Review Article

Chiari malformation: Has the dilemma ended?

ABSTRACT

Chiari malformation as a clinical entity has been described more than hundred years ago. The concepts regarding pathogenesis, clinical features and management options have not yet conclusively evolved. Considering that a variety of treatment methods are being adopted to treat Chiari malformation is suggestive of the fact that confusion still reigns supreme in the minds of treating clinicians. Over the years, the understanding of Chiari malformation has changed from a disease process to a natural protective phenomenon and the treatment from decompression to fixation.

Keywords: Chiari malformation, history, instability, management, pathophysiology

INTRODUCTION

Chiari malformations (CMs) have always been accompanied with an element of confusion. The initial confusion was regarding its nomenclature, and even now, its pathophysiology and management are perplexing. To add to the bafflement are the various anomalies accompanying the CMs, namely, syringomyelia, hydrocephalus, basilar impression, basilar invaginations, and other syndromic associations. In this review, we attempt to summarize the history, pathophysiology, and management strategies of the CMs.

CONFUSION IN NOMENCLATURE

1883

John Cleland also known as the "the Hercules of British Anatomy" in 1883 long before the papers published by Chiari described the pathological findings of an elongated brainstem and fourth ventricle extending into the cervical canal.[1] Cleland particularly noted distortion of the "inferior vermiform process, which extends up so far that what appears to be the pyramid touches the corpora quadrigemina, whereas the uvula looks backward and the laminated tubercle hangs down from an exaggerated velum posticum, as an appendix ¾ of an inch in length, lying in the fourth ventricle." From these observations, he concluded that primary dysgenesis of the brain was responsible for the anomaly. In addition, he ruled out hydrocephalus as a cause of CM. However, unfortunately, his work did not become very widely known at the time.

1891

Hans Chiari, an Austrian pathologist, published in 1891 a paper entitled "Concerning alterations in the cerebellum resulting from cerebral hydrocephalus" in Deutsche Medizinische Wochenscriff.[2]

He described three malformations,[3] which he postulated were the result of consecutive changes in the region of the cerebellum caused by hydrocephalus, namely:

1. Protrusion of the medulla and tonsils of cerebellum into the cervical spinal canal

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2. Protrusion of the inferior parts of the cerebellum and the fourth ventricle into the cervical spinal canal and
3. Displacement of most of the cerebellum into a cervical-occipital bony defect.

Chiari I malformation was characterized by "elongation of the tonsils and medial divisions of the inferior lobules of the cerebellum into cone-shaped projections which go along the medulla oblongata into the spinal canal." The abnormality was restricted to the cerebellum, and the medulla was not involved. However, some authors included a medullary abnormality.

In this initial paper, Chiari defined the Type II malformation as the displacement of parts of the cerebellum and the elongated fourth ventricle into the cervical canal.[2] However, in his second paper, he changed the definition to "displacements of parts of the inferior vermis, pons, and medulla oblongata as well as elongation of the fourth ventricle into the spinal canal."[3]

1894

Arnold of Heidelberg in 1894 described a single case of myelocyst, in which there was a transposition of the embryonic tissue composing the medulla into the cervical canal.[4] He made no reference to Chiari's earlier paper.

1896

Chiari analyzed a series of 63 cases of congenital hydrocephalus with reference to changes in the cerebellum, pons, and medulla.[2]

He divided these malformations into 4 types:

- Type I was when there was a hindbrain herniation without any associated myelomeningocele
- Type II was when there was hindbrain herniation with an associated myelomeningocele
- Type III was an occipitocervical meningoencephalocele
- Type IV consisted of cerebellar hypoplasia without any displacement.

1907

The nomenclature was altered in the year 1907 by Schwalbe and Gredig[5-7] when they were working in Arnold's laboratory in Heidelberg. Arnold in 1894 had described an infant with spinal bifida who had an elongation of the hind part of the cerebellum, covering the fourth ventricle and extending into the cervical canal. Schwalbe and Gredig renamed Chiari's Type II malformation as the Arnold Chiari malformation (ACM). They referred to the cerebellar malformation as Arnold's deformity and the medullary deformity as Chiari's deformity.

