Regressive Giant Bullous Emphysema in Tuberculosis of Adults*

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In the last two years there have appeared several reports on large multiple bullae which develop in the parenchyma of one or both lungs, and tend to grow or to decrease and eventually disappear.

This type of cavity is seen in tuberculous patients under treatment by antibiotics and/or chemotherapy, particularly in those receiving isoniazid. The relationship between the latter drug and the development of such cavities is so close that most writers attribute the formation of the bullae to the isoniazid although they offer no satisfactory explanation for the mechanism of their production.

This phenomenon however should not specifically be due to isoniazid alone, since there are isolated reports of pseudocystic tuberculosis called pneumatocele in patients treated with streptomycin. Behamou, Levy Valensi and Mimouni (1947), Dufourt, Galy and Perrin (1950), Silverthone and Silverman (1950) and others cited by Dufourt1 have recorded such cases. There is even another report by Pruvost and co-workers2 who describe pseudocystic bilateral multiple cavities developing from an apical cavity but which did not regress. This case was not treated by antibiotics before the appearance of the pseudocysts.

Recently Caffey has published reports on the possibility of the appearance of cysts or cyst like cavities in infants in whom these congenital bullae underwent spontaneous regression.

Furthermore, it is already accepted that pseudocysts may develop after pulmonary abscesses and we have published a case of this occurrence although at that time did not realize that possibility.3

Staphylococcal infections may give rise to multiple cavities in the lungs and the possibility of multiple cavities following the course of miliary tuberculosis is also known.

However, development of multiple large cavities with a trend to rapid enlargement and subsequent regression and disappearance has not been reported until recently. We must admit that these reports coincide with the use of isoniazid in tuberculosis.

We have found 25 cases of bullae with the features mentioned recorded particularly in the European and South American literature. It is rather surprising that so distinctive a disease has not been more frequently reported in the United States.

The reports are those of Jacob and co-workers,4, 5 Rossignol et al,6 Etienne Bernard et al,7 and Jacob, Cartier and Treps8 in France; Pablo Purriel et al,7 in Uruguay; Di Filipo in Italy8 and Altman and Ornstein in the United States.10

The main features of the peculiar disease described by these authors are:

I. It appears in patients with acute, severe, unilateral but most frequently bilateral tuberculosis of the upper lobes, with cavities and multiple foci of demonstrable disease.

II. All cases reported have received the isoniazid alone or associated with streptomycin and para-aminosalicylic acid.

III. They have had a favorable outcome with rapid improvement of general condition, weight gain, disappearance of toxic symptoms, bacteriological conversion and improvement of all laboratory findings.

IV. The roentgenological course has been characterized by a trend to clearance of exudative shadows and caseous deposits, while a variable number of thin-walled cavities becomes increasingly evident. These grow in volume and number, with the appearance of large cysts or cystic groups.

V. Further course of the disease shows a marked tendency of the bullae to decrease in number and size and eventually to disappear but those developed from preexistent cavities usually remain, although they have very thin walls and their aspect becomes more and more cyst-like.

VI. In the few cases where intracavitary pressure has been measured a definite patency of the bronchial channels is proved.

VII. The pleural space was found to be definitely occluded when resection was done or when intrapleural pneumothorax was attempted.

VIII. Lack of the usual pathology of cavities as well as the absence of epithelial lining has been demonstrated in resected specimens: the cavity walls consisted of thin connective tissue.

The case that we wish to present is interesting because it demonstrates objectively this new aspect of the disease and because it represents an extreme example of the regressive bullous lung in tuberculosis.

Case Report

N. S. G., male, 29, married, with irrelevant pathological and family history, suffered in the past only from dysentery. In June 1952, after some indefinite gastrointestinal symptoms, he had cough, emesis, yellowish expectoration and vesperal fever, for which he consulted a physician and had some non-specific treatment. As he did not improve he came to Mexico City where he saw another physician who diagnosed pulmonary tuberculosis of the left upper lobe. The first film at our disposal was
taken on Sept. 4, 1952 (Fig. 1). It is important to point out that the physician gave him a dose of subcutaneous Friedmann vaccine at that time. The patient claims that he obtained some relief but noticed no further improvement after four doses of the same vaccine. A second film was taken on Jan. 2, 1953 (Fig. 2).

