UNITED STATES

SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

Form 6-K

Report of Foreign Private Issuer
Pursuant to Rule 13a-16 or 15d-16
under the Securities Exchange Act of 1934

June 2017

Commission file number: 001-36288

Akari Therapeutics, Plc
(Translation of registrant's name into English)

24 West 40th Street, 8th Floor
New York, NY 10018
(Address of principal executive offices)

Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F.

Form 20-F ☒ Form 40-F ☐

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulations S-T Rule 101(b)(1):_____

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulations S-T Rule 101(b)(7):_____

CONTENTS

On June 23, 2017, Akari Therapeutics, Plc, (the "Company") issued a press release announcing that it is presenting a poster at the 22nd Congress of the European Hematology Association (EHA) in Madrid, Spain containing data for the four patients, to date, who have completed the Company's ongoing Phase II trial of Coversin in paroxysmal nocturnal hemoglobinuria (PNH). A copy of the press release and poster are attached hereto as Exhibit 99.1 and 99.2 respectively, and are incorporated herein by reference.

The information contained in this report (including the exhibits hereto) is hereby incorporated by reference into the Company's Registration Statement on Form S-3, File No. 333-207443, Form S-8 (No. 333-198109 and 333-207444), Registration Statement on Form F-3 File No. 333-198107, and the Registration Statements on Post-Effective Amendments to Form F-1 on Form F-3 (333-185247, 333-187826 and 333-191880).

Exhibit No.

99.1 Press Release dated June 23, 2017

99.2 Poster

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

Akari Therapeutics, Plc
(Registrant)

By: /s/ Robert M. Shaw
Name: Robert M. Shaw
General Counsel & Secretary

Date: June 23, 2017

Exhibit 99.1

Akari Therapeutics to Present Data from Phase 2 PNH Trial of Coversin at the 22nd Congress of the European Hematology Association

NEW YORK and LONDON, June 23, 2017 -- Akari Therapeutics (NASDAQ: AKTX), a clinical-stage biopharmaceutical company, is presenting a poster today at the 22nd Congress of the European Hematology Association (EHA) in Madrid, Spain containing data for the four patients, to date, who have completed its ongoing Phase 2 trial of Coversin in paroxysmal nocturnal hemoglobinuria (PNH). Coversin is a second-generation complement inhibitor that is capable of being a self-administered subcutaneous injection.

The poster can be found here:

http://akaritx.com/wp-content/uploads/2017/06/EHA_POSTER_23JUN2017.pdf

About Akari Therapeutics Plc

Akari is a clinical-stage biopharmaceutical company focused on the development and commercialization of life-transforming treatments for a range of rare and orphan autoimmune and inflammatory diseases caused by dysregulation of complement C5 and Leukotriene B4 (LTB4), including paroxysmal nocturnal hemoglobinuria ("PNH"), atypical Hemolytic Uremic Syndrome ("aHUS"), and Guillain Barré syndrome ("GBS"). Akari's lead product candidate, Coversin™ complement inhibitor, a second-generation complement inhibitor, has shown it can act on complement component-C5, preventing the release of C5a and the formation of C5b–9 (also known as the membrane attack complex or MAC), and independently also inhibits LTB4 activity. C5 inhibition is growing in importance in a range of rare autoimmune diseases related to dysregulation of the complement component of the immune system, including PNH, aHUS, and GBS. Exploiting the power of nature, Akari is also developing other tick derived proteins and expects to bring additional compounds to clinical trials over the next several years. The pipeline is focused on developing bioengineered versions of native tick salivary proteins that act as anti-inflammatory compounds allowing the tick to remain on its host. These compounds include PGP sparing LTB4 inhibitors, classical and alternative complement inhibitors, anti-histamines, and serotonin inhibitors as examples. Akari is also developing engineered forms that allow for potential oral absorption, as, for example, a potential orally absorbed C5 inhibitor, and tissue specific proteins, as, for example, Coversin™ that acts specifically at the neuromuscular junction for diseases like myasthenia gravis.