1965

Peach studied the features of the CMs and urged the use of the term "Arnold Chiari malformation to only the Type II abnormality.[8,9] Peach defined it as" a variable displacement of a tongue of tissue, derived from the inferior cerebellar vermis, into the upper cervical canal accompanied by a similar caudal dislocation of the medulla, and fourth ventricle, the medulla often showing a "kink-like" deformity. This definition and name have carried forward into current practice.

Over the years, various authors proposed various clinical and radiological criteria to define and prognosticate the CMs. Authors recognized a Chiari-like syndrome which occurred in patients with syringomyelia without any tonsillar herniation.

The current classification system includes all these variations and consists of 6 types.

- Chiari Type 0 malformation: This is characterized by an altered cerebrospinal fluid (CSF) dynamics at the level of the foramen magnum. Patients with this type have syringomyelia without tonsillar herniation or with only mild tonsillar herniation associated findings
- Chiari Type 1 malformation: There is herniation of the cerebellar tonsils more than 5 mm below the foramen magnum. There is usually an associated syringomyelia. It is not usually associated with brainstem or fourth ventricular herniation or hydrocephalus
- Chiari Type 1.5 malformation: specifically describes patients with Chiari Type I malformations but with the addition of an elongated brainstem and fourth ventricle
- Chiari Type 2 malformation: This is characterized by caudal herniation of the cerebellar tonsils, brainstem, and fourth ventricle through the foramen magnum. It is mostly accompanied by myelomeningocele, hydrocephalus, and sometimes syringomyelia
- Chiari Type 3 malformation: This consists of occipital encephalocele
- Chiari Type 4 malformation: This consists of cerebellar aplasia or hypoplasia associated with aplasia of the tentorium cerebelli.

CONFUSION IN ETIOLOGY AND PATHOPHYSIOLOGY

Hans Chiari's paper attributed hydrocephalus to be the cause of the CMs.[2,3] Gardner suggested that the fundamental mechanism was hydrocephalus with resulting foraminal herniation of the hindbrain.[10] When an associated myelomeningocele was present, the associated cord traction may contribute to the deformity. The hydromyelia was due to extrusion of fluid into the central canal from an obstructed 4th ventricle. He also suggested that the "congenital" form

of platybasia was due to the increased weight of the head and malleability of the skull bones in cases of congenital obstructive hydrocephalus. In the acquired form of platybasia, the CM results due to the encroachment on the volume of the brain caused by the deformity.

Gardner – hydrodynamic theory

In 1965, Gardner published the hydrodynamic theory to explain the CMs and syringomyelia.[10] Gardner, in his paper, noted that Chiari did not attribute the hindbrain herniation to a small posterior fossa. He also failed to note the low lying tentorium in such cases. Gardner proposed that the inadequate permeability of the rhombic roof and foraminal obstruction was responsible for the Chiari and Dandy–Walker malformations. It had been shown by Padget that the anlage of the transverse sinus and tentorium arises far anteriorly and is pushed caudally by the enlarging forebrain. If due to the inadequate permeability of the rhombic roof the physiological hydrocephalus of the forebrain becomes pathological, the expanding forebrain will push the tentorial anlage inferiorly. This will result in a small posterior fossa, which fails to accommodate the growing hindbrain so that its posterior portion will be pushed caudally through the foramen magnum resulting in herniation. He went on to state that if the posterior fossa is severely reduced the earlier developing vermis will herniate resulting in a Chiari II abnormality and if the reduction in the posterior fossa is less severe, the later developing tonsils would herniate resulting in a Chiari I abnormality. Similarly, if the hindbrain was more yielding then the tentorium would fail to migrate inferiorly resulting in a large posterior fossa with a dilated fourth ventricle resulting in a Dandy–Walker malformation. He further explained the occurrence of the accompanying syringomyelia by his "hydrodynamic" theory. He postulated that it was not the mean increased pressure in the ventricles that caused the syrinx formation but rather the water hammer effect of the sudden spurt of ventricular fluid imparted to it by each pulse beat of the choroid plexus. The syrinx develops by a method of hydrodissection similar to that which is responsible for the development of the subarachnoid space in the embryo.

William – theory of craniospinal pressure dissociation

In his article, William speculated that birth injury is probably the most common cause of the CM.[11,12] The compression of bones of the vault may cause downward displacement of the brain and particularly if associated with tentorial tears may cause hindbrain herniation. The local compression of the bones may be due to expulsive force of the uterus or obstetric forceps, and this may cause fracture of the occipital bone. The local hemorrhage around the cistern magna, brain swelling due to anoxia, and transient hydrocephalus may all play a part in starting off the hernia. The propagation of the hindbrain herniation and the associated syringomyelia was explained by the pressure differential between the cranium and the spine.

