purpura, etc. According to the latter authors, these conditions should be distinguished from Banti's syndrome in that they do not show portal hypertension. However, this must be re-evaluated because Banti's syndrome is not necessarily associated with increased portal pressure in its incipient phase of development, as revealed in this paper. Inasmuch as primary hypersplenism is a collective concept for a variety of conditions with different clinical manifestations and probably with different pathogenesis, the discussion will be made on each of the above-mentioned individual conditions.

Congenital hemolytic jaundice, to begin with, is a rather peculiar member of primary hepersplenism. The histology of the spleen exhibits quite a specific pattern for remarkable blood stagnation in pulp cords and resulting compression of venous sinuses. Moreover, spherocytosis is occasionally seen in red corpuscles of peripheral blood. Because of these specific features observed in congenital hemolytic jaundice, further investigations may be required before obtaining a definite conclusion on the identity between this condition and incipient Banti's syndrome.

Primary splenic neutropenia and pancytopenia attract special attention since hematological data in the eight cases of early Banti's syndrome was comparable to some extent to these cases, although Doan and Wright, and Wiseman and Doan distinguished them from Banti's syndrome for lack of portal hypertension and hepatic involvement. As to the splenic pathophysiology in these conditions, the above authors insisted on increased phagocytic activities of reticuloendothelial cells, but histologic evidence was not well documented in their papers. Damashek and Estren reported the cases with non-specific fibrosis of the spleen. On the basis of a thorough histologic study of the spleen in 11 cases of splenic neutropenia and 10 of pancytopenia, v. Haam and Awny emphasized moderate or marked widening of pulp cords, due to proliferation of reticuloendothelial cells, and hypertrophy of lymph follicles as characteristics in these conditions. These findings appear to resemble what we found in the
eight Type I cases of Banti's syndrome, and we are thus convinced that primary splenic neutropenia and splenic pancytopenia are within the same category as Banti's syndrome in its incipient phase.

Felty's syndrome which was described in 1927 by Felty as a complex of splenomegaly, leucopenia and arthritis is one of the conditions in close relation to primary hypersplenism. Although suspecting that such a condition might represent a particular type of early Banti's syndrome, the original author distinguished them with respect to concurrent arthritis. However, as may be suggested from the fact that many investigators consider a mesodermal reaction such as allergy and collagen disease as the most likely etiologic factor for Banti's syndrome, association of arthritis with Banti's syndrome is not a peculiar phenomenon. In fact, past history of rheumatoid arthritis was confirmable in a case (Case 5) of our series of early Banti's syndrome, as well as in some of the cases of chronic splenic neutropenia examined by Doan and Wright. Moreover, the splenic histology in the case of Felty's syndrome was reported to show thickening of pulp cords and enlargement of follicles as in early stage of Banti's syndrome. Thus, we have no sure reason to make a clear-cut differentiation between Felty's syndrome and incipient phase of Banti's syndrome.

Idiopathic thrombocytopenic purpura is somewhat specific in its hematological manifestation but usually exhibits almost the same splenic histology as in primary splenic neutropenia and pancytopenia. In a study of 36 cases of this disease, v. Haam and Awny failed to differentiate it from the latter conditions so far as histopathology of the spleen was concerned. Appearance of large germinal centers as well as enlargement of follicles associated with moderate thickening of pulp cords were also pointed out by other authors as characteristics of splenic histology in this disease. Implications of these facts may be that idiopathic thrombocytopenic purpura is a variety of primary hypersplenism in which splenic dysfunction was directed particularly toward decrease of platelets, and that even this disease may belong to the same category as incipient Banti's syndrome. Based on a comparative study of the splenic histology in various conditions, Suwa advocated that idiopathic thrombocytopenic purpura could possibly be transformed to typical Banti's syndrome if a mechanism develop to increase portal pressure.

CONCLUSION

Eight unusual cases of Banti's syndrome were reported. These cases had most characteristics of this syndrome such as chronic splenomegaly, decrease in number of peripheral blood cells and impaired bone marrow hematopoesis, but were differentiated from typical cases in that they had almost normal portal pressure. On the basis of clinical as well as histologic findings, the evidence was presented to suggest that these cases represent an incipient phase of
Banti’s syndrome.

References

20) Yonekawa, A. The patho-histological changes of the Banti’s splenomegaly—particularly three dimensional approach on the structure of the Malpighian bodies *Yonago Igaku Zasshi* (Jap.), 1958, 9, 1088–1114.
Banti's Syndrome without Portal Hypertension


Fig. 1. Disorderly pattern. Case 7. H and E stain with silver impregnation. 10 × 25.

Fig. 2. Fine-dissociative pattern. Case 5. H and E stain with silver impregnation. 10 × 25.
Fig. 3. Thick-sclerotic pattern. Case 2. H and E stain with silver impregnation.
10 × 25.

Fig. 4. Proliferative pattern. Case 6. H and E stain with silver impregnation.
10 × 25. Note remarkable growth of reticuloendothelial cells in dilated pulp cords.
Fig. 11. Type II histology of the Banti spleen observed in a 56-year-old male patient. H and E stain with silver impregnation. 10 × 25. Newly formed sinuses are seen in proliferated pulp cords (arrowed). In this case, portal pressure was 300 mm H$_2$O and weight of the spleen 810 g.

Fig. 12. Type II histology of the Banti spleen observed in a 45-year-old female patient. H and E stain with silver impregnation. 10 × 25. Scattered foci of sinus hyperplasia are recognized in proliferated pulp cords (arrowed). Portal pressure 310 mm H$_2$O and weight of the spleen 670 g.