Upon close interrogation the patient declared that he took about 300 tablets of 50 miligrs. of rimifon (Total 15 gms.).

Seen at the office on April 1, 1953, he was considered to be critically ill with bilateral tuberculosis. He had marked dyspnea, copious perspiration, slight cyanosis, a hacking cough, a pulse rate of 120 and upon auscultation, diminished breath sounds and a few scattered moist rales. A new film was taken (Fig. 3) in which multiple cavities are observed affecting both lungs with the single exception of the right lower lobe.


On admission this patient was considered beyond recovery.

Pneumoperitoneum, administered in small volumes was well tolerated.

No change was observed immediately after this treatment. Besides, dihydrostreptomycin 1 grm., rimifon 300 mgms., and PAS 12 gms., combined were given daily.

After 20 days he developed severe intestinal atony which required the use of prostigmine and a gastrointestinal tube. The intestines reacted favorably after several days. Pneumoperitoneum was stopped after these symptoms appeared.

On admission the temperature was irregular with a maximum of 39.5 C. It fluctuated around 38.5 C. during the first month, but decreased thereafter while unexpected improvement was observed.

The first tomograms of April 4 1953, are presented in the three figures 4, 5 and 6. In the series of 10 tomograms at that time 14 and 15 bullae could be counted on the right and left lungs respectively.

On May 21 the patient was seized with great dyspnea and cyanosis. The respirations were 50 per minute, the pulse 155, fluoroscopy discovered spontaneous pneumothorax of the right lower lobe without collapse of the upper.

The presence of adhesions prevented complete collapse of the lower lobe and allowed breathing until a catheter could be inserted to aspirate the air. The pressure was plus 3; after aspiration it fell to 0 and returned to plus 3 without effort or cough. The catheter was withdrawn after 10 days without relapse of the spontaneous pneumothorax.

Although bronchography, bronchoscopy, angiography, measurement of the intra-
cavitary pressure, particularly on the left side where a large cavity seemed to be spontaneous pneumothorax were considered, the condition of the patient did not warrant any such procedures during the first weeks.

Slight improvement of bronchoscopy being done, showed merely a congestion of the mucosa and muco-purulent secretion, but the caliber of all bronchi was normal (Aug. 1953).

After further improvement a functional study was done with the following results:

Ventilation at rest: 13.02 Lit./min.
Ventilation per Sq. Meter: 8.6 Lit./min.
Max. volnt. ventilation: 88.0 Lit./min.
Respiratory reserve V.M.V: 85 per cent Minimum Norn: 92 per cent
Vital Capacity: 1,300 c.c.
Brachial artery, rest: 88 per cent (Norm saturation 86-92 per cent)
Brachial artery, exercise: 80 per cent Saturation
Brachial artery with O: 93 per cent Normal: 100 per cent

Cardiac Catheterization: Nov. 25, 1953:
Pulmonary pressure: 42/17 Normal: 30 Systolic.
Angiocardiography showed a good irrigation of the lungs with exception of the left upper lobe (Fig. 7). There is no doubt that the vascularization had much improved and that had an angiogram been done earlier, the results would have been different.
In July 1953 we thought that as in chronic emphysema a possible vascular factor was participating, therefore we gave a tablet of Priscol 3 times a day which later was substituted by 3 tablets (150 mg's. a day) of beta-piridilcarbinol (Roniacol).

Tubercle bacilli were no longer found in the sputum after May 14, 1953 with only two exceptions (one bacillus per 50 and 20 fields respectively).\(^5\) Cultures are still positive from some specimens. Treatment with dihydrostreptomycin was continued until the end of November 1953, but after July 1953 the regimen changed from 1 gm. daily to 1 gm. every two days. Roniacol was continued until the patient left the sanatorium in March 1954.