According to his craniospinal dissociation theory, during moments of raised intra-abdominal pressure, there is venous congestion of the epidural veins causing engorgement around the dural sac. This, in turn, causes displacement of the CSF upward toward the cranial cavity. In normal individuals, this excess CSF would just as easily flow downward, after the episode causing raised intra-abdominal pressure has ended. In patients with CM, the hindbrain acts as a valve not allowing the CSF to flow back. Thus, a pressure differential is created causing a suck effect. This suck effect causes further hindbrain herniation. It also causes the CSF from the fourth ventricle to flow down through the obex into the central canal thus causing syringomyelia. Once the fluid is present within the cord cavity and it reaches a critical size, it pulsates both upward and downward, in response to fluid movements and pressure changes in the subarachnoid space, the most influential being the venous pressure changes. This movement "slosh" causes both upward and downward propagation of the syringomyelia.

The theories of Chiari and Gardner did not explain why a CM does not occur in all patients with congenital hydrocephalus, and likewise, why does platybasia not occur in all patients with congenital hydrocephalus.

Likewise, for syringomyelia to occur there would have to be a patent communication between the fourth ventricle and the central canal, which has not been seen frequently. It has been argued by some that the communication may have been present initially but closes off after a period of time.

Oldfield theory

In 1994, Oldfield proposed another theory to explain the formation and propagation of the syringomyelia based on the normal cardiac cycle.[13] In normal individuals, during systole the brain expands after receiving blood. This causes the CSF to flow from the 4th ventricle into the cisterna magna, and then, onward into the upper cervical canal through the foramen magnum. During diastole, the CSF flows upward from the spinal canal to the cranial cavity across the foramen magnum. When there is obstruction at the region of the foramen magnum, there is impedance to this rapid to and fro movement of CSF in the subarachnoid space across the foramen magnum during systole and diastole. During systole, the brain expansion is accommodated by an abrupt piston-like caudal movement of the tonsils. As there is occlusion of the

rapid upward movement of the CSF, there is a partially isolated spinal subarachnoid space. This movement of the tonsils imparts a pressure wave to the spinal subarachnoid space causing fluid movement of CSF into the cord and also propelling the syrinx fluid in the cord inferiorly.

Theory of paraxial mesodermal insufficiency[14-19]

Marin-Padilla *et al*. postulated that a paraxial mesodermal insufficiency or primary lesion of the somatic structures might induce various neural abnormalities in CMs. The hypothesis proposes that a primary paraxial mesodermal insufficiency (Vitamin A-induced) could affect embryos before, during, and after the closure of the neural folds resulting in a variety of developmental malformations which share a common type of axial skeletal defect and different neurological anomalies which reflect the degree of the dysraphic disturbance. ACM, in spite of its lack of a distinct dysraphic defect, represents an example of this type of developmental disorder, and as such it has been classified.[14]

In 1998, Goel *et al*. observed that the CM in basilar invagination was caused by a reduction in the volume of the posterior cranial fossa.[20]

Nishikawa *et al*., in their morphometric study, found that two parts of the occipital enchondrium (the exocciput and supraocciput) were underdeveloped in patients with adult-type CM, and that all three parts of the occipital enchondrium (the exocciput, supraocciput, and basiocciput) were underdeveloped in patients with basilar invagination.[18] Like the observations of Goel, their paper also implied CM to be a result of normal development of the hindbrain and underdevelopment of the occipital somites.

Goel: Theory of instability[21,22]

The theory of short/small posterior fossa as proposed by Dr. Goel earlier and others has been the most accepted explanation offered for CM. However, this theory did not explain the occurrence of instability in patients with CM. It also does not explain the occurrence of superior cerebellar atrophy in patients with CM. Furthermore, not all patients with CM have a short posterior fossa.