Cautionary Note Regarding Forward-Looking Statements

Certain statements in this press release constitute "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995. These forward-looking statements reflect our current views about our plans, intentions, expectations, strategies and prospects, which are based on the information currently available to us and on assumptions we have made. Although we believe that our plans, intentions, expectations, strategies and prospects as reflected in or suggested by those forward-looking statements are reasonable, we can give no assurance that the plans, intentions, expectations or strategies will be attained or achieved. Furthermore, actual results may differ materially from those described in the forward-looking statements and will be affected by a variety of risks and factors that are beyond our control. Such risks and uncertainties for our company include, but are not limited to: needs for additional capital to fund our operations, an inability or delay in obtaining required regulatory approvals for Coversin and any other product candidates, which may result in unexpected cost expenditures; risks inherent in drug development in general; uncertainties in obtaining successful clinical results for Coversin and any other product candidates and unexpected costs that may result therefrom; failure to realize any value of Coversin and any other product candidates developed and being developed in light of inherent risks and difficulties involved in successfully bringing product candidates to market; inability to develop new product candidates and support existing product candidates; the approval by the FDA and EMA and any other similar foreign regulatory authorities of other competing or superior products brought to market; risks resulting from unforeseen side effects; risk that the market for Coversin may not be as large as expected; inability to obtain, maintain and enforce patents and other intellectual property rights or the unexpected costs associated with such enforcement or litigation; inability to obtain and maintain commercial manufacturing arrangements with third party manufacturers or establish commercial scale manufacturing capabilities; the inability to timely source adequate supply of our active pharmaceutical ingredients from third party manufacturers on whom the company depends; our inability to obtain additional capital on acceptable terms, or at all; unexpected cost increases and pricing pressures; uncertainties of cash flows and inability to meet working capital needs; and risks and other risk factors detailed in our public filings with the U.S. Securities and Exchange Commission, including our Annual Report on Form 20-F filed on March 31, 2017. Except as otherwise noted, these forward-looking statements speak only as of the date of this press release and we undertake no obligation to update or revise any of these statements to reflect events or circumstances occurring after this press release. We caution investors not to place considerable reliance on the forward-looking statements contained in this press release.

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Coversin, a novel C5 complement inhibitor, for the treatment of PNH: results of a Phase 2 clinical trial

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Introduction

Paroxysmal nocturnal haemoglobinuria (PNH) is a rare acquired life-threatening disease, characterized by complement induced haemolysis and a high incidence of thrombosis.

Coversin, a small (16.8kDa) protein C5 complement inhibitor, originally derived from the haematophagous tick *Ornithodoros moubata*, is being developed as a therapy for PNH. Coversin is administered by subcutaneous injection and can be self-administered, making it possible for patients to treat themselves at home.

Following completion of Phase 1 clinical trials in healthy human volunteers, a Phase 2 open label 90 day trial was initiated December 2016. Enrolled patients had not previously received anti-complement therapy.

Presented here are the results of the first four patients to complete the Phase 2 trial.

The **OBJECTIVES** of the trial were to assess the safety and tolerability of Coversin, the efficacy of the dosing regime and whether self-injection by patients is well-accepted.

Methods

All patients enrolled in the trial had a diagnosis of PNH confirmed by flow cytometry. Patients were eligible for inclusion whether or not they had a history of transfusion dependence. The lower age limit for inclusion was 18 years, with no upper age limit. A fixed dose regime was used for all patients with weight limits of 50 – 100kg.

Patients were required to attend a haematology clinic for 2 days whilst Coversin therapy was initiated. After instruction, patients were encouraged to start self-injection as soon as possible. Nursing support at home was provided, if requested, for the first month of the trial.

Patients attended their investigator's clinic at least weekly for the first month and then monthly until the end of the trial. Terminal complement activity and clinical response were monitored. All patients completed daily diary cards to record dosing and adverse events (AEs). EORTC-30 and EQ-5D-5L quality of life instruments were used to assess global healthcare status.

Treatment started with an ablating phase consisting of a single 60mg injection followed by 3 doses of 30mg 12 hours apart. An initiation phase of 26 days followed during which patients received 15mg 12 hourly doses. Upward dose titration to 22.5mg 12 hourly was allowed during this period in the event of inadequate clinical response. On Day 28, patients moved to a single dose of either 30mg or 45mg every 24 hours. The trial protocol permitted an increase of dose during this period from 30mg to 45mg or to split the 45mg dose into two 22.5mg doses, injected at 12 hour intervals if the response was inadequate.

After the 90 day trial, patients had the option to continue Coversin treatment under a long-term safety and efficacy protocol (CONSERVE).

Results

Five patients have been enrolled in the study to date. Patient demographics are shown in Table 1. The duration between initial diagnosis and initiation of Coversin therapy ranged from 8.5 to 79 months (mean 44 months). Four patients completed the 90 day study per protocol and moved into the long-term safety and efficacy study. One patient (Patient E) with a suspected co-morbidity unrelated to the treatment was withdrawn from the study on Day 43.

Table 1: Patient Demographics

Characteristic	Figure
Race, n(%)	Caucasian 5 (100%)
Male : Female	4:1
Age, years	
Mean	45.2
Range	22-69
Weight, kg	
Mean	76
Range	55-84

All patients had a CH50 level below the limit of quantification (≤8 CH50 U Eq/mL) after the ablating phase indicating total blockade of the terminal complement pathway (Figure 1).