On dismissal only five cavities could be seen in the right and six in the left lung. The remaining bullae were smaller and a large one which at the beginning was taken for spontaneous pneumothorax, shrank and showed its wall. Also there were some round shadows suggesting small filled cavities (one at right and three at the left lung). Figs. 8, 9, 10, and 11.

FIGURE 7

*According to another laboratory report the tubercle bacilli recovered in cultures were identified as of human type.
Further tomographic checks after two months showed only four well defined cavities on the right and two on the left.

The patient has no dyspnea; his temperature is normal, his respirations are 20; his pulse 80; his weight on admission was 46 Kgs. (101 Lbs.) it is now 62 Kgs. (136 Lbs.). The blood count is as follows: Erythrocytes: 5,470,000; Hemoglobin: 100 per cent; Leukocytes: 11,200; Lymphocytes: 16; Monocytes: 5; Segmented: 70; Stabs: 5; Juva: 0; Myelo: 0; Eosinophiles: 4; Baso: 0; Sedimentation: 18; Hematocrit: 42.

It seems worth mentioning that during the whole regressive process treatment with dihydrostreptomycin, PAS and isoniazid was not interrupted.

Discussion

The case presented has the features of the group of cases presented by others. The mechanism of development of these bullous cavities is a matter of elaborate discussion and is far from cleared.

Some points are to be stressed: First: Although this is a new type of tuberculous disease which seems to appear under the influence of the new drugs it is rare, given the enormous number of patients so treated in the last years. In our personal experience with a great number of patients treated by streptomycin and a large and growing group treated with isoniazid we have not been able to find another case.

It is surprising that so few instances of this disease have been reported in the literature of the whole world.

In the American literature so far we have been able to find only the case published by Altman and Ornstein who state that Auerbach has not found anything like this pathology in 2000 autopsies.

In the French literature we found 18 cases; six are described by Purriel in Uruguay and one by Di Filipo in Italy.

The rarity of this peculiar disease suggests that the circumstances under which it may develop are complex and exceptional and that they rarely coincide.

E. Bernard states that Steenken has produced bullous disease of the lungs in rabbits infected with tuberculosis and treated by isoniazid.

It is pertinent also to discuss whether these bullae must be considered as real tuberculous cavities in the commonly accepted sense; i.e., whether they are preexistent cavities, cleansed of their tuberculous content and

![FIGURE 8](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21258/ on 06/25/2017)

![FIGURE 9](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21258/ on 06/25/2017)
insufflated by the valvular mechanism already known. 

This possibility has been accepted by E. Mayer and Bernou; however it is open to discussion for the bullae appear not only in areas where cavities were previously observed but in those where no cavities or shadows were present.

The theory of preexistent cysts newly insufflated or even defective spaces (E. Mayer and Caffey) cannot be upheld in view of the findings in the resected specimens in which no characteristic lining of preformed cavities or defects has been observed. These findings on the contrary demonstrate that the cavities are newly formed and are lined with connective tissue (Purriel and E. Bernard).

The assertion of Purriel that these spaces represent cleansed necroses the caseous content of which has been expelled through the bronchi (chemical casectomy), partly explains the mechanism but gives no satisfactory explanation for their insufflation for all authors agree that nearly all these cavities are open to the bronchial tree as far as we can tell from the pressure readings, bronchoscopies, and the study of the resected specimens.

We believe that an analysis of the possible factors involved in insufflation should lead us to a satisfactory answer. These are:

1. The bronchial factor. Stenosis, distortion of the bronchi, edema or bronchial occlusion.
2. Parenchymal destruction of lung tissue.
3. The vasculo-nutrient factor in the lung tissue.
5. Mechanical changes developing from structural alterations of the surrounding tissues, and particularly: \textit{Changes in the pleurae}.

Contrary to the common finding in other tuberculous cavities the bronchial factor is practically absent and even in some cases where it has been claimed, it is not constant. Indeed, it seems to us that the condition develops when other premises concur precisely because bronchial access is open.