Goel postulated that basilar invagination, CM and syringomyelia represent a spectrum of abnormalities wherein the primary etiology is atlantoaxial instability.[21,22] In patients with an acute (or traumatic) presentation atlantoaxial dislocation, there is no basilar invagination, CM, or syringomyelia. In more subtle and longstanding atlantoaxial instability, basilar invagination, CM and syringomyelia may occur singly or in combination. In patients with mild atlantoaxial instability.[21,22] The CM is usually more severe. CM may be Nature's protective mechanism that assists in reducing the effect of instability and cord compression by the odontoid process. Essentially, it has been observed that CM is a manifestation of atlantoaxial instability. The prominence and easy identification of CM on MRI could divert the clinician's attention from the relatively poorly delineated atlantoaxial joint and instability. It appears that CM is unrelated to the reduction in posterior cranial fossa volume, and nor is it attributable to the primary or relative increase in the cerebellar mass. These features suggested that the hypothesis of intracranial or posterior cranial fossa hypertension cranial constriction, or an increase in cerebellar mass in proportion to the volume of the posterior cranial fossa as a cause of cerebellar herniation may not be correct. It appears that the temporary improvement after foramen magnum decompression is akin to deflating a full airbag, and in the long term such a form of surgery can be counter-productive.

CONFUSION IN MANAGEMENT

The first report of surgery for CM was in the year 1932 when Van Houweninge Graftidijik reported his attempt to correct the deformity.[23] He performed removal of posterior fossa bone, opening of the dura, and resection of the redundant tissue. The surgery was aimed at relieving the obstruction of CSF flow. However, his patients died either as a result of the surgery or due to postoperative complications.

In 1938, McConnell and Parker[24] published their results of posterior fossa decompression for Chiari I malformation in five patients. Only two of these patients had successful outcomes.

The 1940's saw a series of case reports and series of patients who were surgically treated for CM. List in 1941 reported three patients with CM who were operated on successfully.[6] Ogyzlo in 1942 reported 7 cases treated surgically.[25] Out of these, 4 patients had spinal bifida and three had CM. The patients were operated on successfully. There were reports of successful treatment from many other authors.

In 1945, Bucy and Lichtenstein[26] reported decompression for a Chiari I malformation of a 40-year-old woman without hydrocephalus and in 1948, Chorobski and Stepien[27] operated a woman with life-altering, Valsalva-induced headache, and Chiari I malformation

In 1950, Gardner and Goodall reported a series of 17 patients operated by them.[28] In 8 of these patients platybasia and basilar invagination were definitely identified, and in another 3, a mild degree of platybasia was noted. Two of the patients

had evidence of Klippel–Feil syndrome. The surgery consisted of a suboccipital craniectomy, an upper cervical laminectomy and opening of the dura. The dura was left open, and they made an attempt to reopen the foramen of Magendie in each case. They also recommended plugging the upper end of the patent central canal at the area of the obex with a small piece of muscle. In this series, 13 patients improved following the surgery, 3 worsened with aggravation of quadriparesis, and 1 died 18 h after the surgery due to respiratory failure.

In these reports of patients treated by earlier surgeons, all patients with CM, that is patients having associated basilar invagination, myelomeningocele, etc., were included and the management consisted of suboccipital decompression, laminectomy, opening of the dura with and without resection of the tonsils. A select group of surgeons (Ricard, List, Vidigal and Lucia) added an occipitospinal fusion to the decompressive procedure using a bone graft or immobilization of the neck in a plaster cast.[29]

Caetano de Barros *et al*. in 1968 published a series of patients with basilar impression and CM treated by them.[29] They also performed a suboccipital craniectomy, tailoring the laminectomy based on individual cases. They advocated opening of the dura with cutting of all the constrictive dural and arachnoid bands. They strongly advised against resection of the tonsils. In their 32 operated cases, two patients were cured, 19 improved following surgery and 4 were unchanged. The mortality rate was 21% (7 cases) which was similar to the rate seen in other surgical series.

Some authors in addition to the above procedure checked the patency of the 4th ventricle during surgery, and some believed that third ventriculostomy before decompression may be necessary in patients with intracranial hypertension.

Even though surgical treatment of this condition continued, the results varied and the mortality rate was high. In 1976 Saez *et al*. reported their experience with 60 adult patients of CM treated at the Mayo Clinic from 1960 to 1970.[30] The majority of the patients had a normal X-ray of the skull and cervical spine. Fourteen patients had basilar invagination. The patients were treated with suboccipital craniectomy and upper cervical laminectomy. The dura was either left open, or a homologous patch was applied. The longest follow-up was 14 years. Out of this group of patients, 65% benefitted from the surgery (20% became asymptomatic, and 45% were definitely improved). However, 18.3% of patients (nearly one-fifth) continued to deteriorate. In some patients, the initial postoperative benefit tended to fade into an insidious progression of neurological deficit.