Fig 1, left: Terminal complement activity measured by MicroVUE CH50 ELISA for the 4 PNH patients who remained in the Phase 2 trial. **Fig 1, right:** Detail onset CH50 inhibition

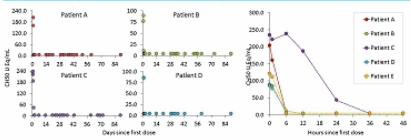


Table 2: Adverse Events

Adverse Event	Related	Possibly related
Injection site reaction	24 (32%)	41 (54%)
Rash		2 (3%)
Hypophosphataemia		2 (3%)
Hypoproteinaemia		2 (3%)
Abdominal discomfort		1 (1.3%)
Arthralgia		1 (1.3%)
Headache		1 (1.3%)
Decreased neutrophil count		1 (1.3%)
Decreased WBC count		1 (1.3%)
TOTAL	24 (32%)	52 (68%)

The study drug was well tolerated and patients reported no difficulty with self administration. There were no serious adverse events (SAEs). The most commonly reported AEs were mild to moderate injection site reactions which declined towards the end of the trial. None of the AEs required specific treatment or were severe enough to cause discontinuation of the study drug. A full listing of AEs is shown in Table 2.

All 4 patients saw declines in LDH levels (Figure 2, left). The primary end point of LDH ≤1.8X ULN at Day 28 was achieved by two patients. LDH as a multiple of ULN (xULN) for the 4 patients (A, B, C and D) at Day 28 was respectively 1.4, 2.2, 2.5 and 1.4; at Day 60 1.5, 2.1, 1.8 and 1.5; and at Day 90 1.6, 2.4, 2.0 and 1.9. Aspartate aminotransferase (AST) levels provide another measure of cellular haemolysis, AST decreased following initiation of dosing (Figure 2, right).

Three of four patients were updosed. Patient A and B were updosed from 30mg to 45mg once daily at Days 40 and 54, respectively. Patient C was updosed to 22.5mg twice daily at Day 24 and moved to 45mg once daily at Day 67. Patient B, the last in to date, did not see a decline in LDH with updosing although his haemoglobin level rose after Day 67.

Fig 2, left: LDH plotted as a multiple of ULN for the 4 patients who remained in the Phase 2 trial and as the mean of multiple ULN. **Fig 2, right:** AST over the same period

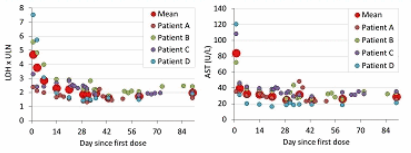
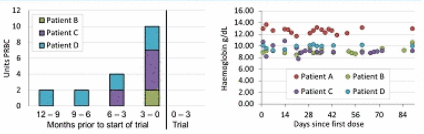
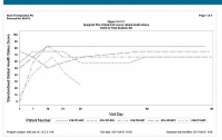


Fig 3, left: Number of units of packed red blood cells (PRBC) transfused to patients prior to and during the 90 day trial. **Fig 3, right:** Haemoglobin levels for 4 patients in trial



None of the four patients required transfusion during the trial while 3 out of 4 patients required a total of 10 units PRBC during the 3 months preceding the trial (Figure 3, left). Patient A had no history of transfusion. Haemoglobin levels for the 4 patients are shown in (Figure 3, right). For two patients (A and D), there was essentially no change in haemoglobin levels from Day 1 to Day 90. For the other two patients, haemoglobin levels increased by 19% (patient B) and decreased by 14% (patient C) from Day 1 to Day 90.

Fig 4: EORTC-30 scores all 5 patients



Results of the EORTC-30 quality of life instrument are shown in Figure 4 and show an improvement at the end of the trial for all four patients. The fifth patient has incomplete quality of life data, as he was withdrawn from the trial due to a suspected comorbidity.

Immunogenicity was analysed, all 5 patients developed low titre antibodies but no neutralising antibodies were observed.

Conclusions

- No SAEs were reported and Coversin, which was self-administered by all four patients, was well tolerated. AEs associated with the administration of monoclonal antibodies such as headache, nausea and backache were absent, except for a single reported occurrence of headache.
- In this trial, an ascending dose design was used for reasons of safety. All four patients experienced complete terminal complement inhibition. Dose increases during the trial, in 3 of the 4 patients, may have been due to an initial sub-optimal dosing regime. No neutralising antibodies were observed. Quality of life improved for all four patients by the end of the trial.
- None of the four patients required transfusion during the trial, while 3 out of 4 patients required transfusions during the 3 months preceding the trial.
- All four patients who completed the trial remain on Coversin in the long-term safety and efficacy study and are self-administering a single subcutaneous daily dose.
- To provide confirmatory information for the proposed Phase 3 trial planned to start Q4 2017, Akari plans to enrol additional patients in this Phase 2 trial using a revised dosage regime under a protocol amendment.

Note: The preceding data is from the eCRFs from Medpace, which will be audited at the end of the Phase 2 trial. The exception is the CH50 and immunogenicity data which is from UCL centrallab.

22nd Congress of the European Haematology Association, Madrid, Spain. June 22 - 25, 2017.

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