\textbf{FIGURE 10} \hspace{1cm} \textbf{FIGURE 11}
The destructive factor is real; all the cases described are severe, of destructive tendency and in most patients large cavities pre-exist. However new bullae develop in other sites where no lesions are observed.

We believe that lesions do exist but that they are minimal and easily escape detection. In our case when we re-examined the first films, we had to admit that some little points which usually are taken for vascular crossings could be spots where bullae developed later.

The vascular factor is worth discussion. The work of Ellis, Grindley and Edward\textsuperscript{13} who were able experimentally to produce large cyst-like cavities in rabbits when the bronchial artery was ligated and the bronchus was obstructed at the same time is suggestive. The vascular component however explains the cyst formation only in part, for the obstructive bronchial component is lacking. On the other hand this explanation helps to clarify the mechanism of regression of the cavities for shunts between the lesser and the systemic circulation tend soon to establish themselves, the tissues in the lung do not long remain ischemic and therefore are able to recover functionally. In this regard the studies of Cudkowicz\textsuperscript{14, 15} are most enlightening. They explain the possible mechanism of pulmonary ischemia in certain areas of tuberculous disease and hyperemia in others, leading at times to hemorrhage through the channels of the systemic circulation, at times to ischemic phenomena.

The other factor that we believe is most important when it is coincidental with others is the pathological change in the surrounding structures and most particularly in the pleurae.

In all cases reported the pleural space is absent or partly occluded as proved by attempts to induce pneumothorax or as found in resected specimens.

No other instance that we know, besides ours, has been reported of development of spontaneous pneumothorax. In our patient the spontaneous pneumothorax was small and faced the unaffected area of the right lower lobe, but the upper and middle did not collapse on account of adhesions. We were misled at the beginning because the left lung seemed to have a large spontaneous space which later was confirmed to be a large bulla (Fig. 8).

It is surprising that spontaneous pneumothorax does not occur more frequently in cases which are characterized by large blebs as it does in chronic emphysema (Rossignol\textsuperscript{16}). The reports of bullae after the induction of intrapleural pneumothorax with a vanishing trend do not invalidate our opinion for they obey other mechanisms such as distortion of the bronchi.

It is our belief that fixation of the surrounding parenchyma to the chest wall by pleural adhesions is sufficient to bring about enlargement of the cavities or to create new ones by a traction effect added to real suction exerted by the cavitory defect. Enlargement of the cavity is favored by patency of the bronchi caused by the cleansing or detergent effect of the new antibiotics and chemotherapy. No other drug has the rapid de-
The detergent effect of isoniazid on the caseous material. This also explains why in many cases of tuberculosis treated by this drug the shadows clear up while the cavities remain. The cavities under these circumstances enlarge by a mechanism opposite to the commonly accepted one of the bronchial check valve.

The passive mechanism of suction without check valve action explains the negative or neutral pressures measured in these large bullae.

The cavity should expand during inspiratory expansion of the chest and the air may flow out freely during expiration, but the space will not deflate.

The innervation of the lung may also act as a contributory factor.

It is possible that the already known sympato-tonic constriction of the lumen of the bronchial arteries caused by isoniazid could act like the ligature of the vessels as observed by Ellis and his co-workers.

Those who thought that the bullae were due to the use of isoniazid discontinued its use. The cavities regressed thereafter; yet it is important to note that although we did not discontinue the use of isoniazid, nor SM and PAS in our patient, nevertheless we observed regression of the cavities.

We administered vasodilators such as Priscol and beta-piridilcarbinol as we do in our treatment of chronic emphysema with the result described. The explanation for the regressive tendency without the interruption of isoniazid may be found in the phenomenon of fatigue of the vasculo-nervous response to the drug or in the restitution of the tissue circulation through newly formed shunts between the systemic and the lesser circulation. The contribution of vasodilators to this is a matter which requires further discussion and research.

It is because circumstances for the development of bullae rarely assemble or coincide, that the phenomenon is rare.

The addition of the detergent action of the new drugs is the most important feature which helps but is insufficient by itself to create this infrequent complex.