Levy *et al*. presented their experience of 127 cases of adult CM treated from 1946 to 1983.[31] In all their patients, a decompression of the foramen magnum up to C3 level with opening of the dura was performed. Other additional procedures performed included an exploration of the 4th ventricle, opening of the membrane over the foramen of Magendie, insertion of a muscle plug at the upper end of the central canal, a syringo-subarachnoid or a syringopleural shunt, a 4th ventricle to subarachnoid shunt and a terminal ventriculostomy either singly or in combination. A long-term follow-up was available in 85 patients. Out of these, 40 patients (47%) improved, 23 patients (27%) were unchanged, and 22 patients (26%) worsened. Out of the 46% who initially improved a few deteriorated and presented to the authors for re-surgery. They concluded that no clearly superior treatment existed for this anomaly. They did not find any clear-cut difference between plugging the central canal or simple decompression. Syrinx shunting was not helpful and did not alter the clinical course. The disease was considered as Lord Brain described it as "relentlessly progressive" and any treatment that slowed or halted the disease was considered as beneficial.

Similarly, Paul *et al*. presented a series of 71 patients of ACM Type I operated by them.[32] A suboccipital craniectomy with an upper cervical laminectomy and duroplasty was performed in all the patients. The patients were followed up over 6 months to 9 years. Despite an early improvement in 82% of patients, 21% of the patients subsequently deteriorated to their preoperative state. Relapse in these patients occurred within 2–3 years after surgery.

Due to these conflicting results that prevailed while treating Chari malformation, many authors reported classification of CM according to the etiology and tailored its management.

Goel *et al*. in 1998 divided basilar invagination into two groups on the basis of presence or absence of CM.[20,33] The prime issue in this classification was the understanding that atlantoaxial dislocation in both groups was considered to be of fixed or of irreducible variety. Essentially, Group I included cases where there was invagination of the odontoid process into the foramen magnum, and it indented into the brainstem. The tip of the odontoid process distanced itself from the anterior arch of the atlas or the inferior aspect of the clivus. The angle of the clivus and the posterior cranial fossa volume were essentially unaffected in these cases. In Group II cases, on the other hand, the assembly of odontoid process, anterior arch of the atlas, and the clivus migrated

superiorly in unison resulting in reduction of the posterior cranial fossa volume, which was the primary pathology in these cases. The CM or herniation of the cerebellar tonsil was considered to be a result of reduction in the posterior cranial fossa volume.[20] In Group I, basilar invagination the tip of the odontoid process "invaginated" into the foramen magnum and was above the Chamberlain line,[34] McRae line of foramen magnum[35] and Wackenheim's clival line.[36] The definition of basilar invagination of prolapse of the cervical spine into the base of the skull, as suggested by von Torklus and Gehle,[37] was suitable for this group of patients. Group II basilar invagination was where the odontoid process and clivus remained anatomically aligned despite the presence of basilar invagination and other associated anomalies. In this group, the tip of the odontoid process was above the Chamberlain's line[34] but below the McRae's[35] and the Wackenheim's lines.[36] As deformity rather than instability was considered to be the issue in pathogenesis, decompression of the neural structures rather than stabilization was considered to be the therapeutic goal. On the basis of this study, Goel recommended transoral decompression with or without atlantoaxial or occipitocervical fixation in Group I patients and foramen magnum decompression in Group II patients. Goel *et al.* also suggested that opening of the dura during posterior fossa decompression was not recommendable and should be avoided in all cases, including those where there was an association of CM and syringomyelia.[20] Before this dural opening and widening of posterior fossa by dural graft placement was an accepted norm. In the year 2004 on further understanding of the subject, Goel *et al*. proposed another classification scheme of basilar invagination. In this paper, basilar invagination was divided into two groups.[37] In one subgroup of patients there was radiological evidence of instability of the region which was seen as distancing of the odontoid process away from the anterior arch of atlas/clivus or the atlantodental or clivodental interval was abnormally increased. The tip of the odontoid process was above the Chamberlain line, McRae line and Wackenheim clival line. This subgroup of patients was labeled as having Group A basilar invagination. The radiological findings suggested that the odontoid process in Group A patients resulted in direct compression of the brainstem. In some Group A patients there was Chiari 1 malformation, and this feature differentiated the present classification from the earlier classification. The pathogenesis in patients with Group A basilar invagination was mechanical instability of the region. In these patients the atlantoaxial joints were in an abnormally inclined or oblique position instead of the normally found horizontal orientation. The alignment of facets of atlas and facets of axis simulated positioning of vertebral bodies in lumbosacral listhesis. Basilar invagination in this subgroup of patients appeared to be related to progressively increasing listhesis of facets of atlas over the facets of axis. The joint in these cases is not fixed or fused but is mobile or hypermobile, thus is probably the primary cause of basilar invagination. As instability or listhesis is the cause of basilar invagination, stabilization as recommended for lumbosacral listhesis formed the baseline of surgical treatment. In Group B the entire complex of the clivus, basiocciput and the craniovertebral junction was rostrally located and the tip of the odontoid process was superior to the Chamberlain line but inferior to the McRae and the Wackenheim lines. In Group B, the pathogenesis appeared to be congenital dysgenesis of the region. The atlantoaxial joint was considered to be stable or fixed and instability was not considered an issue in this group of patients. Foramen magnum decompression was identified to be the treatment for Group B basilar invagination, as small posterior cranial fossa volume was identified to be the pathological issue in this group.