Summarizing our discussion, the factors which concur in the development of these large parenchymal bullae within the lung tissues are:

1. More or less extensive destruction of alveolar tissue, with cleansing and expulsion of caseous material.
2. Absence of bronchial obstruction.
3. Constriction of the vessels through nervous interference.
4. Expansion of the cavities by suction partly due to fixation of the lung to the chest wall and lack of the lobar sliding of the unaffected tissues which otherwise could fill the space.
5. Regression of the bullae provided by the integrity of the parenchymal tissue and restitution of the nutrient bronchial and parenchymal vessels.

The pulmonary tissues initially undamaged would remain airless and ischemic acting as a reserve which may expand when further favorable vascularization occurs.

It has been suggested that similar instances of bulla formation will
be encountered more frequently. We also believe that their frequency will increase; at the same time we believe that their development can be avoided if their mechanical causes are prevented by adequate means which may avoid the distending factors, such as pneumoperitoneum and the graded application of detergent drugs, beginning with streptomycin and PAS prior to the use of isoniazid when indicated in multicavitary lesions with confluent shadows under which necrosis may lie hidden. The use of vasodilators to counteract the possible action of ischemic drugs may be warranted if the hypothesis of a vasoconstrictive factor seems plausible.

Finally in the particular case presented we believe that to the above named factors another could be added; viz: A possible multiple focal reaction provoked by the use of vaccine of living bacilli (Friedmann Vaccine). This might cause a multiple Koch like phenomenon with numerous necrotic foci ending as in staphylococemia and miliary tuberculosis in the production of cystic bullae.

SUMMARY

In the last two years several reports have been published on large multiple bullae developing within the parenchyma of the lung with tendency to grow or to decrease and eventually to disappear.

This type of lesions is seen in tuberculous patients treated with antibiotics and particularly in those treated with isoniazid.

The number of cases reported is not large, the author has been able to find only 25 cases in the European and South American literature and only one in the United States.

The main features of this peculiar type of tuberculous disease are described and a new case is presented with tomograms, angiograms, roentgenograms, etc. illustrating an extreme example of multiple cyst like cavities which during the course of the disease increased to 14 bullae in the right and 15 in the left lung which later regressed to four in the right and two in the left.

The mechanism of this condition is discussed at length and an hypothesis of its development is presented.

According to the author these cavities could develop under conditions which rarely assemble: Destruction of lung tissue large or small but multiple. Changes in the innervation and vasculo-nutrient factors in the lung, and particularly adhesion of both pleurae bringing about a mechanism of suction toward the defect favored by the patency of the bronchi already cleansed and patent, under the action of the antibiotics and especially under the influence of isoniazid.

The regressive trend is explained by the restoration of the blood supply through newly formed vessels or shunts between the lesser and the systemic circulation favored by the phenomenon of fatigue of the constrictive action of the drugs on the bronchial vessels.

In the case presented another rare factor is considered: the use of a vaccine of living bacilli (Friedmann's) could produce a multiple Koch
REGRESSIVE GIANT BULLOUS EMPHYSEMA

RESUMEN

En los últimos dos años han aparecido publicaciones sobre múltiples bullas en el parénquima pulmonar que se han desarrollado en enfermos de tuberculosis. Estas bullas tienen tendencia a crecer en tamaño y en número y después a decrecer y aún a desaparecer.

Esta forma de la enfermedad tuberculosa se observa en pacientes tratados con antibióticos y especialmente, cuando se usa la isoniazida. No sería estrictamente específica puesto que se han relatado casos raros en enfermos tratados con estreptomicina y aún en casos no tratados con antibióticos. Pero sería aún más rara puesto que los casos descritos han sido más numerosos desde que se usa la isoniazida, que antes de ella, aunque no dejan de ser raros.

El número de casos publicados no es grande pues el autor sólo ha podido reunir hasta ahora 25 casos publicados en Europa y en Sudamérica y sólo hay uno publicado en los Estados Unidos.

Se describen las características de esta nueva forma de la enfermedad tuberculosa y se presenta un caso nuevo con estudios tomográficos, angio- y roentgenográficos, etc. que ilustran un ejemplo extremo de esta condición que en el curso de su observación llegó a mostrar 15 bullas en el lado derecho y 16 an el izquierdo, para disminuir a sólo 4 en el derecho y 2 en el izquierdo.

Se discute extensamente el mecanismo de la aparición de estas bullas y se presenta una hipótesis de su desarrollo.

De acuerdo con las ideas del autor estas bullas múltiples se formarían cuando se reúnen condiciones numerosas que rara vez coinciden.

Esas condiciones serían: destrucción de parénquima grande o pequeña pero múltiple, cambios en la inervación y en la vascularización nutricia del pulmón y especialmente adherencia extensa de las pleuras lo que traería un complejo mecanismo de succión hacia la pérdida de parénquima y entrada de aire favorecida por la limpieza de la luz bronquial que sería determinada por la acción de los antibióticos y en especial de la isoniazida.

La tendencia regresiva sería explicada por la restauración de la circulación nutricia por vasos neoformados por intercomunicación entre la circulación mayor y la menor y por el fenómeno de fatiga frente a la acción prolongada de drogas con posible acción constrictiva vascular.

En el caso que se presenta se agrega otro factor raro: El uso de la vacuna de Friedmann, que tiene bacilos vivos, pudo haber producido un fenómeno de Koch múltiple que condujo a numerosas pérdidas de parénquima que después se llenaron por el mecanismo de succión descrito.

RESUME

Au cours de ces deux dernières années, plusieurs publications ont été consacrées à la formation de bulles dans le parenchyme pulmonaire des malades atteints de tuberculose. Ces bulles peuvent augmenter en dimen-

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sion et en nombre, puis diminuer et même disparaître.

Cet aspect de la tuberculose pulmonaire s'observe chez les malades traités par antibiotiques et plus particulièrement par l'isoniazide. Toutefois, il n'y a pas une exclusivité pour ce dernier produit puisque quelques rares cas ont été rapportés chez des malades traités uniquement par la streptomycine, et même chez des malades qui n’avaient reçu aucun traitement antibiotique ou chimiothérapique. C'est depuis l'usage de l'isoniazide en tout cas que ces lésions semblent surtout être connues.

Le nombre d'observations publiées n'est pas élevé, et l'auteur n'en a pu réunir jusqu'à ce jour que 25 en Europe, et en Amérique du Sud, et une seule au Etats-Unis.

L'auteur expose les caractéristiques de cette nouvelle modalité de la maladie tuberculeuse et en présente une nouvelle observation étudiée au point de vue tomographique, angiographique, radiologique, etc. . . . Son cas représente un type extrême de l'affection, puisqu'il se constituait jusqu'à 15 bulles du côté droit, et 16 du côté gauche, qui finalement se réduisirent à 4 à droite, à deux à gauche.

L'auteur discute le mécanisme de l'apparition de ces bulles et en propose une explication. D'après lui, ces multiples bulles se constitueront quand se trouveraient réunies une série de conditions que en pratique, coïncident rarement.

Ces conditions seraient : l'existence de zones destructives du parenchyme, de petite ou de grande étendue, mais multiples ; certaines modifications de l'innervation et de la vascularisation nourricière du poumon ; et plus spécialement des adhérences pleurales qui seraient à l'origine d'un mécanisme complexe de succion et d'entrée d'air, favorisé par l'action des antibiotiques et surtout par la propreté de la lumière bronchique produite par l'isoniazide. La tendance régressive s'expliquerait par la restauration de la vascularisation nourricière à l'aide des vaisseaux néoformés par intercommunication entre grande et petite circulation, et par la fenomene de fatigue due à l'action prolongées des drogues, qui ont provoqué constriction vasculaire.

Dans l'observation rapportée, s'ajoute un autre facteur rare, c'est l'emploi du vaccin du Friedmann, celui-ci contenant des bacilles vivants, qui ont pu produire un phénomène de Koch, occasionnant de nombreuses destructions parenchymateuses, qui ont été ultérieurement comblées par le mécanisme de succion décrit.

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