Milhorat in 2010 measured the posterior fossa volume in patients with CMs due to varying etiologies.[38] He suggested the following causal mechanisms as the genesis of CMs: cranial constriction, cranial settling, spinal cord tethering, intracranial hypertension, and intracranial hypotension. The authors found that the posterior fossa volume was reduced only in patients with the classical CM, that is, without any etiological factors.

Kleklamp presented his analysis of 644 patients of CM operated between 1985 and 2010.[39] Out of the 644 patients, 359 patients underwent 371 decompressions which consisted of suboccipital craniectomy, C1 laminectomy, arachnoid dissection, and duroplasty. There was a complication rate of 21.8% with permanent surgical morbidity of 3.2% and surgical mortality of 1.3%. After 3 months of surgery, 73.6% of the patients reported improvement, whereas the rest 21% remained unchanged. Further, of the patients who had improved, 14.3% demonstrated a neurological deterioration within 5 years and 15.4% within 10 years. The factors predicting outcome were number of previous decompressions, severity of arachnoid pathology, arachnoid handling, type of duroplasty, and surgical experience. In 2012, Kleklamp in a subsequent article analyzed the reasons for the neurological deterioration in operated cases of CM.[40] They found that neurological deterioration in patients after a foramen magnum decompression for CM-I may be related to new spinal pathologies, craniocervical instability, or recurrent CSF flow obstruction at the foramen magnum.

In 2015, Goel published his theory of atlantoaxial instability as the cause of CM.[21] In the series of 65 patients operated

by him, 46 patients had either Group A or Group B basilar invagination. Nineteen patients had no craniovertebral region bony abnormality. All the patients were treated with atlantoaxial fixation. No foramen magnum decompression or duroplasty was performed in any of the patients. Out of the 65 patients operated, one patient died in the immediate postoperative period, which was related to vertebral artery injury. Only 1 patient had persistent symptoms. All the other patients improved in their symptoms, and the improvement was sustained at follow-up.

CONCLUSIONS

The pathogenesis and management of CM have stupefied generations of neurosurgeons. The most common surgical treatment has been posterior fossa bony decompression with or without dural opening and with or without tonsillar resection. Although the results of this kind of treatment have benefitted quite a few individuals, there remains a big number who have not done well following the surgery or worsened after an initial improvement. The goal of treatment has always been to halt the progression of disease and not clinical improvement. The decompressive surgery is sometimes accompanied by fixation either in the same sitting or at a later date after an initial failed surgery. In science, most complex problems have at the end always had a simple rationalization. Atlantoaxial fixation can be the plain answer to the vexing problem of CM. If at all it fails, the posterior fossa decompression can be performed at a later date.

Facts which at first seem improbable will, even on scant explanation, drop the cloak which has hidden them and stand forth in naked and simple beauty– Galileo Galilei

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